

PASSIVE RECOVERY PROMOTES SUPERIOR PERFORMANCE AND REDUCED PHYSIOLOGICAL STRESS ACROSS DIFFERENT PHASES OF SHORT-DISTANCE REPEATED SPRINTS

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ABSTRACT

Scanlan, AT and Madueno, MC. Passive recovery promotes superior performance and reduced physiological stress across different phases of short-distance repeated sprints. *J Strength Cond Res* 30(9): 2540–2549, 2016—Limited research has examined the influence of recovery modalities on run-based repeated-sprint (RS) performance with no data available relative to the sprint phase. This study compared run-based RS performance across various sprint phases and underlying physiological responses between active and passive recoveries. Nine students (21.8 ± 3.6 years; 171.3 ± 6.4 cm; 72.8 ± 12.2 kg) completed 2 bouts (active and passive recoveries) of 10×20 m sprints interspersed with 30 s recoveries in a randomized crossover fashion. Sprint times and decrements were calculated for each split (0–5, 5–15, 15–20, and 0–20 m) across each sprint. Blood lactate concentration ($[BLa^-]$), heart rate (HR), and rating of perceived exertion (RPE) were measured at various time-points. **Passive recovery promoted improved performance times ($p \leq 0.005$) and decrements ($p \leq 0.045$) across all splits, and lower post-test $[BLa^-]$ ($p \leq 0.005$), HR (bout 3 onwards) ($p \leq 0.014$), and RPE (bout 4 onwards) when compared with active recovery.** Performance differences between recoveries were less pronounced across the 0–5 m split. Temporal analyses showed significant ($p \leq 0.05$) increases in sprint times and decrements primarily with active recovery. The present data indicate that passive recovery promoted superior performance across run-based RS, with earlier performance deterioration and greater physiological load evident during active recovery. These findings can aid the manipulation of interbout activity across RS drills to promote physiological overload and adaptation during

training. Further, coaches may develop tactical strategies to overcome the detrimental effects of active recovery and optimize sprint performance in athletes during game-play.

KEY WORDS acceleration, fatigue, active recovery, passive recovery, blood lactate, heart rate

INTRODUCTION

Repeated-sprint (RS) performance is a key property in many team sports whereby athletes are required to undertake near-maximal to maximal efforts (≤ 10 seconds) interspersed with recovery periods (≤ 60 seconds) consisting of rest or low-moderate intensity activity (18). More precisely, analyses of competitive game-play have shown players to rely extensively on the repeated execution of sprint activities across various team sports including soccer (1), basketball (32), rugby league (16), and American football (40). In addition, training plans incorporating RS stimuli have been shown to improve various aspects of performance in team sports (36). Thus the existing literature emphasizes the importance of RS in team sports, with multiple researchers conducting further physiological examination to better understand the metabolic demands involved.

Repeated-sprint activity requires a high rate of adenosine triphosphate (ATP) consumption and thus rephosphorylation (18). Consequently, RS have been suggested to use anaerobic and aerobic sources for ATP provision, with the reliance placed on each metabolic pathway dependent on RS protocol configuration (18,19). Phosphocreatine (PCr) reserves present an immediate pathway for ATP resynthesis during sprinting activity (18). However, PCr stores diminish with each subsequent bout during RS, and may only be partially restored during the brief recovery periods (17). Likewise, extensive reductions in glycolytic recruitment have been demonstrated across RS (17), with progressive physiological inhibitory mechanisms thought to underpin this response (18). For instance, Gaitanos et al. (17) estimated total anaerobic ATP production to decrease from 89.3 ± 13.4 to 31.6 ± 14.7 mmol·kg⁻¹·dry wt⁻¹, including

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reductions in sources from PCr (44.3 ± 4.7 vs. 25.3 ± 9.7 mmol·kg·dry wt⁻¹, $p < 0.01$) and glycolysis (39.4 ± 9.5 vs. 5.1 ± 8.9 mmol·kg·dry wt⁻¹) between the first and 10th 6 s cycling sprint interspersed with 30 s recovery period. In contrast, oxidative metabolism has been shown to contribute little to ATP resynthesis across initial sprinting bouts, with increased reliance accompanying repeated efforts (4). For example, McGawley and Bishop (26) recorded an increased oxygen uptake ($\dot{V}O_2$) across the tenth 6 s cycling sprint compared with the initial bout (3.14 ± 0.61 vs. 1.08 ± 2.86 L·min⁻¹) showing a higher aerobic contribution with RS progression. Although these temporal changes in metabolic responses have been identified as key determinants of fatigue during RS (18), the recovery modalities adopted are likely to influence physiological load and subsequent performance.

Recovery modalities between bouts during RS have received increased interest, with active and passive approaches primarily compared across cycling (5,11,13,35) and swimming (37–39) exercises. However, these findings have limited transfer to run-based RS. In turn, the paucity of research examining recovery modality during run-based RS (7,8,12) has shown passive recovery to promote better performance and reduced fatigue across varied sprint and recovery durations. More precisely, Buchheit et al. (7) recorded a greater mean sprint distance (16.48 ± 1.32 vs. 15.24 ± 1.54 seconds, $p < 0.001$) and lower sprint decrement (3.2 ± 2.4 vs. $7.1 \pm 1.1\%$, $p < 0.001$) during passive recovery (standing) compared with active recovery (jogging at $2 \text{ m} \cdot \text{s}^{-1}$) across 6×4 s treadmill sprints with 21 s recoveries. Similarly, Castagna et al. (8) observed a reduced mean sprint time (6.17 ± 0.10 vs. 6.32 ± 0.10 seconds, $p = 0.03$) and sprint decrement (3.4 ± 2.3 vs. $5.1 \pm 2.4\%$, $p < 0.001$) during passive recovery (standing) compared with active recovery (50% maximal aerobic speed [MAS]) across 10×30 m shuttle sprints with 30 s recoveries. In addition, Dupont et al. (12) recorded a longer time to exhaustion (745 ± 171 vs. 445 ± 79 seconds, $p < 0.001$) during passive recovery (standing) than active recovery (50% MAS) across repeated 15 s efforts (120% MAS) interspersed with 15 s recoveries. Various physiological mechanisms were proposed to explain the superior RS performance with passive recovery including reduced cardiorespiratory stress, oxygen (O_2) cost, and muscle deoxygenation (7), elevated myoglobin and hemoglobin O_2 restoration (12), and increased PCr resynthesis (12,22).

Thus, limited insight has been provided regarding the influence of recovery modalities on run-based RS performance. Further, existing studies comparing active and passive recoveries have used exercise durations (15 s) or distances (30 m) exceeding those typically observed in various team sports (8,12), treadmill-based exercise (7), protocols working subjects to exhaustion (12), and limited physiological measurement (8), which restrict the applicability of the available data to practice. Moreover, previous examinations have been conducted across entire sprinting bouts, with more in-depth anal-

yses of temporal changes in performance relative to sprint phase absent in the literature. Consequently, the aim of this study is to examine the between- (active vs. passive recovery) and within-condition (temporal changes) differences in performance and physiological responses across different sprint phases during run-based RS. **Based on the available evidence regarding the influence of recovery modalities on run-based RS (7,8,12), it was hypothesized that passive recovery would promote better performance and reduce physiological demands compared to active recovery.**

METHODS

Experimental Approach to the Problem

A randomized crossover study design was administered whereby subjects completed 2 bouts of the RS protocol. Each bout was separated by at least 24 hours as previously adopted in RS research (22). The RS protocol consisted of 10×20 m run-based sprints with 30 s of either active or passive recovery (independent variables) between sprinting bouts. The active recovery protocol involved subjects decelerating after each sprint and then continuously jogging at 50% of maximal sprint speed. This intensity equated to approximately $2.7 \pm 0.2 \text{ m} \cdot \text{s}^{-1}$, which is similar to that used in other RS protocols (7) and categorized as “jogging” or “low-intensity activity” using defined speed zones in team sports (14,23,33). The passive recovery protocol involved subjects decelerating and then standing (7,8,12). The RS protocol follows similar configurations of established tests with supported reliability for sprint times (intraclass correlation coefficient [ICC] = 0.88–0.96) (15,20), sprint decrement (ICC = 0.75) (21), and physiological responses (ICC = 0.72–0.78) (20). The RS protocol was chosen to allow analyses of sprint phases through split times measured across 0–5, 5–15, and 15–20 m for each sprint and to represent typical sprint and recovery durations based on game-play observations made in team sports (1,9).

All subjects were familiar with sprinting activity encountered in their normal exercise routines and sporting experience. However, verbal explanation, demonstration, and submaximal attempts were used to familiarize subjects with the RS protocol before testing. Performance-based dependent variables were measured across each sprint split (0–5, 5–15, 15–20, and 0–20 m splits) and included (a) performance times across each sprint split for each sprint, (b) total time across the entire protocol, and (c) performance decrement across each sprint split for each sprint. Physiological-based dependent variables included (a) blood lactate concentration ($[BLa^-]$) measured before, immediately after, and 5 minutes post testing, (b) heart rate (HR) immediately before and after each sprint, and (c) rating of perceived exertion (RPE) immediately after each sprint.

Subjects

Nine sport science students volunteered to participate in this study (mean \pm standard deviation, age: 21.8 ± 3.6 years;

(range: 18–28 years) height: 171.3 ± 6.4 cm; body mass: 72.8 ± 12.2 kg; males: $n = 6$; females: $n = 3$). All subjects were meeting current activity guidelines (150 minutes per week of moderate-intensity activity or 75 minutes per week of vigorous-intensity activity or a relevant combination of these) (2) and were required to possess a recent participation history in intermittent team sports, at least at a competitive, recreational level (7). Subjects were prescreened for any health conditions or injuries that contraindicated participation and the study aims, procedures, risks, benefits, and the freedom to withdraw were thoroughly explained to all subjects before obtaining verbal and written consent. Body mass and height were recorded before testing using electronic scales (BWB-600; Tanita Corporation, Tokyo, Japan) and a digital stadiometer (model 274; Seca, Hamburg, Germany), respectively. Testing procedures were granted prior approval by an institutional human research ethics committee.

Procedures

A warm-up consisting of jogging at approximately 50% of maximal sprint speed for 5 minutes followed by a series of dynamic stretches was completed by each subject before each test. Dual-beam electronic timing lights (Fusion Sport, Cooper Plains, QLD, Australia) were used to assess RS performance, placed 5, 15, and 20 m after the initial set of timing lights on an indoor, sprung, hardwood surface. The positioning of each timing light was marked with tape to ensure consistent placement across sessions. Before testing, subjects were familiarized with the test protocol (10 × 20 m sprints) and completed 3 × 20 m reference sprints with 2 min recoveries to determine maximal sprint speed (8). The fastest sprint was used to calculate 50% of individual maximal speed for the active recovery intensity. The active recovery protocol involved subjects jogging for 27 s and returning to a stationary start position for each subsequent sprint at a marked line 30 cm before the initial set of timing lights (34). The distance to be covered during active recovery between bouts was calculated for each subject as $[(20 \text{ m}/\text{best reference sprint time [s]}) \times 50\% \times 27 \text{ s}]$. An individualized path was marked on the floor to ensure subjects covered the predefined distance at the relevant intensity and experienced a timed return to the start position after 27 s. Subjects were monitored during recovery using a timing device and verbally instructed to adjust speed if not moving at the specified intensity. The passive recovery protocol involved subjects decelerating after each sprint and then walking to the start position for each subsequent sprint, where they remained stationary (standing) (8). Across conditions, the start position alternated between ends of the sprint path, and a verbal countdown was provided 3 s before each sprint. In addition, the first sprint in each condition had to be performed at or faster than 95% of the maximal sprint speed attained during the reference sprints measured before testing (8). This approach was adopted to ensure adequate effort was

applied from the outset of testing, with all subjects fulfilling this criteria.

Performance times for each split (0–5, 5–15, 15–20, and 0–20 m) were calculated individually for each sprint ($\text{split}_{\text{sprint number}}$, e.g., 0–5₁) and 0–20 m time was summed across the entire protocol. Sprint decrement was accumulatively calculated for each split across each sprint following the initial bout using the following formula $([(\text{total time}/\text{ideal time}) \times 100] - 100)$, where ideal time was the best time multiplied by the number of sprints completed (20). This method has been reported to be the most reliable to assess fatigue during RS tests (20). Blood lactate concentration was determined using 30 μl capillary blood samples taken after the warm-up, immediately after the test protocol, and after 5 min of seated rest after test completion. Heart rate monitors (RS800CX; Polar Electro Oy, Kempele, Finland) were affixed to the torso of each subject before testing and discrete measurements were taken 2–3 s before ($\text{pre-HR}_{\text{sprint number}}$) and after ($\text{post-HR}_{\text{sprint number}}$) each sprint. Rating of perceived exertion was measured using a modified Borg scale (6–20) 2–3 s after each sprint ($\text{RPE}_{\text{sprint number}}$).

Statistical Analyses

The Shapiro-Wilks statistic and Levene's test for equality in variances were conducted for all data and confirmed normality and homogeneity of variances. Mixed analysis of variances were used to assess between- and within-condition differences in all outcome measures collected at various time points (sprint time, sprint decrement, $[\text{BLa}^-]$, HR, and RPE) with partial eta squared (η_p^2) determined to show the effect size (*small* = 0.01–0.03; *medium* = 0.06–0.09; *large* = >0.14). Mauchly's test was consulted and Greenhouse–Geisser corrections were applied if sphericity was not met. Where significant interactions were observed, simple main effect analyses were performed. Fisher's least significant difference post hoc tests were used to locate any significant differences. Conditional differences in total sprint time for each condition were assessed using paired sample *t*-tests. Effect sizes were also calculated for all pairwise comparisons between conditions using Cohen's *d* (*small* = 0.20–0.49; *medium* = 0.50–0.79; *large* = >0.80). An a priori power analysis using a two-tailed alpha value of 0.05, power of 0.80, and effect size of 0.80 based on previous research comparing recovery modalities during RS protocols (7,8,12) recommended a sample size of 7 (3) supporting the present analyses ($n = 9$). All statistical analyses were performed using IBM SPSS Statistics (v20.0; IBM Corporation, Armonk, NY, USA). Statistical significance was set at $p \leq 0.05$. All reported data are expressed as mean \pm SD.

RESULTS

Sprint Times

Total sprint times summed across all sprints were significantly different between conditions (active 37.73 ± 2.50 ,

TABLE 1. Sprint times and accumulated sprint decrement (mean \pm standard deviation) relative to interbout recovery modality across 0–5, 5–15, 15–20, and 0–20 m splits during 10 \times 20 m repeated sprints ($n = 9$).*

Outcome measure	Sprint split and bout number				
Active recovery					
0–5 m split	0–5 ₁	0–5 ₂	0–5 ₃	0–5 ₄	0–5 ₅
Time (s)	1.25 ± 0.11	1.26 ± 0.11	1.29 ± 0.12	1.32 ± 0.10	1.35 ± 0.12†
Decrement (%)	–	1.83 ± 1.78‡	3.01 ± 1.35‡	4.35 ± 2.19§	5.62 ± 3.26
5–15 m split	5–15 ₁	5–15 ₂	5–15 ₃	5–15 ₄	5–15 ₅
Time (s)	1.53 ± 0.10 ¶#	1.58 ± 0.10 ¶	1.63 ± 0.11†	1.64 ± 0.13†	1.73 ± 0.15†
Decrement (%)	–	2.11 ± 2.32**	3.82 ± 1.85†¶	4.80 ± 2.71†††	6.51 ± 3.63‡
15–20 m split	15–20 ₁	15–20 ₂	15–20 ₃	15–20 ₄	15–20 ₅
Time (s)	0.69 ± 0.06‡‡¶	0.74 ± 0.08¶	0.71 ± 0.08 ¶	0.78 ± 0.08†	0.81 ± 0.10†
Decrement (%)	–	3.27 ± 3.52**	6.17 ± 5.6¶	8.67 ± 6.9††	11.20 ± 7.67
0–20 m split	0–20 ₁	0–20 ₂	0–20 ₃	0–20 ₄	0–20 ₅
Time (s)	3.47 ± 0.25‡‡	3.57 ± 0.25**	3.64 ± 0.27§§	3.74 ± 0.30	3.89 ± 0.34†
Decrement (%)	–	1.88 ± 1.44**	3.10 ± 1.56§	4.41 ± 2.56†††	6.11 ± 3.72†
Passive recovery					
0–5 m split	0–5 ₁	0–5 ₂	0–5 ₃	0–5 ₄	0–5 ₅
Time (s)	1.25 ± 0.11	1.26 ± 0.12	1.30 ± 0.14	1.26 ± 0.07	1.25 ± 0.07
Decrement (%)	–	2.03 ± 1.61	3.54 ± 2.41	4.62 ± 2.75	4.47 ± 2.07
5–15 m split	5–15 ₁	5–15 ₂	5–15 ₃	5–15 ₄	5–15 ₅
Time (s)	1.54 ± 0.07	1.54 ± 0.09	1.54 ± 0.10	1.53 ± 0.10	1.53 ± 0.10
Decrement (%)	–	0.96 ± 0.81‡	1.74 ± 0.91	2.23 ± 1.21	2.09 ± 0.87
15–20 m split	15–20 ₁	15–20 ₂	15–20 ₃	15–20 ₄	15–20 ₅
Time (s)	0.69 ± 0.05	0.70 ± 0.06	0.69 ± 0.06	0.68 ± 0.06	0.68 ± 0.08
Decrement (%)	–	2.22 ± 2.08	3.85 ± 3.51	5.33 ± 5.26	6.52 ± 5.70
0–20 m split	0–20 ₁	0–20 ₂	0–20 ₃	0–20 ₄	0–20 ₅
Time (s)	3.48 ± 0.20	3.49 ± 0.25	3.52 ± 0.25	3.47 ± 0.18	3.46 ± 0.20
Decrement (%)	–	0.85 ± 0.84	1.86 ± 1.47	1.99 ± 1.45	2.04 ± 1.34

Outcome measure	Sprint split and bout number				
Active recovery					
0–5 m split	0–5 ₆	0–5 ₇	0–5 ₈	0–5 ₉	0–5 ₁₀
Time (s)	1.35 ± 0.10	1.35 ± 0.08†	1.34 ± 0.07†	1.33 ± 0.08	1.36 ± 0.08†
Decrement (%)	6.84 ± 3.05	7.46 ± 3.41	7.79 ± 3.49	7.97 ± 3.39	8.49 ± 3.41†
5–15 m split	5–15 ₆	5–15 ₇	5–15 ₈	5–15 ₉	5–15 ₁₀
Time (s)	1.68 ± 0.13†	1.72 ± 0.12†	1.73 ± 0.12†	1.74 ± 0.13†	1.72 ± 0.15†
Decrement (%)	7.10 ± 4.04†	7.94 ± 4.29†	8.63 ± 4.42†	9.29 ± 4.60†	9.65 ± 5.11†
15–20 m split	15–20 ₆	15–20 ₇	15–20 ₈	15–20 ₉	15–20 ₁₀
Time (s)	0.79 ± 0.08†	0.82 ± 0.07†	0.83 ± 0.10†	0.83 ± 0.09†	0.84 ± 0.12†
Decrement (%)	12.31 ± 8.00	13.64 ± 8.06†	14.79 ± 8.00†	15.65 ± 7.97†	16.57 ± 8.93†
0–20 m split	0–20 ₆	0–20 ₇	0–20 ₈	0–20 ₉	0–20 ₁₀
Time (s)	3.82 ± 0.29†	3.89 ± 0.26†	3.89 ± 0.27†	3.90 ± 0.28†	3.91 ± 0.30†
Decrement (%)	6.88 ± 4.09†	7.73 ± 4.42†	8.38 ± 4.58†	8.91 ± 4.69†	9.39 ± 5.22†
Passive recovery					
0–5 m split	0–5 ₆	0–5 ₇	0–5 ₈	0–5 ₉	0–5 ₁₀
Time (s)	1.28 ± 0.10	1.28 ± 0.11	1.26 ± 0.08	1.30 ± 0.12	1.29 ± 0.09
Decrement (%)	4.71 ± 2.08	4.87 ± 2.19	4.83 ± 1.92	5.12 ± 2.15	5.26 ± 2.20
5–15 m split	5–15 ₆	5–15 ₇	5–15 ₈	5–15 ₉	5–15 ₁₀
Time (s)	1.53 ± 0.09	1.54 ± 0.10	1.54 ± 0.10	1.56 ± 0.10	1.55 ± 0.08
Decrement (%)	2.14 ± 0.77	2.24 ± 0.74	2.37 ± 0.71	2.52 ± 0.72	2.60 ± 0.73
15–20 m split	15–20 ₆	15–20 ₇	15–20 ₈	15–20 ₉	15–20 ₁₀
Time (s)	0.70 ± 0.06	0.68 ± 0.08	0.70 ± 0.05	0.69 ± 0.07	0.70 ± 0.04
Decrement (%)	6.90 ± 6.05	6.75 ± 5.40	7.00 ± 5.29	7.06 ± 5.01	7.20 ± 5.25

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0–20 m split Time (s)	0–20 ₆ 3.51 ± 0.23	0–20 ₇ 3.51 ± 0.26	0–20 ₈ 3.51 ± 0.19	0–20 ₉ 3.55 ± 0.25	0–20 ₁₀ 3.53 ± 0.18
Decrement (%)	2.22 ± 1.33	2.29 ± 1.23	2.38 ± 1.15	2.55 ± 1.18	2.69 ± 1.22

*All significance is at $p \leq 0.05$.

†Significantly greater than passive recovery.

‡Significantly lower than sprints 4–10.

§Significantly lower than sprints 6–10.

||Significantly lower than sprint 5.

¶Significantly lower than sprints 7–10.

#Significantly lower than sprint 6.

**Significantly lower than sprints 5–10.

††Significantly lower than sprints 8–10.

‡‡Significantly lower than sprint 4.

§§Significantly lower than sprints 9–10.

|||Significantly lower than sprint 10.

passive 35.02 ± 2.10 seconds, $p = 0.002$; $d = 1.17$, *large*). Mean ($\pm SD$) performance times and decrements across each split (0–5, 5–15, 15–20, and 0–20 m) for all sprints (1–10) in each condition are shown in Table 1. Significant interactions between condition and time were found for 0–5 m ($F_9 = 2.20$, $p = 0.025$; $\eta_p^2 = 0.21$, *large*), 5–15 m ($F_9 = 8.99$, $p < 0.001$; $\eta_p^2 = 0.36$, *large*), 15–20 m ($F_9 = 5.89$, $p < 0.001$; $\eta_p^2 = 0.28$, *large*), and 0–20 m ($F_9 = 8.62$, $p < 0.001$; $\eta_p^2 = 0.35$, *large*) split times. Main effect analyses revealed a significantly faster time in passive vs. active recovery during 0–5_{7–10} ($p \leq 0.042$; $d = 0.73$ – 1.06 , *moderate-large*), 5–15_{3–10} ($p \leq 0.013$; $d = 0.86$ – 1.72 , *large*), 15–20_{4–10} ($p \leq 0.008$; $d = 1.27$ – 1.86 , *large*), 15–20_{4–10} ($p \leq 0.008$; $d = 1.27$ – 1.86 , *large*), and 0–20_{5–10} ($p \leq 0.005$; $d = 1.18$ – 1.63 , *large*). Temporal comparisons showed significantly different sprint times between bouts only in active recovery. More precisely, significantly ($p \leq 0.021$) faster sprint times were observed during 5–15_{1,2} vs. 5–15_{5,7–10}, and 5–15₁ vs. 5–15₆. Similarly, significantly ($p \leq 0.050$) faster sprint times were evident during 15–20_{1–3} vs. 15–20_{7–10}, 15–20₁ vs. 15–20₄, and 15–20₃ vs. 15–20₅. Faster sprint times were also apparent during 0–20₁ vs. 0–20_{4–10}, 0–20₂ vs. 0–20_{5–10}, and 0–20₃ vs. 0–20_{9–10}.

Sprint Decrement

Significant interactions were observed between condition and time for sprint decrement across 0–5 m ($F_8 = 7.11$, $p = 0.002$; $\eta_p^2 = 0.31$, *large*), 5–15 m ($F_8 = 18.92$, $p < 0.001$; $\eta_p^2 = 0.54$, *large*), 15–20 m ($F_8 = 4.57$, $p = 0.018$; $\eta_p^2 = 0.22$, *large*), and

0–20 m ($F_8 = 15.69$, $p < 0.001$; $\eta_p^2 = 0.50$, *large*) splits. Main effect analyses showed a significantly lower sprint decrement during passive vs. active recovery across 0–5₁₀ ($p = 0.028$; $d = 1.13$, *large*), 5–15_{3–10} ($p \leq 0.045$, $d = 1.22$ – 2.06 , *large*), 15–20_{7–10} ($p \leq 0.029$, $d = 1.00$ – 1.29 , *large*), and 0–20_{4–10} ($p \leq 0.039$, $d = 1.16$ – 1.86 , *large*). Temporal comparisons demonstrated a significantly ($p \leq 0.027$) lower sprint decrement across 0–5_{2–3} vs. 0–5_{4–10}, and 0–5₄ vs. 0–5_{6–10} during active recovery. A significantly lower decrement was evident during 5–15₂ vs. 5–15_{4–10}, and 5–15₃ vs. 5–15₁₀ in passive recovery ($p \leq 0.034$), and 5–15₂ vs. 5–15_{5–10}, 5–15₃ vs. 5–15_{7–10}, and 5–15₄ vs. 5–15_{8–10} during active recovery ($p \leq 0.036$). A significantly ($p \leq 0.049$) lower sprint decrement was also apparent during 15–20₂ vs. 15–20_{5–10}, 15–20₃ vs. 15–20_{7–10}, and 15–20₄ vs. 15–20_{8–10} only in active recovery. Similarly, a significantly ($p \leq 0.039$) lower sprint decrement was observed

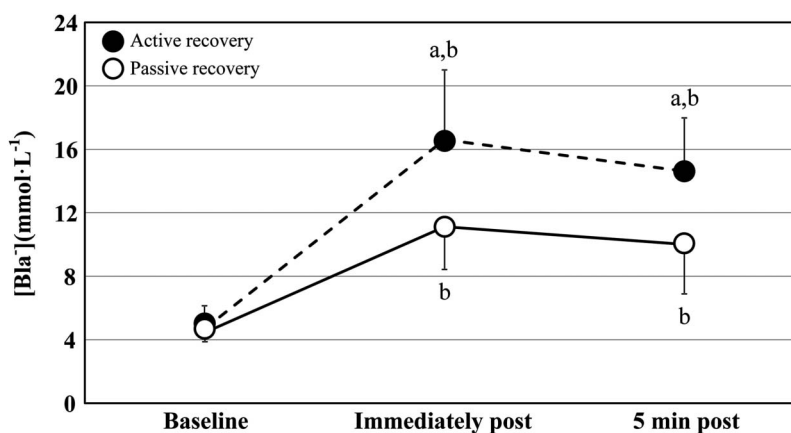


Figure 1. Blood lactate concentration ([Bla⁻]) responses during active and passive recovery performed between bouts during 10 × 20 m repeated sprints ($n = 9$). Note: a) significantly greater than passive recovery ($p \leq 0.005$); b) significantly greater than baseline measure ($p < 0.001$).

during 0–20₂ vs. 0–20_{5–10}, 0–20₃ vs. 0–20_{6–10}, and 0–20₄ vs. 0–20_{8–10} when completing active recovery.

Blood Lactate Concentration

Mean (\pm SD) [BLa[−]] at each time point in each condition is displayed in Figure 1. A significant interaction between con-

dition and time ($F_9 = 4.96$, $p = 0.013$; $\eta_p^2 = 0.24$, *large*) was found. Subsequent main effect analyses revealed significantly higher (BLa[−]) in active vs. passive recovery immediately ($p = 0.005$; $d = 1.51$, *large*) and 5 minutes ($p = 0.002$; $d = 1.42$, *large*) after test completion. A significantly ($p < 0.001$) elevated (BLa[−]) was evident immediately and 5 minutes

TABLE 2. Heart rate (HR) and rate of perceived exertion (RPE) (mean \pm standard deviation) relative to interbout recovery modality across each bout during 10 \times 20 m repeated sprints ($n = 9$).^{*,†}

Outcome measure	Sprint bout number				
	1	2	3	4	5
Active recovery					
Presprint HR (b·min ^{−1})	114.1 \pm 15.8 _‡	156.3 \pm 15.6 _‡	169.2 \pm 12.5 _‡	175.7 \pm 12.9 _§	178.6 \pm 11.7 _§
Postsprint HR (b·min ^{−1})	124.7 \pm 15.7 _‡	163.1 \pm 14.1 _‡	171.4 \pm 13.3 _{§‡}	176.3 \pm 12.7 _§	179.2 \pm 12.1 _§
RPE (AU)	7.9 \pm 1.8 _{††}	9.4 \pm 2.5 _{‡‡}	12.0 \pm 2.8 _{††}	13.3 \pm 2.4 _{§††‡‡}	14.6 \pm 2.2 _{§§§§ #}
Passive recovery					
Presprint HR (b·min ^{−1})	107.4 \pm 13.8	143.7 \pm 12.0	152.6 \pm 11.0	159.0 \pm 12.3	161.7 \pm 12.0
Postsprint HR (b·min ^{−1})	114.9 \pm 13.2	147.0 \pm 10.4	153.0 \pm 12.2	157.6 \pm 12.7 _‡	160.9 \pm 11.6
RPE (AU)	8.0 \pm 1.7 _{††}	8.9 \pm 1.4 _{‡‡}	10.3 \pm 1.4 _{‡‡}	11.0 \pm 1.1 _{§§ #}	11.9 \pm 1.7 _{§§ #}
Outcome measure	Sprint bout number				
	6	7	8	9	10
Active recovery					
Presprint HR (b·min ^{−1})	181.4 \pm 11.4 _{§ #**}	182.0 \pm 10.4 _{§ #**}	183.6 \pm 9.9 _§	185.0 \pm 8.6 _{§**}	185.6 \pm 8.7 _§
Postsprint HR (b·min ^{−1})	181.9 \pm 11.2 _{§ #**}	183.2 \pm 10.7 _{§#**}	183.9 \pm 10.0 _{§**}	185.2 \pm 9.2 _§	187.0 \pm 8.0 _§
RPE (AU)	16.2 \pm 1.8 _{§#}	16.3 \pm 2.5 _{§#}	17.6 \pm 1.7 _§	17.2 \pm 2.2 _§	18.4 \pm 1.3 _§
Passive recovery					
Presprint HR (b·min ^{−1})	164.3 \pm 12.0 _{§§ #**}	166.0 \pm 11.7 _#	166.4 \pm 11.9	168.7 \pm 12.7	169.1 \pm 11.7
Postsprint HR (b·min ^{−1})	164.0 \pm 12.1 _{§§ #**}	165.2 \pm 13.0 _{§§ #**}	167.2 \pm 12.1	165.6 \pm 15.1	168.9 \pm 11.3
RPE (AU)	12.3 \pm 1.9 _{§§ #}	13.0 \pm 1.8 _#	13.8 \pm 1.9	14.9 \pm 2.2	15.2 \pm 2.1

*AU = arbitrary units.

†All significance is at $p \leq 0.05$.

‡Significantly different from all other sprints in same condition.

§Significantly greater than passive recovery.

||Significantly different from all other sprints in same condition except corresponding pre-/post-sprint.

††Significantly different from pre-HR₉/RPE₉.

#Significantly different from pre-HR₁₀/RPE₁₀.

**Significantly different from post-HR₁₀ ($p \leq 0.05$).

†††Significantly different from all sprints except the subsequent sprint.

‡‡Significantly different from all sprints except the previous sprint.

§§Significantly different from post-HR₉/RPE₉.

||||Significantly different from all sprints in same condition except post-HR₅.

||†Significantly different to pre-HR₇/RPE₇.

after test completion vs. baseline measures in both conditions.

Heart Rate

Mean (\pm SD) HR and RPE responses for all sprints in each condition are presented in Table 2. Significant condition ($F_1 = 9.93$, $p = 0.006$; $\eta_p^2 = 0.38$, *large*) and time ($F_{19} = 212.07$, $p < 0.001$; $\eta_p^2 = 0.93$, *large*) effects were apparent for HR response. Post hoc analyses showed active recovery promoted significantly higher HR responses from post-HR₃ onwards ($p \leq 0.014$; $d = 1.33$ – 1.57 , *large*). Temporal comparisons showed pre-HR₁₋₃ and post-HR₁₋₃ were significantly ($p \leq 0.042$) different from all other HR measures during active recovery, whereas pre-HR₄₋₅ and post-HR₄₋₅ were significantly ($p \leq 0.042$) different from all other measures except the corresponding pre- or post-sprint measure for that bout. Pre-HR₆₋₇ and post-HR₆ were significantly ($p \leq 0.026$) lower than pre-HR₉₋₁₀ and post-HR₁₀. In addition, post-HR₇ was significantly ($p = 0.020$) lower than pre-HR₁₀ and post-HR₁₀, whereas post-HR₈ and pre-HR₉ were significantly ($p \leq 0.048$) lower than post-HR₁₀. During passive recovery, pre-HR_{1-3,5} and post-HR_{1-3,5} were significantly ($p \leq 0.019$) different from all other measures in the same condition except the corresponding pre- or post-sprint measure for that bout. Pre-HR₄ was significantly different from all other measures ($p \leq 0.019$) except post-HR₅, whereas post-HR₄ was significantly ($p \leq 0.019$) different from all other measures. Pre-HR₆ and post-HR₆ were significantly ($p \leq 0.036$) lower than pre-HR_{7,9,10} and post-HR_{8,10}. Furthermore, pre-HR₇ was significantly ($p \leq 0.021$) lower than pre-HR_{9,10}, whereas post-HR₇ was significantly ($p \leq 0.030$) lower than post-HR_{8,10} and pre-HR_{9,10}.

Rating of Perceived Exertion

A significant interaction between condition and time ($F_9 = 4.96$, $p = 0.013$; $\eta_p^2 = 0.24$, *large*) was observed for RPE. Main effect analyses revealed significantly ($p \leq 0.009$; $d = 1.05$ – 2.11 , *large*) higher RPE values during active vs. passive recovery from RPE₄ onwards. Temporal analyses showed RPE significantly ($p \leq 0.042$) increased across all sprints in active recovery except between RPE₁₋₂, RPE₃₋₄, RPE₄₋₅, RPE₅₋₇, RPE_{6,8,9}, RPE₇₋₉, and RPE₈₋₁₀. Similarly, RPE significantly ($p \leq 0.024$) increased across all sprints during passive recovery except between RPE₁₋₂, RPE₂₋₃, RPE₃₋₄, RPE₄₋₆, RPE₅₋₇, RPE_{7,8}, and RPE₈₋₁₀.

DISCUSSION

This study provides novel insight regarding the influence of interbout recovery modality on short distance (≤ 20 m), run-based RS performance representative of team sport demands (1,16,33,34) during field-based settings. Furthermore, the provided data are the first detailing performance differences between recovery modalities across sprint phases using various splits (0–5, 5–15, 15–20, and 0–20 m) during run-based RS. Passive recovery promoted superior sprint times and performance maintenance compared with active recovery,

particularly across the 5–15, 15–20, and 0–20 m splits. Furthermore, the better performance during passive recovery was accompanied by reduced blood lactate accumulation, cardiovascular demands, and perceived exertion.

Our findings concur with previous research examining active and passive recovery approaches during run-based RS across longer bouts (15 seconds and 30 m) (8,12) and using treadmill protocols (7). Previous studies have shown passive recovery to promote superior RS performance with greater average speed (7), distance (7,12), and time to exhaustion (12), as well as reduced sprint decrement (7,8) and performance times (8) observed across varied protocols. Reduced RS performance with active recovery has been attributed to various sources, including limitations in energy provision (18) because of an increased O₂ requirement in working musculature to complete submaximal workloads between sprinting bouts (12). In turn, given the reliance on oxidative metabolism for PCr restoration (27), the reduced O₂ demand during passive recovery likely allows for increased PCr availability across sprints. Phosphocreatine degradation is an important energy source for ATP production across RS, being reported to comprise 50 and 80% of the anaerobic contribution across the first and final bouts during a 10 × 6 s RS cycling protocol, respectively (17). In addition, greater O₂ availability might promote improved reloading of myoglobin and hemoglobin for oxidative metabolism (12). This suggestion is supported by previous observations that showed greater deoxyhemoglobin levels (94.4 ± 16.7 vs. $83.4 \pm 4.7\%$) in the *vastus lateralis* muscle during active recovery compared with passive recovery across run-based RS (7). This response is particularly important for performance maintenance across RS given latter bouts have been shown to rely more extensively on oxidative metabolism than initial bouts (4,26). Based on the established relationship between HR- $\dot{V}O_2$ during submaximal and intermittent exercise (6), the higher HR responses we observed during active recovery support the notion of an increased O₂ requirement.

The higher HR and potential O₂ cost of active recovery in this study might also explain other physiological responses observed. More precisely, elevated (BLa⁻) immediately and 5 min post test as well as RPE from the fourth bout onwards were evident with active recovery. Consequently, the reduced metabolic load experienced during passive recovery might have permitted greater lactate oxidation compared with active recovery, likely carrying an augmented O₂ demand (8,12). This response might have promoted less lactate accumulation with sprint progression, predisposing to lower (BLa⁻) after the test protocol in the passive recovery condition. Furthermore, given, HR and (BLa⁻) have been shown to be significant determinants ($R^2 = 0.58$, $p < 0.001$) of perceived exertion during intermittent activity (10), the concomitant increase in RPE we observed during active recovery can be expected. However, other physiological factors such as metabolic

acidosis, hormone concentrations, and body temperature might have also contributed to the observed RPE response (31), and warrant further investigation across different recovery modalities.

Though the present physiological data provide useful insight into potential mechanisms underlying the observed performance differences, equivocal findings are apparent in the wider literature. In line with our results, Buchheit et al. (7) observed elevated HR (160 ± 8 vs. 155 ± 8 b·min⁻¹), $\dot{V}O_2$ (3.64 ± 0.44 vs. 2.91 ± 0.47 L·min⁻¹), and post-test [BLa⁻] at 5 min (13.5 ± 2.5 vs. 12.7 ± 2.2 mmol·L⁻¹) during active recovery compared with passive recovery across 6×4 s run-based RS (7). In contrast, Castagna et al. (8) recorded no significant between-condition difference in post-test (BLa⁻) at 3 min (active: 13.2 ± 2.9 mmol·L⁻¹; passive: 14.1 ± 2.9 mmol·L⁻¹) during 10×30 m run-based shuttle sprints. Likewise, Dupont et al. (12) reported no significant between-condition difference in HR (active: 174 ± 9 ; passive: 176 ± 8 b·min⁻¹), $\dot{V}O_2$ (active: 51.6 ± 6.5 ; passive: 49.4 ± 6.8 ml·kg⁻¹·min⁻¹) or post-test (BLa⁻) (within 15 min) (active: 10.7 ± 2.0 ; passive: 11.7 ± 2.1 mmol·L⁻¹) across 15 s run-based RS until exhaustion. The inconsistent observations in the literature might be attributed to methodological variations across studies (7,8,12). Specifically, varied sprint protocols were used with different bout frequencies (6-exhaustion), durations (4–15 s), and distances (15–30 m) (7,8,12) which have been shown to influence the physiological demands relative to recovery modality during RS in swimming (37–39) and cycling (22). Given the active recovery intensity and that sprint distances presently used mostly resemble those adopted by Buchheit et al. (7), we propose that the physiological demands of shorter (≈ 3.5 –4 s or 15–20 m) run-based RS might be more heavily influenced by recovery modality than longer sprints (≈ 15 s or 30 m). Indeed, shorter RS protocols (15 vs. 40 m) have been shown to elicit greater HR and RPE possibly through more rapid decelerations and eccentric muscle action (24), potentially eliciting greater accumulative physiological loads when combined with active recovery compared with longer sprints. Given the limited investigation in this area, further research is encouraged directly comparing recovery modalities across run-based RS using varied sprint configurations.

Previous run-based research has largely examined the effects of recovery modalities during RS globally across protocols, with limited available research reporting on temporal changes across individual sprints (7,8). Previously, Castagna et al. (8) reported significant diminished performance from sprints 4 and 7 onwards during active and passive recovery, respectively, across 10×30 m run-based shuttle sprints with 30 s recoveries. Moreover, Buchheit et al. (7) highlighted a significant between-condition difference only during the final sprint across a 6×4 s run-based RS with 21 s recovery. We provide more extensive data regarding time-course changes, with sprint times and decre-

ments (across splits) being observed to diminish primarily after sprints 2 and 3 (across 10×20 m) and mostly during active recovery. Furthermore, the improved sprint times and decrements with passive recovery compared with active recovery typically manifested after sprints 3 and 4 across 5–15 m, 15–20 m, and 0–20 m splits, with differences not being identified until sprints 7 and 10 for the 0–5 split. These findings suggest that earlier performance declines might develop during active recovery when using shorter run-based RS than previously thought using field-based data reported across longer sprints (30 m) (8).

Our study also provides first insight into the effects of active and passive recoveries during run-based RS according to sprint splits. The superior performance responses accompanying passive recovery and the greater temporal decrements with active recovery were mostly evident across later sprint splits (5–15 and 15–20 m) and the entire sprint bout (0–20 m). Minimal between- and within-condition differences were apparent for 0–5 m sprint time and decrement. Early acceleration phases across 5–10 m have been proposed to hold great importance during team sport game-play given many of these efforts tend to occur in crucial competitive scenarios (e.g., evading opponents, gaining possession) (30). As such, the present data suggest that recovery modality and fatigue have less influence on this sprint phase. These observations might in part be due to the interplay between recovery modality, stride frequency, and fatigue. Previously, stride frequency has been shown to be a nonsignificant discriminating factor for early acceleration performance across 0–5 m during sprinting (25), becoming more important across longer sprint phases (0–25 m) (25,28). In turn, reduced stride frequencies have been reported with active recovery compared with passive recovery (1.95 ± 0.19 vs. 1.84 ± 0.22 Hz, $p < 0.001$) during 15 m RS (7) and with sprint-induced fatigue across 6 s RS (4.10 ± 0.21 vs. 3.61 ± 0.26 Hz, $p < 0.001$) (29). Thus the kinematic detriments to stride frequency accompanying active recovery and fatigue might not be as pronounced across early acceleration phases. Given stride frequency was not measured in this study, further research is recommended to confirm this postulation.

In conclusion, the present data indicate passive recovery promoted superior performance across short distance, run-based RS. Temporal analyses also showed earlier performance deterioration with active recovery. These observations might be due to an increased O₂ requirement by working musculature during active recovery, as evidenced by an increased physiological cost ([BLa⁻], HR, and RPE). In addition, early acceleration sprint performance across 0–5 m appeared more resistant to fatigue and homogenous across recovery modalities than later sprint phases.

PRACTICAL APPLICATIONS

Although our data are representative of the included student sample, the relevance of the RS protocol used in various

team sports (e.g., soccer, basketball, rugby league) (1,16,33,34) suggests that the findings may hold benefit for wider application. As such, this study provides useful information for personal trainers, strength and conditioning professionals, and sports coaches regarding training approaches, tactical strategies, and optimization of game performance.

Training drills to improve health and fitness in the general population and athletes regularly incorporate RS (36). Consequently, personal trainers, and strength and conditioning professionals should be aware of the added physiological cost associated with short-distance RS drills that incorporate active recovery. To increase the metabolic demands of the training stimulus and promote physiological overload, personal trainers, and strength and conditioning professionals might incorporate low-moderate intensity movements between sprinting bouts when implementing RS training drills for clients. However, the administration of these types of drills should be done safely and for low-risk clients, given the heavy demands experienced.

In opposition, the detrimental effects of active recovery between RS indicate that athletes required to execute low-moderate intensity movements between high-intensity efforts may be less likely to maintain sprint performance across game-play. Indeed, our findings suggest that performance deteriorates earlier (after 2 bouts in some measures) across shorter RS incorporating active recovery than previously shown across longer sprints (8). Team sport coaches should be aware of these temporal fatigue patterns, and develop tactical strategies (e.g., substitutions, time-outs, adjustments in game pace, sparing behavior) (8) to provide enhanced recovery between high-intensity efforts, and ensure athletes are able to optimally execute sprints between 5 and 20 m at crucial stages of game-play.

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