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Jump height loss as an indicator of fatigue during sprint training

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ABSTRACT

This study analysed the acute mechanical and metabolic responses to a sprint training session focused on maintaining maximal speed until a given speed loss was reached. Nine male high-level sprinters performed 60 m running sprints up to a 3% in speed loss with 6 min rests between sets. Mechanical responses (countermovement jump (CMJ) height and speed loss) and metabolic responses (blood lactate and ammonia concentrations) were measured pre-exercise and after each set was performed. Jump height loss showed almost perfect relationships with both lactate (r = 0.91) and ammonia (r = 0.91) concentrations. In addition, nearly perfect relationships were observed for each athlete between CMJ height loss and lactate (r = 0.93–0.99) and ammonia (r = 0.94–0.99). Very large correlations were found between speed loss and lactate (r = 0.83), and ammonia (r = 0.86) concentrations. Furthermore, close relationships were observed for each athlete between speed loss and lactate (r = 0.86–0.99), and ammonia (r = 0.88–0.98). These results suggest that the CMJ test may allow more accurate setting of training loads in sprint training sessions, by using an individualised sprint dose based on mechanical and physiological responses rather than a standard fixed number of sprints for all athletes.

ARTICLE HISTORY
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KEYWORDS
Countermovement jump; lactate; ammonia; sprint monitoring; neuromuscular fatigue

Introduction

Sprint ability is a key factor in many sports and is the focus of many training programs (Faude, Koch, & Meyer, 2012; Haugen & Buchheit, 2016). The ability to produce a large forward acceleration with a high maximum running velocity, and to maintain that velocity, contribute to successful performance in a sprint race (Morin et al., 2015; Slawinski et al., 2017). Maximum running velocity in elite sprinters is achieved between 40 and 60 m into the race (Mero, Komi, & Gregor, 1992). This maximal intensity action requires very high energy production in just a few seconds. Metabolic energy is provided mainly by anaerobic glycolysis and phosphocreatine (PCr) metabolism in skeletal muscle cells. Therefore, PCr stores are vitally important in sprint performance, and are severely depleted after 5–7 s of sprinting (Hirvonen, Rehunen, Rusko, & Härkönen, 1987). During a longer distance sprint, such as a 100 m track and field event, anaerobic glycolysis provides the bulk of the adenosine triphosphate (ATP) needed to complete the sprint with minimal impairment of velocity (55–75% of metabolic energy) (Dawson et al., 1997; Hautier et al., 1994; Hirvonen et al., 1987). However, when sprints must be repeated, during competition or training sessions, this may lead to a significant reduction in PCr and ATP concentration and an accumulated loss of adenine nucleotides (Balsom, Seger, Sjödin, & Ekblom, 1992). This large ATP depletion may require a long recovery time and cause impairment in muscle force production (Gorostiaga et al., 2012). In addition, from a metabolic point of view, there are some plausible explanations for fatigue as a result of hydrogen ion (H^+) accumulation and increase in inorganic phosphate (Pi) levels (Allen, Lamb, & Westerblad, 2008). Likewise, an increase in blood ammonia level during short-term, high-intensity exercise is usually interpreted as indicative of accelerated ammonia production in muscles, resulting from the deamination of AMP to IMP. The purine nucleotide cycle (PNC) serves, among other functions, to maintain a high ATP/ADP ratio (Hellsten-Westing, Norman, Balsom, & Sjödin, 1993) and acts as an emergency mechanism to prevent muscle ATP from falling to critical levels under conditions of high metabolic stress. Therefore, knowledge of changes in blood lactate and ammonia concentrations during training sessions would provide valuable information about the physiological stress induced.

Blood lactate and ammonia measurements are expensive and invasive techniques, which means they are not feasible during regular training. However, relationships (r = 0.85–0.96) have been observed between these blood metabolites (lactate and ammonia) and jump height loss during resistance training (Sánchez-Medina & González-Badillo, 2011), typical 400 m running sessions (Gorostiaga et al., 2010), and repeated short sprints with brief (30 s) (Morcillo et al., 2015) and medium recovery times (4 min) (Jimenez-Reyes et al., 2016). These strong correlations (0.92–0.97) observed between jump height loss and blood lactate and ammonia support the use of jump height for monitoring the fatigue induced in training sessions (Gorostiaga et al., 2010; Jimenez-Reyes et al., 2016; Morcillo

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et al., 2015; Sánchez-Medina & González-Badillo, 2011). However, little is known about the relationships between jump height loss and metabolite concentration during a typical sprint session, including the maximal velocity phase (~ 60 m), and the theoretical full recovery period.

Neuromuscular fatigue has been described as any exercise-induced reduction in the maximal voluntary force or power produced by a muscle or muscle group (Gandevia, 2001). Most previous studies that examined mechanical and metabolic responses to repeated sprints used protocols with a fixed number of sprints and recovery periods shorter than one minute (Bishop & Spencer, 2004; Dawson et al., 1997). Fixing the number of sprints induces great variability between athletes in terms of the increments in running times (45.3 ± 9.7%), which is expressed as the fatigue index (FI) induced by the exercise (J.-B. Morin, Dupuy, & Samozino, 2011). It has been suggested that determining a given level of fatigue induces a more homogeneous response (Morin et al., 2011), which might be useful in protocols aimed at studying fatigue during training sessions. Indeed, previous studies in resistance training have used a similar approach, setting a target fatigue level (performance impairment) rather than a target number of repetitions (Pareja-Blanco, Rodríguez-Rosell, et al., 2017; Pareja-Blanco, Sánchez-Medina, Suárez-Arrones, & González-Badillo, 2017).

In the absence of direct laboratory-based measurements, it could be useful to obtain information about acute responses through an actual field-based measurement in order to obtain practical information related to neuromuscular fatigue. This would allow coaches to make real-time decisions using parameters that are easier to monitor during a sprint training session, such as speed or jump height losses. For instance, in strength training it has been verified that velocity loss is a good marker of neuromuscular fatigue (Sánchez-Medina & González-Badillo, 2011) as is performance in the countermovement jump (CMJ) (Cormack, Newton, McGuigan, & Cormie, 2008; Sánchez-Medina & González-Badillo, 2011). This approach would begin to build a scientifically based training method to individualize the sprint load prescription, using a variable that expresses performance impairment and its relationship with physiological responses. Therefore, the aims of the present study were: 1) to analyze the acute mechanical (speed and jump height loss) and metabolic (blood lactate and ammonia) responses to typical sprint training focused on maximal speed, performed until each athlete achieved the same speed loss (3%); and 2) to determine whether mechanical or metabolic responses could be used to monitor fatigue during a sprint training session.

Method

Subjects

Nine male athletes at national and international level (age: 23.1 ± 4.4 years, body mass: 73.7 ± 4.6 kg, height: 177.6 ± 5.9 cm; body fat: 9.6 ± 2.9%) participated in this study. Their best performances over 100 m were in the range 10.29–11.17 s (seven of them ran under 11.00 s in 100 m races) and they had been competing for at least 5 seasons. All of them were highly trained and familiarized with the testing exercises. One of them was the national record holder over 200 m (20.47 s) at the time of the measurements. No physical limitations or musculoskeletal injuries that could affect testing were reported. All participants were fully informed about the procedures, potential risks and benefits of the study and they all signed written informed consents prior to the tests. The present investigation met the ethical standards of this journal and was approved by the Ethics Committee of Pablo de Olavide University, Seville, Spain.

Experimental design

Athletes performed bouts of 60 m sprints at the highest possible speed, with a recovery period of 6 min between attempts, until there was a 3% decrease from the best time recorded for each athlete during the training session. We used a percentage-based FI score to individualize the prescription for sprint loads and attenuate the increased variability seen in fixed sprint protocols (Morin et al., 2011). An FI target of 3% was chosen in the present study since the athletes were specialists in short distances (maximum 200 m). Thus, a high FI was not advisable for improving their performance. Furthermore, a previous study that analysed the acute response to typical sprint training session using 40 m sprint distance established a level of 3% of FI (Jimenez-Reyes et al., 2016). In addition, 10 s after each sprint, a CMJ was performed. Blood lactate and ammonia concentrations were measured 1 min after each bout (Figure 1). Testing sessions were always carried out after a full resting day, at the same time of day (18:00–20:00 h) and under similar environmental conditions (20-22°C and 55–65% humidity). A standardised warm-up protocol was used, consisting of: 1) 5 min of running at a self-selected easy pace; 2) 5 min of joint mobilization exercises; 3) two 30 m running accelerations; 4) one 50 m running acceleration; 5) five CMJs with increasing intensity.

Measures

60 m sprint time

Sprint times were recorded for 60 m distances using photocell timing gates (Polifemo Radio Light, Microgate, Bolzano, Italy). The sprint test was conducted on a synthetic outdoor running track (Mondo, Turin, Italy). A standing start with the lead-off foot placed 1 m behind the first timing gate was used. The bouts were separated by 6 min for recovery. The sprint was standardised as follows:

Participants used a standard crouched position start with the toes of their preferred leg behind the start line. Once in position, participants were instructed to lean forward and hold their body mass over their forward leg, where they were then given a "3–2-1-go" countdown. Athletes were prompted to accelerate maximally, and to attempt to cover the sprint distance as fast as possible. Wind speed was less than 1.5 $m \cdot s^{-1}$, and wind conditions were monitored constantly using an Oregon Scientific WMR-918 (Oregon Scientific, Tigard, OR, USA) meteorological station. Test-retest reliability of 60 m

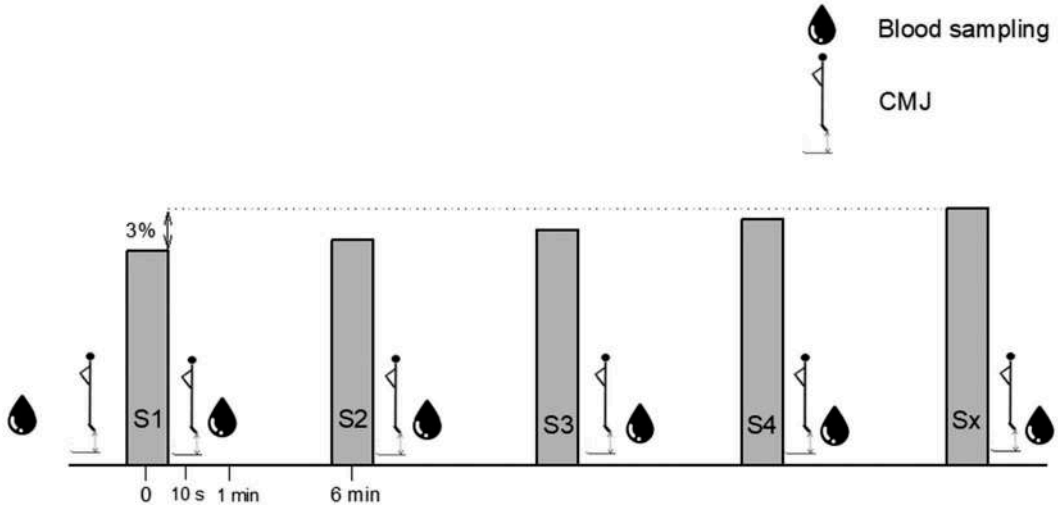

Figure 1. Overview of the experimental protocol.

sprint times was measured using coefficients of variation (CV) and intraclass correlation coefficients (ICC) with 95% confidence intervals (95%CI). This test showed very high reliability (CV: 1.5%, ICC (95%CI): 0.92 (0.84–0.96)).

Countermovement jump height

Jump height was calculated from flight time using an infrared platform (Optojump, Microgate, Italy). The infrared platform estimates the jump height from the flight time using the following equation: $h = t^2 \times 1.22625$, with h being the jump height in meters and t being the flight time of the jump in seconds. During the CMJ, the subject was instructed to rest his hands on his hips while performing a downward movement to reach about 90º of knee flexion, followed by a maximal vertical jump. All subjects were instructed to keep their knees straight during the flight phase of the jump, and to land in an upright position in order to negate the possibility of overestimation of jump height through the athlete "tucking". For the pre-values, three maximal CMJ, separated by 30 s rests, were performed before the first 60 m sprint. The average value of the three jumps was used for the subsequent statistical analysis. The reliability for the CMJ test was CV: 2.6%; and ICC (95% CI): 0.97 (0.92–0.98).Ten seconds after each sprint, a single CMJ was performed in an attempt to minimize the fatigue induced by the full CMJ test.

Mechanical measurements of fatigue

Two different methods were used to quantify the extent of the fatigue induced by the sprint session. The first method analysed the percentage increment in time from the fastest sprint [(fastest time – last time)/fastest time·100]. Athletes performed 60 m sprints until an FI of 3% was attained twice consecutively. The second method involved the calculation of the percentage change in CMJ height experienced after each sprint compared with the pre-test values.

Blood lactate and ammonia analyses

Capillary blood samples for the determination of lactate and ammonia concentrations were obtained from the fingertip before exercise and 1 min after each bout. The Lactate Pro LT-1710 (Arkray, Kyoto, Japan) portable lactate analyzer was used for lactate measurements. Ammonia was measured using a PocketChem BA PA-4130 (Menarini Diagnostics, Florence, Italy). Both devices were calibrated before each exercise session according to the manufacturer's specifications. The reliability of these devices has been previously reported. The CV ranged from 2.6% to 4.1% for lactate and from 3.0% to 5.2% for ammonia (Sánchez-Medina & González-Badillo, 2011).

Statistical analysis

Values are reported as mean ±standard deviation (SD). Statistical significance was established at the $P \leq 0.05$ level. The relative reliability was assessed using ICC and 95% CI calculated using the one-way random effects model. Test-retest absolute reliability was measured using the standard error of measurement, which was calculated as the root mean square of total mean square intra-subject. This standard error was used for the calculation of CV. The reliability was assessed in repeated measurements in the same testing session (2 sprints and 3 jumps), with an interval of 6 minutes between sprints and 1 minute for jumps. The variability was assessed using the CV calculated as the SD divided by the mean and multiplied by 100. The normal distribution of the data was verified with the Kolmogorov-Smirnov test. Relationships with Pearson's coefficients (r) were used to calculate the respective relationships between variables. The standard error of the estimate (SEE) was calculated. The magnitude of correlation was assessed with the following thresholds: <0.1, *trivial*; <0.1–0.3, *small*; <0.3–0.5, *moderate*; <0.5–0.7, *large*; `0.7–0.9, *very large*; and <0.9–1.0, *almost perfect* (Hopkins, Marshall, Batterham, & Hanin, 2009). An ANOVA with repeated measures with *Bonferroni* adjustment was used to examine the effects of all running sprints across time on mechanical and metabolic responses. The importance of the differences found between pre- and post-values for different variables was assessed through the effect size (Cohen's *d* coefficient), interpreted as follows: small difference: 0.15 <d <0.4, medium difference: 0.40 <d <0.75, large difference: 0.75 <d <1.10 and

Table 1. Repetitions, mechanical and metabolic acute effects over the session up to a velocity loss of 3%..

	1st Repetition	1% Velocity Loss	ES	2% Velocity Loss	ES	3% Velocity Loss	ES
Repetitions (n)	1.0 ± 0.0	4.3 ± 1.6*		6.6 ± 2.0*		9.2 ± 2.7*	
Time 60 m (s)	7.00 ± 0.26	7.07 ± 0.25*	0.26	7.18 ± 0.24*	0.69	7.26 ± 0.16*	1.26
CMJ Height Loss (%)	6.7 ± 1.4*	11.0 ± 2.4*	2.03	13.4 ± 2.0*	3.62	16.0 ± 2.5*	4.48
Lactate (mmol·l^{-1})	7.3 ± 1.6*	11.8 ± 1.4*	2.94	13.9 ± 1.5*	4.18	16.4 ± 1.7*	5.37
Ammonia (umol·l^{-1})	46.9 ± 11.9*	94.7 ± 15.6*	3.28	122.8 ± 16.4*	5.06	152.4 ± 11.3*	8.62

VL: Velocity loss during running sprint, quantified through a fatigue index calculated as follows: (fastest time – last time)/fastest time·100]; ES: Effect Size; Repetitions: Number of sprints performed until achieving the speed loss indicated; Time 60 m: Sixty m running sprint time; CMJ height loss: Relative jump height decrement experienced from the pre-values jump height for the velocity loss indicated; Lactate: Blood lactate concentration attained 1 min after the completion of the 60 m sprint corresponding to the speed loss indicated; Ammonia: Blood ammonia concentration attained 1 min after the completion of the 60 m sprint corresponding to the speed loss indicated. *Denotes significance at: $P < 0.05$ with respect to first measurement.

very large difference: $d > 1.10$. The statistical analyses were performed using SPSS software version 18.0 (SPSS Inc., Chicago, IL).

Results

Descriptive characteristics of the protocol used are reported in Table 1. The fastest 60 m sprint time was 7.00 ± 0.26 s (Table 1), and pre-values for CMJ height were 46.4 ± 5.4 cm. Blood lactate and ammonia concentrations in basal conditions were 1.5 ± 0.5 mmol·l^{-1} and 31.7 ± 3.2 µmol·l^{-1}, respectively. The number of sprints completed until a speed loss of 3% was reached was 9.2 ± 2.7 sprints. Figure 2 shows an example of the evolution of sprint times, CMJ height loss, together with blood lactate and ammonia concentrations for a representative subject and protocol. The first 6 sprints induced greater variability for each variable than 1, 2 and 3% of velocity loss (CMJ height: 1%: 21.8%, 2%: 14.9%, 3%: 15.6%, and 6 first sprints: 23.4%; Lactate: 1%: 11.9%, 2%: 10.8%, 3%: 10.4%, and 6 first sprints: 21.8%; and Ammonia: 1%: 16.5%, 2%: 13.4%, 3%: 7.4%, and 6 first sprints: 24.2%).

CMJ height losses

The decrease in CMJ height pre-post exercise was greater as the number of performed repetitions approached the 3% of speed loss proposed. Post-exercise CMJ height was significantly different ($p < 0.05$) than pre-exercise following all repetitions performed (Table 1). The CMJ height loss observed was 6.7 ± 1.4, 11.0 ± 2.4, 13.4 ± 2.0 and 16.0 ± 2.5% after the first sprint and for 1, 2 and 3% of speed loss induced during 60 m running sprints.

Blood lactate and ammonia responses

Blood lactate and ammonia concentrations increased as the number of repetitions each subject performed approached the maximum possible number of repetitions up to the 3% of speed loss proposed (Figure 3). Both lactate and ammonia levels were significantly higher than pre-exercise resting values from the first sprint performed ($p < 0.05$, Table 1). Blood lactate and ammonia concentrations were 16.5 ± 1.7 mmol·l^{-1} and 153.1 ± 11.6 µmol·l^{-1} after the last sprint.

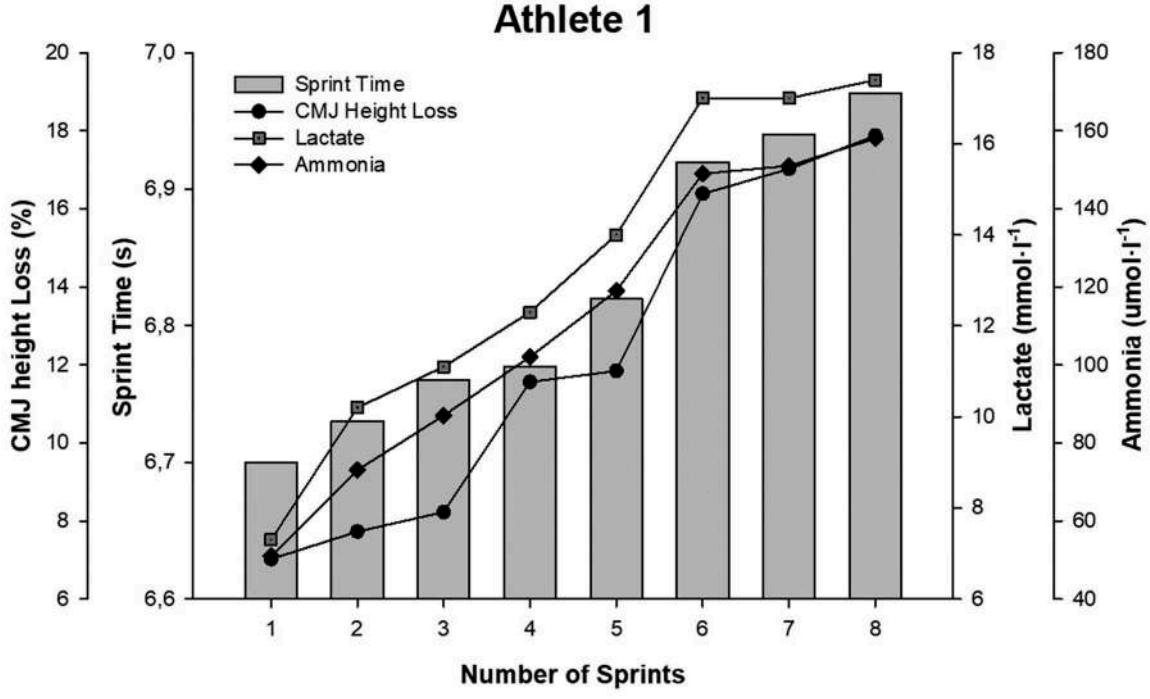

Figure 2. Example of the evolution of sprint times, CMJ height loss, blood lactate and ammonia concentrations for a representative subject.

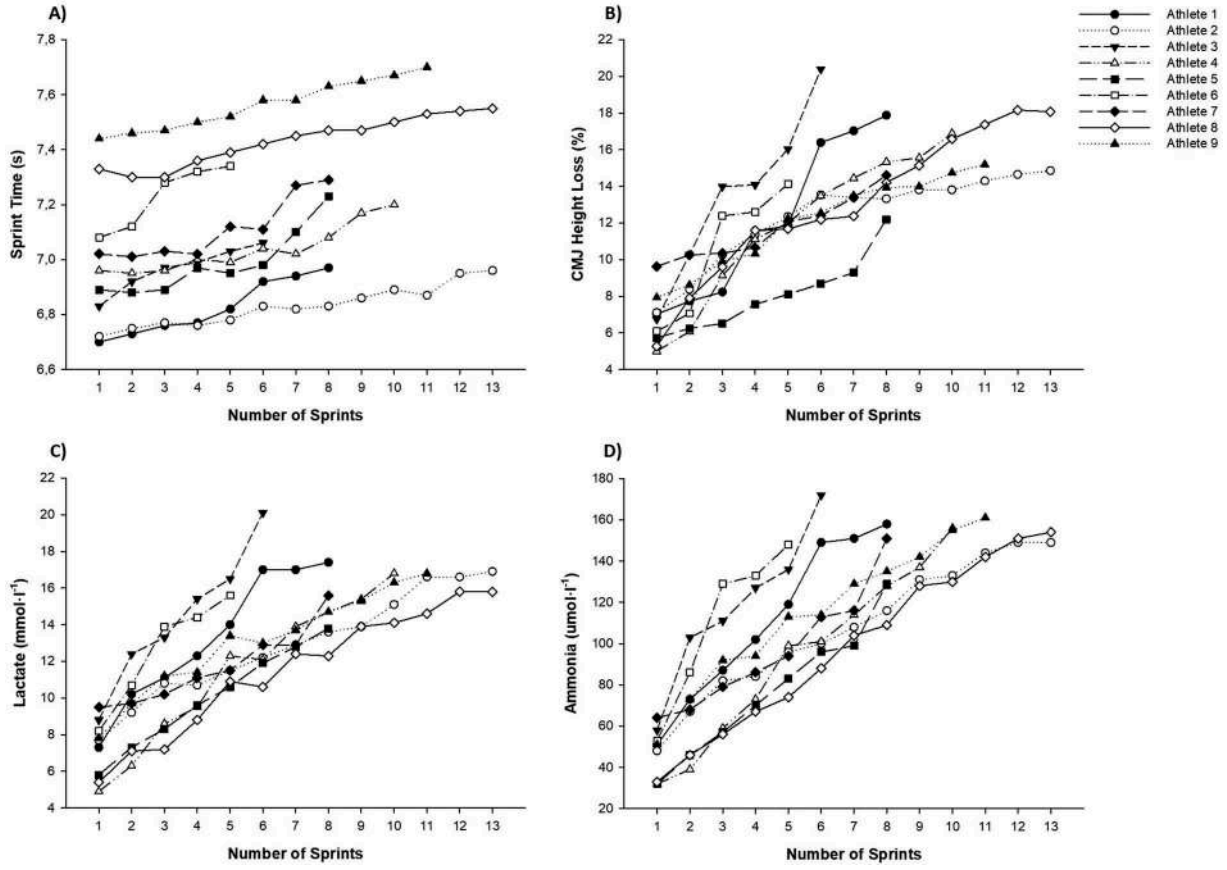

Figure 3. Evolution of sprint times, CMJ height loss, blood lactate and ammonia concentrations for each subject.

Relationships between jump height loss and velocity loss in 60 m running sprint, blood lactate and ammonia concentrations

When the data from the nine athletes were pooled, almost perfect/very large relationships were observed between CMJ height loss and blood lactate (r = 0.91), ammonia (r = 0.91), and velocity loss in 60 m running sprints (r = 0.76) (Table 2). In addition, nearly perfect relationships were observed for each athlete between CMJ height loss and blood lactate (r = 0.93–0.99), blood ammonia (r = 0.94–0.99), and velocity loss (r = 0.84–0.98) (Table 2).

Relationships between velocity loss in 60 m running sprint and blood lactate and ammonia concentrations

When all data were pooled, very large correlations were found between velocity loss and blood lactate (r = 0.83) and ammonia (r = 0.86) concentrations (Table 3). Furthermore, almost

Table 2. Regression equations and estimation error for the relationship between relative jump height loss and relative velocity loss, blood lactate and ammonia concentrations for each subject.

	LACTATE			AMMONIA			VELOCITY LOSS		
Athletes	r	*Equation*	SEE	r	*Equation*	SEE	r	*Equation*	SEE
1	0.97	*LA = 0.813JH + 3.360*	±1.77	0.98	*AM = 8.801JH + 3.814*	±15.47	0.98	*VL = 0.339JH − 2.238*	± 0.34
2	0.92	*LA = 1.100JH − 0.730*	±2.24	0.94	*AM = 12.438JH − 45.895*	±21.68	0.84	*VL = 0.452JH − 4.035*	±0.59
3	0.98	*LA = 0.805JH + 3.491*	±1.51	0.98	*AM = 7.901JH + 10.524*	±14.80	0.97	*VL = 0.207JH − 0.702*	±0.24
4	0.99	*LA = 0.969JH − 0.078*	±1.10	0.98	*AM = 10.123JH − 26.664*	±16.37	0.85	*VL = 0.314JH − 2.459*	±0.71
5	0.93	*LA = 1.246JH − 0.008*	±2.01	0.97	*AM = 14.796JH − 42.449*	±15.09	0.97	*VL = 0.876JH − 5.443*	±0.46
6	0.98	*LA = 0.815JH + 4.133*	±1.06	0.98	*AM = 10.550JH + 0.339*	±13.69	0.98	*VL = 0.476JH − 2.992*	±0.34
7	0.97	*LA = 1.142JH − 1.646*	±0.98	0.97	*AM = 16.261JH − 93.380*	±13.85	0.95	*VL = 1.011JH − 10.300*	±0.60
8	0.97	*LA = 0.842JH + 0.437*	±1.65	0.98	*AM = 10.040JH − 32.749*	±16.04	0.97	*VL = 0.358JH − 2.828*	±0.31
9	0.98	*LA = 1.092JH − 0.168*	±1.09	0.99	*AM = 13.546JH − 49.231*	±9.53	0.96	*VL = 0.499JH − 4.414*	±0.34
Overall	0.91	*LA = 0.843JH + 2.263*	±1.38	0.91	*AM = 9.528JH − 10.045*	±14.91	0.76	*VL = 0.310JH − 1.935*	±0.84

r: Relationships between blood lactate, blood ammonia and velocity loss, and percentage of jump loss induced by the successive 60 m running sprints for each athlete; Equation: Prediction equation to estimate blood metabolite concentrations and relative velocity loss in 60 m running sprint from jump height loss for each athlete; SEE: Standard Error of Estimate; JH: Relative jump height decrement experienced from the pre-values jump height; LA: Blood lactate concentration attained 1 min after the completion of the 60 m sprint; AM: Blood ammonia concentration attained 1 min after the completion of the 60 m sprint; VL: Velocity loss during running sprint, quantified through a fatigue index calculated as follows: (fastest time − last time)/fastest time·100]

Table 3. Regression equations and estimation error for the relationship between relative velocity loss and blood lactate and ammonia concentrations for each subject.

LACTATE				AMMONIA			
Athletes	r	*Equation*	SEE	r	*Equation*	SEE
1	0.98	*LA = 2.054VL + 9.719*	±0.57	0.98	*AM = 22.879VL + 70.587*	±6.72
2	0.95	*LA = 2.334VL + 9.193*	±0.89	0.94	*AM = 25.466VL + 68.091*	±9.73
3	0.95	*LA = 3.626VL + 6.833*	±1.09	0.94	*AM = 31.784VL + 53.481*	±10.97
4	0.87	*LA = 2.365VL + 8.580*	±1.80	0.91	*AM = 27.039VL + 59.540*	±17.23
5	0.90	*LA = 1.186VL + 8.370*	±1.11	0.93	*AM = 14.455VL + 55.504*	±11.21
6	0.97	*LA = 1.332VL + 10.310*	±0.53	0.98	*AM = 17.349VL + 80.123*	±5.83
7	0.86	*LA = 0.973VL + 10.230*	±1.14	0.88	*AM = 13.935VL + 75.973*	±14.80
8	0.99	*LA = 2.434VL + 6.864*	±0.53	0.98	*AM = 29.695VL + 41.965*	±7.21
9	0.97	*LA = 1.930VL + 10.020*	±0.63	0.97	*AM = 24.274VL + 76.428*	±7.00
Overall	0.83	*LA = 1.839VL + 9.293*	±1.63	0.86	*AM = 21.758VL + 67.434*	±16.53

r: Relationships between blood lactate and ammonia concentrations and velocity loss induced by the successive 60 m running sprints for each athlete; Equation: Prediction equation to estimate blood metabolite concentrations from relative velocity loss in 60 m running sprint for each athlete; SEE: Standard Error of Estimate; LA: Blood lactate concentration attained 1 min after the completion of the 60 m sprint; AM: Blood ammonia concentration attained 1 min after the completion of the 60 m sprint; VL: Velocity loss during running sprint, quantified through a fatigue index calculated as follows: (fastest time − last time)/fastest time·100].

perfect/very large relationships were observed for each athlete between velocity loss and blood lactate (r = 0.86–0.99), and blood ammonia (r = 0.88–0.98) concentrations (Table 3).

Discussion

To the best of our knowledge, only one study has used mechanical and metabolic responses to objectively monitor the fatigue induced in the typical sprint training performed by sprinters (Jimenez-Reyes et al., 2016). That study analysed 40 m running sprints with a recovery period of 4 min between bouts until there was a 3% decrease from the best time recorded for each athlete, whereas the present study analysed 60 m running sprints with a recovery period of 6 min between bouts and the same 3% decrease in performance criteria. Therefore, the ratio "distance travelled:recovery" is the same for both studies, being 1 min for every 10 m of distance travelled. With this in mind, it is worth noting that the distance and recovery used in our study were chosen according to habitual workout routines for sprinters, in order to have practical applicability. The high correlations observed between CMJ height loss and metabolic responses suggest that CMJ height might be used as a simple method for determining fatigue during sprint-training to individualize load prescriptions. This information is relevant because it provides meaningful feedback to coaches about the metabolic response induced by specific sprint training protocols in relation to the resulting deterioration in acute jumping performance.

Although several studies have examined the acute effects of sprint training, the number of repetitions performed were the same for all the subjects and compared the effect of the same amount of training for all athletes (Glaister, 2005; Morin et al., 2011), in which the number of repetitions was fixed beforehand and consequently induced great variability in mechanical and metabolic stimuli. In contrast to this fixed-dose sprint training, our study aimed to induce the same level of fatigue for all subjects, since mechanical and metabolic responses were induced by the same level of effort or fatigue (3% of speed loss) despite the number of repetitions to reach this criterion being different (9.2 ± 2.7 sprints). When we compared the variability induced by both methods, the first 6 sprints showed greater variability than an individualized level of fatigue, in this case, assessed through the speed loss during the sprint. This approach may allow a better determination of the amount of sprint effort necessary to reach a given level of performance decrease or fatigue. This new insight for monitoring sprint training has only been analysed by the aforementioned study (Jimenez-Reyes et al., 2016), and it analysed a 40 m distance. Therefore, analysing performance over 60 m can confirm the tendencies and allow better understanding of a very typical training distance for sprinters with a maximal speed focus, since over 60 m the two main sprint phases (acceleration and maximal speed) occur (Slawinski et al., 2017).

Since fatigue is postulated to be a continuous rather than a failure-point phenomenon, muscle fatigue can be seen as a decrease in the force-generating capability of the muscles involved (Enoka & Duchateau, 2008). Therefore, the gradual decrease in running speed and vertical jump height that take place during repeated maximal sprints can be interpreted as evidence of impaired neuromuscular function; its measurement could provide a relatively simple yet objective means of quantifying the extent of fatigue in individual athletes (Jimenez-Reyes et al., 2016; Sánchez-Medina & González-Badillo, 2011). Indeed, the decrease in CMJ height was greater as the number of performed repetitions approached 3% of velocity loss (CMJ height loss: 6.7 ± 1.4, 11.0 ± 2.4, 13.4 ± 2.0 and 16.0 ± 2.5% after the first sprint and for 1, 2 and 3% of velocity loss, Table 1). In essence, all models of fatigue involve two components: fatigue induction and fatigue quantification (Maffiuletti & Bendahan, 2009). In the present study, changes in performance were quantified using two different methods: 1) the percentage decline in speed over all the repetitions performed until a 3% speed loss was achieved; and 2) the percentage change in CMJ height pre and post each sprint performed. The use of CMJ height loss for quantifying changes in performance is not a new approach (Gathercole, Sporer, Stellingwerff, & Sleivert, 2015a; Gathercole, Sporer, Stellingwerff, & Sleivert, 2015b; Rusko, Nummela, & Mero, 1993; Sánchez-Medina & González-Badillo, 2011). However, the use of this simple and non-fatiguing test during actual sprint training sessions, for quantifying the changes in performance in real time, has not been analysed in detail.

The validity of using the percentage of vertical jump height loss to changes in performance during sprint training is further supported by the relationships observed between mechanical measures of fatigue (speed and jump height loss: r = 0.76) and between CMJ height loss and metabolic stress (acute lactate: r = 0.91, and ammonia responses: r = 0.91, Table 2). Blood lactate and ammonia increased as the number of performed repetitions approached the 3% of speed loss used for this study (lactate concentration: 16.5 ± 1.7 mmol·l^{-1} and ammonia concentration: 153.1 ± 11.6 μmol·l^{-1}, Table 1). Previous studies that used the same ratio of "distance travelled:recovery" also reported metabolite concentrations. Johnston et al. (Johnston et al., 2016) reported lactate concentrations of 9.3 ± 1.7 mmol·l^{-1} after 6 sprints of 50 m with 5 min recoveries between repetitions. Jiménez-Reyes et al. (Jimenez-Reyes et al., 2016) also reported lower metabolic response after performing 40 m sprints with 4 min recoveries until there was a 3% speed loss (lactate concentration: 14.3 ± 3.4 mmol·l^{-1} and ammonia concentration: 122 ± 33 μmol·l^{-1}). Thus, the same ratio of "distance travelled:recovery" might not induce the same metabolic response for a similar performance impairment (3% speed loss). Therefore, the metabolic stress associated with different sprint training sessions should be considered when planning volumes and recoveries. However, from the equations reported in the present study and by Jiménez-Reyes et al. (Jimenez-Reyes et al., 2016), it seems that a jump height loss of 10–12% induced by performing repeated sprints corresponds to approximately 10–12 mmol·l^{-1} and 90–100 μmol·l^{-1} of blood lactate and ammonia concentrations respectively, regardless of the distance travelled, except when the same ratio of "distance travelled:recovery" is used. In addition, nearly perfect relationships were observed between CMJ height loss and blood lactate (r = 0.93–0.99), blood ammonia (r = 0.94–0.99), and velocity loss (r = 0.84–0.98) for each athlete (Table 2). Therefore, when these equations are individualised for each athlete they allow a more accurate assessment of the extent of fatigue induced during a typical sprint training session. In contrast, lower relationships were found between speed loss during 60 m running sprints and metabolic responses both for all pooled data (blood lactate: r = 0.83; and ammonia: r = 0.86) and for each athlete (blood lactate: r = 0.86–0.99; and ammonia: r = 0.88–0.98, Table 3). Therefore, given its stronger relationship with metabolic responses, it may be even more beneficial to base the number of sprints to perform during a sprint training session on CMJ performance and not sprint velocity. To the best of our knowledge no data are currently available relating to when a sprint training session should be interrupted; in fact, the appropriate dose in sprint training sessions is controversial (Gorostiaga et al., 2010; Morin et al., 2011). However, our findings support the idea that the criteria for stopping a sprint session should be based on performance impairment or metabolic stability instead of the previously widely-used fixed number of sprints.

In this regard, a likely explanation for the performance impairment induced during a typical sprint training session might be decreased functioning of the contractile mechanisms of the muscle fiber in the presence of the metabolites produced during exercise (H^+, ADP, Pi) (Johnston et al., 2016). It is unclear whether intracellular acidosis originating from H^+ accumulation is the major cause of fatigue. It has been suggested that the muscle function impairment previously attributed to H^+ accumulation is largely a result of accumulated Pi, as a consequence of the rapid rate of ATP turnover during each sprint, inhibiting calcium release in the sarcoplasmic reticulum (Westerblad, Allen, & Lännergren, 2002). In any case, there has been demonstration of a strong association between low intracellular pH and decreased force and power (Degroot et al., 1993). On the other hand, the decrease in performance observed in repeated short and maximal intensity actions may be partly due to insufficient restoration of PCr (Balsom et al., 1992). A decline in PCr concentration reduces in muscle ATP levels, which is closely linked to an increase in AMP content (Stathis, Febbraio, Carey, & Snow, 1994). AMP is deaminated, increasing IMP levels and ammonia concentration (Stathis et al., 1994). In this way, an increase in blood ammonia level during short-term, high-intensity exercise is usually interpreted as accelerated purine nucleotide degradation and loss of total adenine nucleotides from muscle. This finding suggests that decrements in mechanical variables such as speed or vertical jump height are probably not desirable, especially in sprint athletes, since they induce excessive performance impairment and could also compromise recovery for subsequent sprint training sessions. Indeed, although a large part of the exercise-induced loss of nucleotides may be recovered, de novo synthesis in muscle is a slow and energy-consuming process and muscle performance can remain impaired for up to 48–72 h following high-intensity sessions (Hellsten-Westing et al., 1993).

Our results indicate that by monitoring vertical jump height during training it is possible to reasonably estimate the metabolic stress and neuromuscular fatigue induced by typical sprint training sessions. CMJ height loss information could provide valuable information for coaches since it provides a non-invasive, low-cost and easy way to indicate the point at which ammonia concentrations rise significantly above resting values. A previous study also showed that the CMJ test offers superior sensitivity to altered neuromuscular function than other jump and sprint tests (Gathercole, Sporer, et al., 2015b; Jimenez-Reyes et al., 2016). In addition, it has been shown that sprint performance requires a much shorter restoration time compared to CMJ performance (Gathercole, Sporer, et al., 2015b). These authors concluded that assessment of sprint capacity using time-based variables alone may lack the necessary sensitivity to determine neuromuscular fatigue status (Gathercole, Sporer, et al., 2015b). Anyway, speed loss may also be a good marker of internal load during the sprint training due to the high correlations observed with metabolic and mechanical changes.

Practical applications

The large variation induced by typical fixed-dose sprint training stresses the importance of following an individualised modeling approach to monitor training sessions in sprint athletes. CMJ performance may provide a more sensitive indicator of the metabolic load induced by sprint training than sprint performance itself. As such, practitioners may be better served by CMJ testing despite less task-specificity. Therefore, jump-

based measurements during training may lead to a more accurate setting of training loads in sprint training sessions, by using an individualised sprint dose based on mechanical and physiological responses rather than a standard fixed number of sprints for all athletes. The practical application for objective control of training loads is that the measurement of CMJ could be used by coaches and athletes to indirectly estimate the functional state of the muscle contractile machinery associated with the ability to regenerate ATP at high rates. Thus, if, during a training session, neither speed nor blood lactate or ammonia concentrations can be measured accurately, the CMJ test should be used in the monitoring and dosage of the training load. CMJ height loss provides precise information to inform decisions about when the subject should interrupt a training session. Taking this in account, it would be interesting to analyse the use of CMJ height to assess readiness to sprint. More studies are warranted to further explore this individualised approach to monitor internal load during sprint training. The present study is expected to contribute to the field of exercise science by allowing a more rational characterization of the sprint training stimulus.

Conclusions

In conclusion, the findings obtained in the present study strongly support the use of CMJ height for monitoring sprint training and quantifying mechanical and metabolic fatigue, because of the knowledge of metabolic stress induced by the sprint training session. The high relationships observed between CMJ height loss and metabolic response (lactate and ammonia) support this conclusion. In addition, when these equations are individualised for each athlete they provide a more accurate estimation of the mechanical and metabolic responses induced during a sprint training session. The use of a simple and non-fatiguing test such as CMJ could help monitor sprint training sessions without the need to measure blood lactate or ammonia concentrations, and would be more accurate than recording sprint times. Thus, CMJ could be a very useful and robust indirect measure of mechanical and metabolic changes induced during a sprint training session.

Disclosure statement

No potential conflict of interest was reported by the authors.

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