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Respiratory and muscular response to acute non-metabolic fatigue during ramp incremental cycling

Alessandro L. Colosio, Emmanuele Baldessari, Enrico Basso, Silvia Pogliaghi*

Department of Neurosciences, Biomedicine and Movement Sciences, University of Verona, Via Casorati 43, Verona, 37131, Italy

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ABSTRACT

We tested the hypothesis that acute, non-metabolic fatigue, by reducing maximal power output and possibly increasing muscle recruitment at a given exercise intensity, will reduce indexes of exercise tolerance during incremental cycling. Ten subjects performed three ramp incremental tests respectively after static stretching (STRC), dropjumps (DJ) or control (CTRL). Fatigue was assessed as reduction in maximal power output ($_{sprint}$PO) during isokinetic sprints. During the ramps we measured: oxygen consumption (VO_2), power output (PO), and surface electromyography. $_{sprint}$PO was reduced after STRC and DJ ($p = 0.007$) yet not after CTRL. During the ramps, the interventions augmented muscle excitation vs CTRL ($p \leq 0.001$). Peak PO and VO_2 were reduced after STRC (302 ± 39W $p = 0.033$, 3365 ± 465 ml/min $p = 0.015$) and DJ (300 ± 37W $p = 0.023$, 3413 ± 476 ml/min $p = 0.094$) vs CTRL (314 ± 41W, 3505 ± 486 ml/min). Interventions were associated with early occurrence of the ventilatory thresholds and increased VO_2 vs CTRL ($p = 0.029$). The physiological response after acute non-metabolic fatigue suggests a link between exercise intolerance and the decreased ability to produce force.

1. Introduction

Exercise tolerance, i.e. the ability to sustain a specific amount of force/power to complete a movement task, is fundamental in maintaining independence (for work, sport and leisure activities) and quality of life (American College of Sports Medicine, 2017). During whole-body exercise above the Gas Exchange Threshold (GET) and in particular above the Critical Power, exercise intolerance is associated with an increased cost of locomotion expressed as augmented gain of oxygen consumption (VO_2) for a given gain in absolute workload, compared to below threshold intensities. This loss of efficiency of locomotion is experimentally described as "excess-VO_2" during incremental exercise, and "VO_2 Slow Component" during constant load exercise (Grassi et al., 2015; Jones et al., 2011). Previous studies determined that roughly 85% of this phenomenon originates from the contracting muscles, while the remaining 15% corresponds to the increased VO_2 cost of ventilation (Poole et al., 1991). Several researchers focused on the possible causes of the muscular component of the loss of efficiency using a series of approaches (Jones et al., 2011 for a summary) and two main theories have been proposed: *i) decreased metabolic stability of type I muscle fibers*, caused by the negative effect of physiological metabolites (P_i, IMP, AMP, H^+, K^+), associated with increased O_2 cost of ATP resynthesis and/or increased ATP cost of contraction (Grassi et al., 2015; Jones et al., 2011) *ii) recruitment of fast-fatigable intrinsically inefficient type II muscle fibers,* to obtain/maintain the external power output above a certain intensity threshold (e.g. Critical Power) (Grassi et al., 2015; Jones et al., 2011; Jones and Poole, 2005). However, the exact physiological mechanisms underpinning the loss of efficiency remain elusive, one of the reasons being the difficulty to selectively affect either metabolic stability or type II fibres recruitment in human models. In fact, the different manipulations used in interventional studies (e.g. speed of movement, intensity modulation, aerobic training, priming exercise, nutritional interventions) affect to some extent both metabolic stability and fibres recruitment (Jones et al., 2011).

In this context, different authors explored the effect of acute, non-metabolic fatigue interventions, on the loss of efficiency. These interventions reduce the ability of muscles to produce force without inducing metabolic changes within the fibers. As such, non-metabolic fatigue interventions, should elicit increased muscle activation at a given absolute intensity (recruitment theory), while avoiding the confounding effect of intracellular homeostasis perturbations (metabolic instability theory). Among these authors, Hopker et al. (Hopker et al., 2016) evaluated the effect of 100 dropjumps (DJ) on VO_2 Slow Component during cycling exercise at the heavy-to-severe boundary.

* Corresponding author at: Department of Neurosciences, Biomedicine and Movement Sciences, University of Verona, Via Felice Casorati, 43, 37131 Verona, VR, Italy.
E-mail address: silvia.pogliaghi@univr.it (S. Pogliaghi).

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Notwithstanding a significant acute fatigue, the authors found no effect of DJ on VO_2. On the contrary, Esposito et al. (2012) found that when maximal force was acutely reduced following stretching (STRC) manoeuvres, the oxygen cost of locomotion increased both during ramp incremental and constant load exercises (Esposito et al., 2012; Limonta et al., 2015). However, given that measures of muscles recruitment (e.g. electromyography, EMG) are missing in the above cited studies, a link between muscle recruitment and loss of efficiency remains to be conclusively demonstrated/dismissed.

Considering the above contrasting results with acute, non-metabolic fatiguing interventions, further research is needed to investigate the possible link between fatigue, muscle excitation and augmented VO_2 at a given absolute workload.

Accordingly, this study was designed to investigate the effects of acute, non-metabolic fatigue induced by either DJ or STRC interventions on muscle excitation (i.e. EMG) and oxidative metabolism (i.e. VO_2) during incremental cycling. Considering the possible role of increased muscular activation (necessary to maintain the same workload when fatigued) as explanatory theory of the loss of efficiency phenomena, we hypothesis that both STRC and DJ *i)* will reduce maximal muscle force; *ii)* in turn, force loss will translate in increased muscle excitation at a given absolute workload; *iii)* finally, increased muscle excitation will impair maximal and submaximal indexes of exercise tolerance and reduced efficiency during a ramp incremental test performed to exhaustion.

2. Methods

2.1. Participants

Ten active men gave written informed consent to participate in the study (25 ± 4 years age, 80 ± 13 kg body mass 176 ± 8 cm stature, 25.6 ± 2.3 BMI). Inclusion criteria were male sex and age between 20 and 35 years; exclusion criteria were smoking and any condition that could influence the physiological responses during testing. The study was approved by Departmental Ethics Committee and adhered to the principles of the declaration of Helsinki. All participants were instructed to avoid caffeine consumption and physical activity respectively for at least 8 h and 24 h, respectively, before each testing session.

2.2. Protocol

Subjects visited the laboratory on five occasions within a maximum of three weeks. In the first two occasions, they were familiarized with the experimental procedure (isokinetic sprinting and incremental cycling) and the position on the ergometer was recorded for the successive appointments. On the third, fourth and fifth visit, separated by no less than 2 days of recovery, subjects performed the following identical protocol:

i) PRE-isokinetic cycling sprints to measure maximal power output at baseline.
ii) a 40-minutes intervention (either STRC, DJ, or control (CTRL)), with the DJ always executed as last session to avoid interference due to the long lasting effects of eccentric exercises (Twist and Eston, 2005).
iii) POST-isokinetic cycling sprints.
iv) a ramp incremental test to exhaustion.

A schematic representation of the protocol is presented in Fig. 1 (panel A).

Tests were conducted at the same time of the day in an environmentally controlled laboratory (22–25 °C, 55–65% relative humidity), after a standardised meal as previously described in (Keir et al., 2015).

2.3. Isokinetic sprints

Isokinetic maximal sprints were performed on an electromagnetically braked cycle-ergometer equipped with a pedal force measurement system and controlled by computer (Sport Excalibur PFM, Lode, Groningen, NL). The ergometer was set up into an isokinetic mode that limited the peak pedalling frequency and used to measure maximal force expressed on the pedals. Frequencies of 60 and 120 rpm were chosen to measure velocity-specific peak power as proposed by Cannon et al. (Cannon et al., 2011), and controlled by the electromagnetic breaking system of the flywheel. Each sprints session was composed of 4, five-seconds maximal sprints alternating between 60-120-60-120 rpm. The 4 maximal sprints were separated by a 2-min passive rest, to maximise recovery while limiting the total duration of the sprints session. For each five-seconds sprint, participants started to cycle with the bike set at freewheeling, gradually attaining the required rpm within 30 s. Sprints procedure (schematised in Fig. 1, panel B) was completed within 9 min.

2.4. Ramp incremental tests

The ramp incremental tests were performed on an electromagnetically braked cycle ergometer (Sport Excalibur, Lode, Groningen, NL) and consisted of a 4-min baseline cycling at 20 W, followed by a 25-W/min increase in power output (PO) until volitional exhaustion. Participants were asked to pick a self-selected cadence in the range of 70–90 rpm and to maintain it throughout all tests. Breath-by-breath pulmonary gas exchange, ventilation and heart rate were continuously measured using a metabolic cart (Quark B^2, Cosmed, Italy) as previously described (De Roia et al., 2012). Surface EMG of the left *vastus lateralis* muscle was continuously recorded by means of a wireless system (Wave wireless EMG, Cometa, Milan, Italy). A pair of surface Ag/AgCl electrodes (Blue sensor, Ambu®, Ballerup, Denmark) was attached to the skin with a 2-cm inter-electrode distance. The electrodes were placed longitudinally with respect to the underlying muscle fibers arrangement, according to the recommendations by Surface EMG for Non-Invasive Assessment of Muscles (Hermens et al., 2000). Before electrode application, the skin was shaved, scratched with sand-paper and cleaned with alcohol in order to minimize impedance. Semi-permanent ink marks allowed consistent re-positioning of the electrodes between sessions. The EMG transmitter connected to the electrodes was well secured with adhesive tape to avoid movement-induced artifacts.

Capillary blood samples (20 µl) were drawn from the ear lobe before and at the 1^{st},3^{rd}, 5^{th} and 7^{th} min after exhaustion. Samples were immediately analysed using an electro-enzymatic technique (Biosen C-Line, EKF Diagnostics, Barleben, Germany) and the highest value was considered as the peak of blood lactate accumulation for the incremental test.

2.5. Interventions

CTRL consisted in 40 min of resting in a sitting position under the control of the examiner.

STRC: six cycles of STRC, in which each position was maintained for 80 s, were used to maximise acute force reduction (Behm et al., 2016). The standardised stretching protocol sequentially involved the quadriceps of the right leg, the right hamstrings, left quadriceps, left hamstrings, with no recovery between exercises. Subjects were continuously encouraged to stretch muscles to the point of discomfort. The total duration of the STRC intervention was about 40 min. STRC effectiveness in increasing flexibility was measured pre and post STRC and CTRL (before the isokinetic sprints) using a sit-and-reach test (Limonta et al., 2015).

DJ: participants dropped 100 times from a 40-cm high platform down to 90° knee angle, with a resting period between each DJ of 20 s

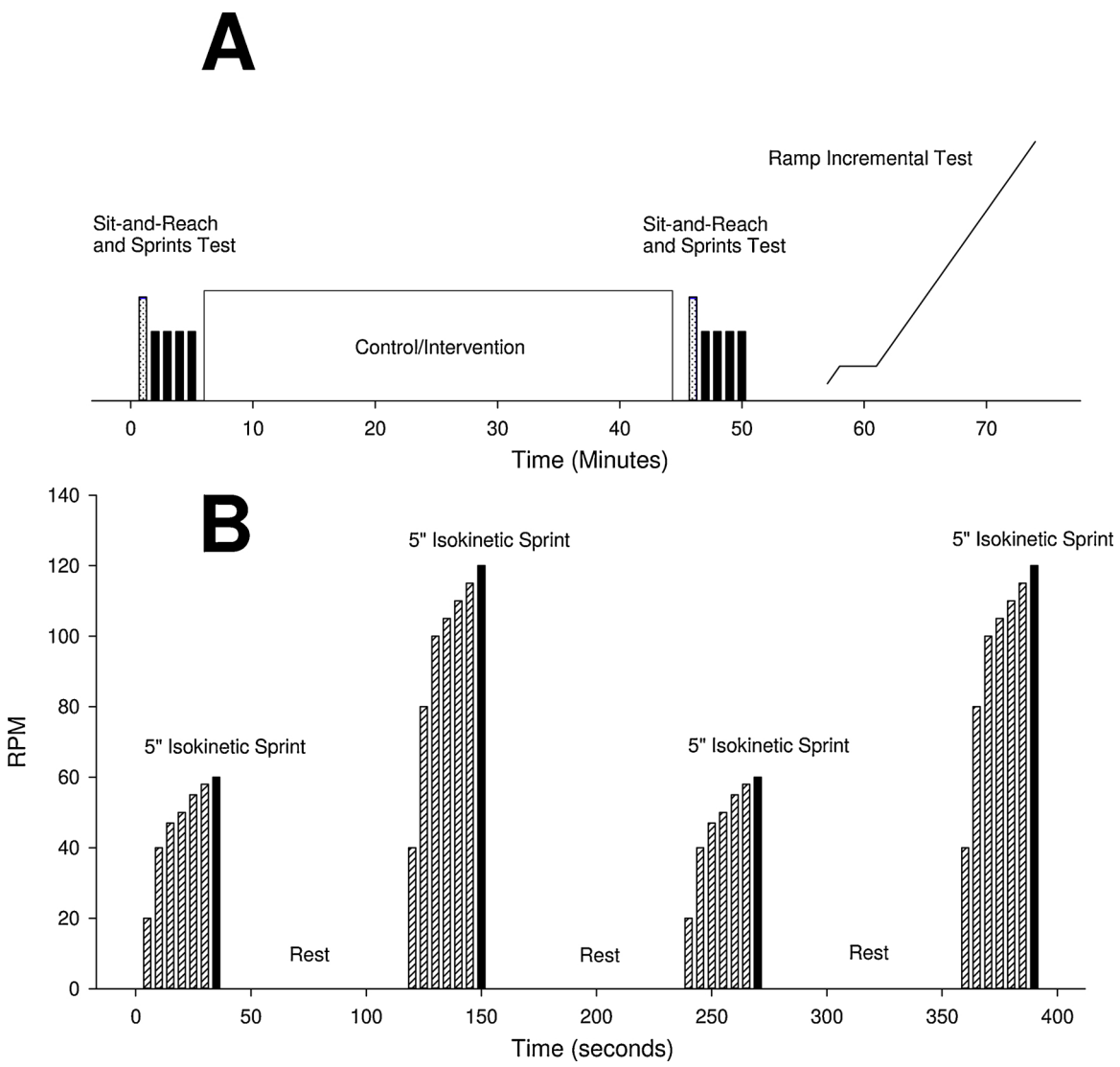

Fig. 1. Panel A: session protocol; Panel B: Isokinetic sprints test protocol; the black bar represents the 5 s sprints while the shaded bars are the 30 s of freewheeling cycling necessary to approach the required number of rpm.

(Hopker et al., 2016; Skurvydas et al., 2000). While the original version of this protocol entails a maximal jump after each drop, this part of the protocol was omitted to avoid the confounding effect of metabolic fatigue being added to the non-metabolic fatigue induced by eccentric exercise. DJ intervention lasted 40 min. In order to confirm minimal metabolic activation during DJ (Hopker et al., 2016), capillary blood samples were drawn before and at the 1st,3rd, 5th and 7th minutes after the end of the 100-DJ to determine peak lactate accumulation.

Data analysis

Isokinetic Sprints test: Crank torque was measured independently from the two crank arms by strain gauge transducers (maximal recordable force 2000 N, < 0.5 N resolution and measurement uncertainty of $< 3\%$). Angular velocity of the crank was recorded every 2 degrees using three independent sensors sampling in series with uncertainty of measurement $< 1\%$. Overall power for each pedaling cycle was calculated as the sum of the left and the right crank as resulted by the pedal force measurement analysis software. The initial and the last pedaling cycles of each sprint were excluded from computation. Then, maximal power expressed during each pedaling cycle was detected and cycles were averaged to obtain a mean peak power output for every sprint. Finally, mean peak power output ($_{sprint}$PO) of the two repetitions of the 60 and 120 rpm sprints performed either pre-or post-intervention were averaged to increase measure reliability and the relative change between pre and post conditions. ($\Delta\%_{sprint}$PO) was calculated as follows:

$$\Delta\%_{sprint}PO = [(_{sprint}POpost - _{sprint}PO\ pre)/_{sprint}POpre) \times 100]$$

Ramp incremental test: The raw EMG signal was rectified and smoothed using a fourth-order band-pass Butterworth digital filter with a frequency range set between 20 and 500 Hz. Root mean square (RMS) was calculated every second and averaged at 5 s intervals from the raw signal and was used as an index of the total muscle excitation (Vigotsky et al., 2018). The RMS recorded during the last 2 min of 20 W baseline for each test was used to normalize the ramp portion of the tests (Iannetta et al., 2017).

Gas exchange variables and heart rate were sampled breath by breath; aberrant data-points that lay 3 SD from the local mean were removed and thereafter data were interpolated at 5 s intervals; finally, gas exchange threshold (GET), respiratory compensation point (RCP), peak VO_2 (VO_{2peak}) and peak PO (PO_{peak}) were determined as previously described (Fontana et al., 2015). Briefly, VO_{2peak} was determined as the highest VO_2 obtained over a 10 s interval and PO_{peak} was defined as the highest mechanical power output achieved upon exhaustion during the RI exercise. GET and RCP were estimated by visual inspection from gas exchange variables by three blinded expert reviewers (Beaver et al., 1986; Whipp et al., 1989).

In addition, VO_2 and RMS signals obtained during CTRL, DJ and STRC were compared at the same absolute workload by performing a linear interpolation every 10% of the PO_{peak} reached during CTRL. Finally, RMS and VO_2 changes as a function of workload (% of the PO_{peak} reached during CTRL) were expressed as multiples of 20 W baseline values and the RMS/VO_2 ratio was calculated using these normalized units.

2.6. Statistics

After assumptions verification (i.e., normality, homogeneity of variance), repeated measures ANOVA was applied to compare flexibility values (pre and post sit-and-reach after CTRL/STRC) and blood lactate accumulation before and after the DJ and CTRL.

Pre and post $_{sprint}$PO at 60 and 120 rpm were compared within and between STRC, DJ, and CTRL conditions using a two-way repeated measures ANOVA (time x condition); $\Delta\%_{sprint}$PO (condition x pedalling frequency) among interventions at the two speeds were compared by two-way repeated measures ANOVA.

A two-way repeated measures ANOVA was performed to compare VO_2, RMS and RMS/VO_2 ratio between conditions over different workload percentages (workload x condition). Finally, a one-way repeated measures ANOVA was used to compare PO_{peak}, VO_{2peak}, GET, RCP, incremental Lactate peak, ventilation, heart rate and post-DJ Lactate peak between conditions.

Data are presented as means ± SD. 95% Confidence intervals around mean differences (95% ΔCIs [lower limit, upper limit]) and effect sizes of those differences (Cohen's *d,* ranked as trivial (0-0.19), small (0.20-0.49), medium (0.50-0.79) and large (0.80 and greater) (Cumming, 2014)) are also reported as objective and standardized measures to quantifying the magnitude of difference after intervention vs control condition (Winter et al., 2014). In effect size calculation, the SD in the control condition was used to standardize the mean difference for each contrast (Field et al., 2012).

All statistical analyses were performed using Sigmaplot version 12 and α was set in advance at the 0.05 level; statistical significance was accepted when $p < \alpha$.

3. Results

Flexibility, as measured by sit-and-reach test, was not significantly different at baseline between CTRL and STRC and significantly improved only after STRC (+0.4 ± 7.6 cm *pre* vs +5.9 ± 6.5 cm *post* STRC, $p < 0.001$, $d = 0.847$, 95%CI = +1.881, +9.969; +0.9 ± 5.2 cm *pre* vs +0.9 ± 5.3 cm *post* CTRL, $p = 0.832$, $d = 0.009$, 95%CI = -2.389, +4.189). Blood lactate concentration was not significantly different at baseline between CTRL and DJ and was not significantly affected by either DJ protocol (1.0 ± 0.3 mmol/L *pre* vs 1.1 ± 0.2 mmol/L *post* DJ, $p = 0.110$, $d = 0.349$, 95%CI = 0.305, 1.660) or CTRL (1.0 ± 0.2 mmol/L *pre* vs 1.1 ± 0.3 mmol/L *post* CTRL, $p = 0.274$, $d = 0.405$, 95%CI = 0.338, 1.690).

During isokinetic sprints, A significant interaction between "condition" and "time" was detected for the 60 ($p = 0.002$) and the 120 ($p = 0.008$) rpm. Post-hoc analysis revealed that $_{sprint}$PO was significantly reduced by DJ and STRC during the lower speed, 60 RPM sprints and during the higher speed, 120 RPM sprints (Table 1). On the contrary, no changes were found between pre and post after CTRL for both the pedaling frequencies (Table 1). Regarding $\Delta\%_{sprint}$PO, a main effect of "condition" was detected ($p = 0.007$), while there was no main effect of "pedaling frequency" ($p = 0.532$). No interaction was found between "condition" and "pedaling frequency" ($p = 0.097$).

During the ramp incremental tests, muscle excitation increased as a function of workload during CTRL; both the interventions significantly affected muscle excitation (i.e. RMS, main effect: $p \leq 0.001$) that was increased at a given absolute workload vs CTRL (Fig. 2). Post-hoc analysis revealed significantly higher muscle excitation for DJ compared to CTRL at workloads ≥ 20% of CTRL peak power output; furthermore, a significantly higher muscle excitation was observed for STRC compared to CTRL at workloads ≥ 40% of the CTRL peak power output.

Acute fatigue and increased muscular excitation translated in reduced peak power output after DJ compared to CTRL and reduced peak power output and VO_{2peak} after STRC. Moreover, both thresholds occurred at a lower W and VO_2 after DJ and STRC. These data are presented extensively in Table 2 together with the blood lactate, ventilation and heart rate values measured in different conditions.

A significant main effect of intervention was detected for VO_2 ($p = 0.029$) as a function of workload; both interventions resulted in an increased VO_2 at a given absolute workload vs CTRL (Fig. 2).

Finally, the stability of RMS/VO_2 ratio as a function of exercise intensity in all conditions indicates that muscle activation relative to metabolic intensity was not affected by workload (main effect of intensity: $p = 0.375$, Fig. 2). On the contrary, a significant main effect of interventions was demonstrated on RMS/VO_2 ratio (main effect: $p = 0.023$) (Fig. 2). However, post-hoc analysis revealed significant increases in muscle activation relative to metabolic intensity only at < 40% of CTRL PO_{peak} following DJ and at 90% of CTRL PO_{peak} following STRC compared to CTRL (Fig. 2). Between 40 and 80% of CTRL PO_{peak} the increase in muscle activation was matched by an equivalent increase in VO_2.

4. Discussion

In this study, we tested the physiological response to ramp incremental exercises performed in separated days in CTRL conditions and after two distinct acute fatiguing interventions (i.e. DJ and STRC). Both interventions caused acute, non-metabolic fatigue (detected as reduced maximal cycling power); in turn, acute fatigue augmented muscle recruitment for a given absolute workload during ramp incremental cycling, reduced maximal (peak power output and VO_{2peak}) and submaximal indexes of exercise tolerance (GET, RCP), compared to CTRL. Moreover, both acute fatiguing interventions were associated with metabolic loss of efficiency (i.e. higher VO_2 at an identical submaximal workload). These findings suggest a possible link between exercise intolerance/loss of efficiency and the observed decreased ability to produce force as a result of acute, non-metabolic fatigue interventions.

The isokinetic sprints test used in this investigation was able to detect a velocity-specific peak power output impairment after both interventions compared to CTRL. No changes in velocity-specific peak power output occurred after CTRL (CTRL: 60 rpm + 1.3 ± 3.8%, 120 rpm: -0.7 ± 2.4%). On the contrary, maximal $_{sprint}$PO at both velocities was significantly reduced by both interventions (STRC: 60 rpm: -4.8 ± 4.7%; 120 rpm: -2.9 ± 3.0%; DJ: 60 rpm: -4.9 ± 3.1%; 120 rpm: -2.3 ± 3.5%). The amplitude of $_{sprint}$PO impairments after STRC measured in our study were consistent with previous investigations that used a variety of techniques to quantify maximal strength/power (Behm et al., 2016; Behm and Chaouachi, 2011). On the contrary, force reduction after DJ was lower than the 10% reduction reported by Hopker et al. (Hopker et al., 2016), during 6 s isokinetic sprints at 90 rpm. Their protocol included maximal jumping after dropping while our protocol did not; this is the likely cause of the larger fatigue effect reported by Hopker et al. compared to our study. Furthermore, our study was the first to investigate fatigue at two sprint velocities following DJ and STRC, in the attempt to detect a possible differential impairment of fast vs slow motor units. Previous studies had proposed that STRC (Limonta et al., 2015) may preferentially affect fast motor units, and therefore the higher sprint velocity. However, our data did not demonstrate differences in the velocity-specific peak power output at higher compared to lower pedaling frequency (Cannon et al., 2011). This finding favours the idea that STRC and DJ induce acute, non-metabolic fatigue to a similar extent in both fast and slow motor units.

Table 1
Mean ± SD peak power output during isokinetic sprints pre and post conditions.

		pre (W)	post (W)	*p*	*d*	95%CI [LL, UL]	Δ (W)	Δ (%)	*p*
Control	**60 RPM**	640 ± 54	649 ± 60	0.334	+0.167	610, 691	+4 ± 15	+0.6 ± 3.8	*CTRL vs DJ*
	120 RPM	949 ± 169	942 ± 171	0.469	−0.056	868, 1016	−7 ± 19	−0.7 ± 2.4	0.019
Dropjumps	**60 RPM**	**653 ± 48**	**621 ± 49**	**0.002**	**−0.668**	**589, 654**	−32 ± 30	−5.2 ± 3.1	*CTRL vs STRC*
	120 RPM	**929 ± 114**	**908 ± 96**	**0.034**	**−0.185**	**846, 971**	−21 ± 32	−2.3 ± 3.5	0.011
Stretching	**60 RPM**	**663 ± 59**	**631 ± 84**	**0.003**	**−0.536**	**590, 671**	−38 ± 29	−5.4 ± 4.7	*STRC vs DJ*
	120 RPM	**973 ± 97**	**944 ± 95**	**0.003**	**−0.294**	**881, 1008**	−29 ± 10	−2.9 ± 3.0	0.634

Isokinetic sprints peak power output is presented for the 60 and 120 RPM pedalling frequencies pre and post each intervention with p-values, confidence intervals and Cohen's effect size (*d*). Bolded values represent significant differences between pre and post absolute values. Δ represents the mean difference between pre and post sprints. Regarding $\Delta\%_{sprint}PO$, a main effect of "condition" was detected (p = 0.007), while there was no main effect of "pedaling frequency" (p = 0.532) nor interaction between "condition" and "pedaling frequency" (p = 0.097).

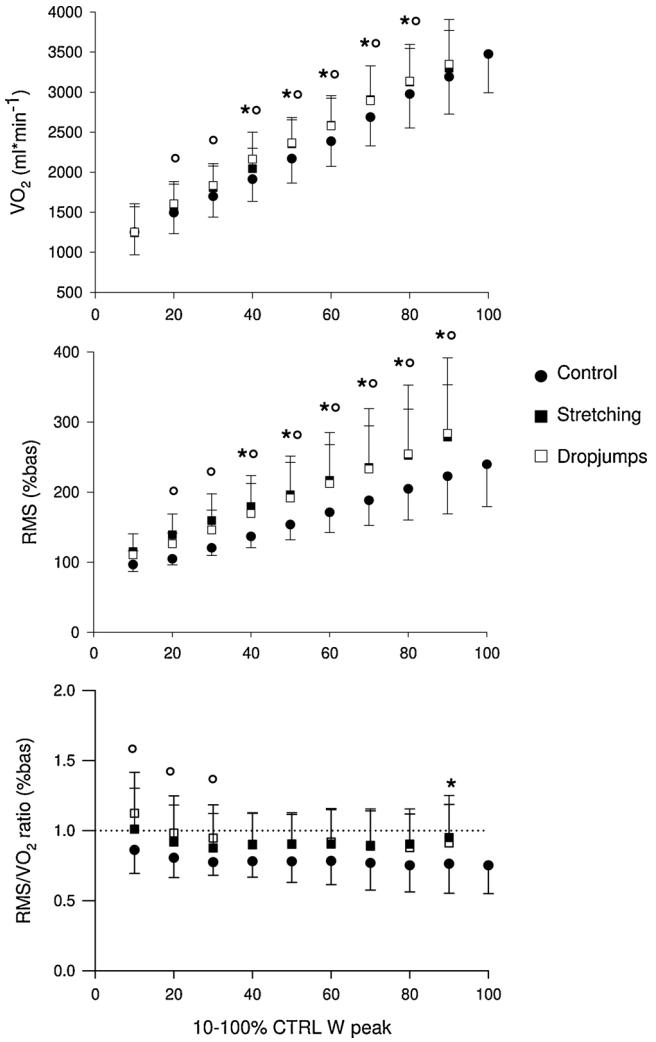

Fig. 2. Mean ± SD VO_2 (top panel) and Root Mean Square (RMS, medium panel) values and RMS/VO_2 ratio every 10–100% of Control W are presented. Symbols represent: **black**dots = control condition, **black**squares (Stretching), **white**squares (dropjumps). Significant main effect of intervention was detected for VO_2 (p = 0.029) and RMS (p ≤ 0.001). Statistical differences resulting from the post-hoc analysis are represented by blank dots (DJ vs CTRL) and asterisks (STRC vs CTRL).

This is the first time that acute fatiguing interventions were used to impair maximal force while contextually measuring metabolism and muscular excitation. The values measured during CTRL condition revealed an average aerobic fitness of our sample similar to the reference, sedentary population of young male adults (absolute VO_{2peak} 3505 ± 486 ml/min; normalized VO_{2peak}: 43.9 ± 6 ml/min/kg corresponding at the 50° percentile of the ACSM's guidelines) (American College of Sports Medicine, 2017), and a mean peak power output of 314 ± 41 W. Both the interventions caused a small impairment of VO_{2peak} (STRC: 3365 ± 465 ml/min (≈ -4% vs CTRL, p = 0.015); DJ: 3413 ± 476 ml/min (≈ -2.6% vs CTRL, p = 0.094)) and significant impairments of peak power output both after STRC and DJ (STRC: 302 ± 39 W (≈ -4% vs CTRL, p = 0.033); DJ: 300 ± 37 W (≈ -4.5% vs CTRL, p = 0.023)).

Impairment of maximal indexes of performance was accompanied by a higher metabolic activation at the same absolute workload compared to the control condition (Fig. 2) throughout the incremental test. In specific, a statistically significant loss of efficiency was identified at absolute workloads in the range of 40–80 % of CTRL-peak power output after STRC and of 20–80 % of CTRL-peak power output after DJ. Our results agree with previous work (Limonta et al., 2015) that reported a raised VO_2/W ratio during ramp incremental exercises performed after passive STRC. On the contrary, our data are in contrast with Hopker et al., that, in spite of a reduced exercise tolerance following DJ, reported similar VO_2 values for a given intensity (Hopker et al., 2016). Unfortunately, in the above investigations, a direct measure of muscular excitation was lacking, making it difficult to establish a clear relationship between possible alterations of muscle recruitment and VO_2 at a given workload. In our study, the RMS/VO_2 ratio data suggest that, between 40 and 80% of CTRL PO_{peak}, the increase in VO_2 observed following the fatiguing interventions was proportional to the augmented muscle excitation. It should also be noted that the fatiguing interventions did not alter the ventilation patterns during the different ramp incremental tests (Table 2), supporting the idea that the changes in VO_2 found after DJ and STRC were mostly due to a loss of efficiency in the working muscles rather than to an increased cost of ventilation (Coast and Krause, 1993).

This is also indirectly supported by the occurrence of GET (STRC: −195 ml/min; DJ: - 162 ml/min) and RCP (STRC: −268 ml/min; DJ: - 298 ml/min) at lower absolute power outputs compared to the control condition. These thresholds represent the boundaries of the "heavy" and "very heavy" exercise domains (Keir et al., 2015), and consequently the edge of an augmented involvement of type two muscle fibers (Jones and Poole, 2005). Importantly, the "shift" of these boundaries towards lower power outputs could lead to fatigue and exercise intolerance for a previously well tolerated load (Keir et al., 2016, 2015). Therefore, our results suggest a link between the ability of the body to maintain metabolic stability and the muscle's absolute capacity to produce force. Moreover, given that changes in VO_2/W ratio during ramp incremental exercise (i.e. "excess" VO_2) are considered equivalent to the VO_2 slow component measured during constant load cycling (Grassi et al., 2015), it is reasonable to speculate that fatiguing interventions would elicit increased muscular excitation and metabolic activation also when cycling at a fixed workload.

Table 2
Mean ± SD cardiorespiratory data during the 20 W warm-up, at peak, Gas Exchange Threshold (GET), and Respiratory Compensation Point (RCP).

		Control	95%CI [LL,UL]	Dropjumps	*p*	*d*	95%CI [LL,UL]	Stretching	*p*	*d*	95%CI [LL,UL]
Warm-up	VO_2 (ml/min)	1103 ± 320	904, 1301	1167 ± 289	0.612	+0.221	988, 1346	1088 ± 339	0.833	−0.044	878, 1298
	HR (b/min)	107 ± 14	98, 116	103 ± 10	0.321	−0.421	97, 109	98 ± 18	0.149	−0.467	87, 110
	VE (L/min)	16 ± 2	15, 18	16 ± 3	0.997	0.000	14, 18	16 ± 2	0.999	0.000	15, 17
Peak	W	314 ± 41	289, 340	**300 ± 37**	**0.023**	**−0.380**	**277, 323**	**302 ± 39**	**0.033**	**−0.314**	**278, 326**
	VO_2 (ml/min)	3505 ± 486	3204, 3806	3413 ± 476	0.094	−0.194	3118, 3707	**3365 ± 465**	**0.015**	**−0.301**	**3077, 3653**
	HR (b/min)	183 ± 11	176, 189	179 ± 11	0.131	−0.298	173, 186	181 ± 9	0.804	−0.132	176, 187
	VE (L/min)	151 ± 17	141, 162	154 ± 24	0.688	+0.125	139, 169	154 ± 17	0.578	+0.176	140, 162
	[La^-] (mmol/L)	12 ± 1	11, 13	**10 ± 2**	**0.038**	**−0.941**	**9, 11**	10 ± 1	0.247	−1.189	10, 11
	Time (sec)	704 ± 98	643, 765	**671 ± 87**	**0.022**	**−0.379**	**617, 725**	679 ± 82	0.073	−0.305	628, 730
GET	W	153 ± 44	125, 180	136 ± 52	0.070	−0.325	104, 128	136 ± 48	0.054	−0.363	106, 165
	VO_2 (ml/min)	2150 ± 464	2133, 2167	**1998 ± 524**	**0.050**	**−0.291**	**1673, 2322**	**1955 ± 484**	**0.018**	**−0.403**	**1655, 2255**
	HR (b/min)	142 ± 17	131, 152	**133 ± 19**	**0.027**	**−0.480**	**121, 144**	134 ± 17	0.051	−0.452	124, 144
	VE (L/min)	56 ± 12	49, 63	53 ± 17	0.572	−0.176	42, 64	52 ± 14	0.460	−0.286	49, 63
RCP	W	236 ± 44	209, 263	**205 ± 45**	**0.003**	**−0.692**	**177, 233**	**209 ± 34**	**0.007**	**−0.796**	**188, 230**
	VO_2 (ml/min)	2883 ± 488	2580, 3185	**2585 ± 451**	**0.004**	**−0.659**	**2305, 2865**	**2615 ± 402**	**0.006**	**−0.666**	**2366, 2864**
	HR (b/min)	164 ± 14	155, 173	**153 ± 16**	**0.002**	**−0.710**	**163, 173**	159 ± 15	0.091	−0.340	150, 168
	VE (L/min)	87 ± 18	76, 99	**76** ± 17	**< 0.001**	**−0.647**	**65, 87**	83 ± 22	0.126	−0.182	76, 98

Acronyms represent: W: power measured in watts, VO_2: oxygen consumption, HR: heart rate, VE: ventilation, [La^-]: peak blood lactate concentration. Bolded values represent significant differences versus control, no significant differences were found between DJ and STRC.

The main cause of STRC-induced loss of performance has been suggested to be the reduction in neural drive caused by prolonged periods of sensory stimulations (possibly at peripheral, spinal or supraspinal level), rather than the accumulation of metabolites within the muscles (Behm et al., 2016; Trajano et al., 2017). In addition, low lactate values measured following DJ intervention by this and other investigation confirmed low to null metabolic activation (Hopker et al., 2016). However, the exact physiological mechanisms underlying the impairment of force production after both STRC and DJ have not been clearly identified (Skurvydas et al., 2000; Trajano et al., 2017; Twist and Eston, 2005). Therefore, we cannot exclude that some extent of metabolic activation may be present also in these mostly non-metabolic fatiguing interventions.

5. Conclusion

Two acute, non-metabolic fatiguing interventions significantly reduced maximal cycling power output while augmenting muscle recruitment during ramp incremental cycling. Augmented muscle recruitment impaired maximal and submaximal indexes of exercise tolerance and led to metabolic inefficiency. Although further studies are warranted to identify a direct cause-effect relationship, these findings suggest a possible link between exercise intolerance/loss of efficiency and the observed decrease in the ability to produce force as a result of acute, non-metabolic fatigue.

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Declaration of Competing Interest

None.

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