

Title: Exercise-related sensations contribute to decrease power during repeated cycle sprints with limited influence on neural drive

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Abstract

Purposes: We manipulated the inspired oxygen fraction (FiO_2) to examine the effects of physiological perturbations on exercise-related sensations and the neural drive of the quadriceps during repeated, brief, maximal cycle sprints.

Methods: Nine active males completed a repeated sprint cycle protocol (10×4 -s maximal sprints with 30 s of passive recovery) in normoxia (NM; FiO_2 0.21) and severe normobaric hypoxia (HY; FiO_2 0.13). Peak power, quadriceps Root Mean Squared electromyography (RMS EMG), physiological (heart rate, arterial oxygen saturation, blood lactate concentration) and perceptual responses were recorded.

Results: The 10 sprints in HY were associated with lower arterial oxygen saturation values compared to NM (80.7 ± 0.9 vs. $95.6 \pm 0.6\%$; $P < 0.001$; Effect Size [ES]=0.98), higher blood lactate values (11.9 ± 0.4 vs. $9.9 \pm 0.9 \text{ mmol.L}^{-1}$; $P = 0.05$; ES=0.36), and greater exercise-related sensations ($\sim 36\%$; $P < 0.001$; ES>0.47). Mean power for sprints 1-10 were lower ($-13 \pm 3\%$; $P = 0.001$; ES=0.79), and sprint decrement was more pronounced in HY compared to NM (21.4 ± 3.7 vs. $13.2 \pm 2.7\%$; $P = 0.003$). There was a 17% decrease in RMS EMG activity from the first to the last sprint ($P < 0.001$; ES=0.65), independent of condition ($P = 0.597$; ES=0.04).

Conclusions: Despite severe hypoxia exacerbating both physiological and perceptual perturbations, the performance decrement observed during the repeated sprint protocol did not coincide with an accentuated decline in RMS EMG activity. These data suggest that higher-than-normal exercise-related sensations or perceptions coincide with fatigue during repeated sprinting, independent of changes in neural drive, when the task characteristics are known beforehand.

Keywords: Perceptual cues, Repeated-sprint ability, Hypoxia, Central fatigue, Overall perceived exertion.

ABBREVIATIONS

EMG: Electromyography

FiO₂: Fraction of inspired oxygen

HY: Hypoxic conditions

MP: Mean power

NM: Normoxic conditions

RMS EMG: Root Mean Squared electromyography

RPE: Ratings of perceived exertion

RSA: Repeated-sprint ability

INTRODUCTION

Repeated-sprint exercise requires muscles to perform intermittently at maximal contraction rates (Rorresperalta et al. 2016). It is widely recognised that muscle disturbances (*i.e.*, muscle excitability, limitations in energy supply, metabolite accumulation) have a major contribution to the subsequent musculature fatigue, which manifests as an inevitable reduction in mechanical power during successive efforts (Mendez-Villanueva et al. 2012). A decline in neural drive to the active musculature, as assessed via surface electromyography (EMG) signals (*i.e.*, a reasonable proxy for net motor unit activity), also becomes apparent when there is a substantial (*e.g.*, above 8%) sprint decrement (*i.e.*, the decline in repeated sprint performance relative to the first sprint) (Girard et al. 2011). The mechanisms underpinning fatigability and the associated performance decrements are complex, and influenced by the complex interplay between physiological disturbances and exercise-related sensations (Minett and Duffield 2008).

Homeostatic disturbances of multiple regulatory systems (*i.e.*, skeletal muscles, the heart and lungs) provide exercise-related sensations or perceptions that accompany reduction in voluntary contractile force (Taylor and Gandevia 2008; Marcora and Staiano 2010). While ratings of perceived exertion (RPE) have been used in numerous models of endurance exercise performance regulation (*i.e.*, psychophysiological model: Smirmaul 2012; inhibitory afferent feedback model: Amann 2011) to reflect the conscious sensation of how hard, heavy and strenuous a physical task feels (Marcora 2010), little agreement exists over where this perception of effort (also referred as exertion) originates. In fact, the continued use of a single measure of global perception of effort during exercise (*i.e.*, RPE) limits investigations into the possible role of different exercise-related sensations (*e.g.*, pain and fatigue perception in the muscles or lungs) in altering power (pacing) during maximal exercise.

In a pioneering study, O'Connor and Cook (2001) demonstrated that humans are able to differentiate between the perception of pain and the perception of effort during moderate-intensity (~70% of peak maximal oxygen uptake) cycling. In fact, each sensation is linked with different underlying neurophysiological mechanisms (O'Connor and Cook 1999). Recently, we have also reported that humans have the ability to differentiate between "sense of effort" or a "conscious awareness of the central motor command" (*i.e.*, a cognitive feeling of work associated with voluntary actions: Pageaux 2016) and other sensations related to physical activity. These other sensations, originating via sensory afferent feedback from various organs that include skeletal muscles, the heart, and lungs (Christian et al. 2014), include among

others difficulty breathing (*i.e.*, dyspnea) or limb heaviness (*i.e.*, the degree of hurt experienced in a specific muscle group: O'Connor and Cook 2001). To date, our understanding of what role these sensations play in regulating exercise performance, particularly during repeated maximal effort exercise, is incomplete. Hence, in the repeated-sprint ability (RSA) literature, there is a pressing and relevant need to differentiate between RPE that is typically exacerbated with effort repetitions (Billaut et al. 2011; Girard et al. 2015) and other exercise-related sensations or perceptions. This limits the implementation of effective training, as well as nutritional and recovery strategies, to optimise performance.

The ability to repeated maximal efforts, also known as RSA, is an important determinant of team-sport performance (Girard et al. 2011). However, typical RSA protocols pose a methodological problem. Deception studies involving either short-duration, maximal, isolated muscle contractions (Halperin et al. 2014) or repeated cycle sprints (Billaut et al. 2011) report a reduced neural drive. This highlights the existence of pre-exercise anticipatory pacing strategies, in accordance with the participant's belief that a longer task duration may be more detrimental. Nonetheless, the nature of the sensory signals involved in perception of effort generation is still debated, with afferent feedback, corollary discharge, and combined models put forward to explain the generation of perceived exertion (Tucker 2009; Smirmaul 2012). In order to circumvent any influence of conscious pacing strategies, maximal-effort tasks (*i.e.*, briefs maximal "all-out" voluntary contractions) of short (< 10 s) duration are increasingly employed (Christian et al. 2014; Halperin et al. 2014). In fact, the presence of any pre-determined anticipatory pacing strategy during a maximal-effort exercise task is likely to decrease with shorter effort durations (Wittekind et al. 2011; Girard et al. 2016). Repeated "all-out" efforts of brief duration (< 5 s) would therefore provide an appropriate model to explore the time course of changes in perception of effort and exercise-related sensations or perceptions during maximal efforts.

The aim of this study was to exacerbate physiological perturbations through exposure to severe hypoxia likely impairing exercise tolerance (*i.e.*, presumably also inducing higher afferent feedback: Amann 2011; Girard et al. 2014), and explore the consequences on effort perception and exercise-related sensations during brief, repeated, maximal cycle sprints. It has been reported that an earlier and larger development of peripheral fatigue in an O₂-deprived environment leads to a more pronounced decrease in quadriceps neural drive, higher-than-normal RPE readings, and eventually a dampened RSA, compared to a sea-level control (Billaut et al. 2013; Billaut and Aughey 2013; Brocherie et al. 2016). Furthermore, an exacerbated perception of effort

induced by a decrease in force production capacity or mental exertion are signs of fatigue (Enoka and Stuart 1992; de Morree et al. 2012). With this in mind, it was hypothesised that decreased performance during repeated maximal-effort exercise under hypoxic exposure would be accompanied by an exaggeration of exercise-related sensations or perceptions.

METHODS

Participants

Nine male, team-sport athletes volunteered for this study (mean \pm SD age 29.4 ± 5.0 y, stature 1.80 ± 0.38 m, body mass 82.6 ± 7.1 kg). Participants were deemed “trained” according to the unified classification system proposed by De Pauw et al. (2013). Each participant completed a minimum of three individual 90-min sessions of high-intensity intermittent exercise training per week. Participants were all English-native speakers. They gave written informed consent before the commencement of the study after all the experimental procedures, associated risks, and potential benefits of participation had been explained. The study was approved by the Victoria University Human Research Ethics Committee. All procedures conformed to the Declaration of Helsinki.

Experimental Design

Each participant performed one familiarization session. This was followed by two experimental trials under either acute normoxic (NM; simulated altitude/fraction of inspired O₂ [FiO₂]: 0 m/0.21) or hypoxic conditions (HY; ~4000 m/0.13) using a randomised, single-blind research design.

Familiarization session

Participants reported to the laboratory one week prior to the first experimental session where they were familiarised with cycling on the SRM cycle ergometer (Schoberer Rad Meßtechnik, Jülich, Germany) and for the determination of their optimal cycling sprint cadence (*i.e.*, the pedalling rate that would allow participants to produce the greatest amount of mechanical work during the maximal sense of effort 4-s bout; Martin and Spirduso 2001). Briefly, participants completed two sets of 5 maximal 4-s isokinetic cycling sprints on the SRM cycle ergometer, with cadences ranging from 100-140 rpm, and separated by 3 min of passive recovery. The one revolution peak power for each sprint was recorded via the SRM torque software (Version 12.98 SRM GMBH), and a parabolic curve fitted to the peak power of the 5 sprints, with the optimal cycling cadence being determined as the highest predicted peak power using the equation of best fit

for any given cadence. During the preliminary visit, participants were familiarised with the various modified Borg CR10 scales and particular attention was paid regarding the distinction between sense of effort and perceptual responses.

Participants were instructed that the “sense of effort” scale is used to set the level of subjective awareness of mental or physical effort expended during the exercise task (Abbiss et al. 2015) and was assessed from the question: *‘How hard are you trying?’* (i.e., with the anchor points provided ranging from 0 or “no effort” to 10 or “maximum effort”). The illustration that “a brief maximal effort requires a maximal voluntary effort despite only inducing a small amount of peripheral discomfort” was explained to all participants (Smirmaul 2012). Sense of effort as well as rating of overall perceived exertion, perceived lower-limb heaviness, and perceived difficulty breathing, were recorded based on modified Borg CR10 scales (Christian et al. 2014). During the familiarization session all participants were thoroughly instructed on the distinction between task effort and awareness (sense of effort) and perceived exertion assessed with the various perceptual scales. Specifically, participants were instructed that these perceptual scales are used to evaluate their “degree of heaviness and strain experienced in the task” or subjective perception of (1) overall perceived exertion, (2) specific lower limb (quadriceps only) heaviness and (3) difficulty breathing. The questions: “*What is your overall perceived exertion?*”, “*How difficult does it feel to breathe?*” and “*How heavy do your legs feel?*” were printed above modified Borg CR10 scales (i.e., with the anchor points provided ranging from 0 or “nothing at all” to 10 or “maximal”) and visible to participants at all times (Christian et al. 2014).

Experimental trials

All trials (including familiarization session) were completed in a normobaric hypoxic chamber (Colorado Mountain Room System: Colorado Altitude Training, Boulder, CO). The efficacy of the blinding procedure was evaluated after each experimental session by questionnaires in which participants were asked whether they believed to be exercising in NM or HY. The observation that only 8 out of a possible 18 sessions were correctly identified indicates that the blinding procedure was effective. Trials were separated by at least 5 days and performed at the same time of day. Participants were asked to avoid vigorous exercise for 24 h, caffeine for 12 h, and food for 2 h before each trial. For both conditions (i.e., NM or HY), they entered the normobaric hypoxic chamber ~30 min before commencement of the repeated sprint protocol, and the total duration of the testing session was ~40 min.

Following entry to the hypoxic chamber, participants rested in a seated position for 10 min (wash-in period) while all equipment was attached. Afterwards, they completed a warm-up consisting of 5 min of continuous cycling on the SRM ergometer in the open-end mode at a subjective ‘sense of effort’ of 3 using a modified Borg CR10 scale (Christian et al. 2014). This was followed after 1 min of rest in a seated position by five progressive 4-s submaximal cycling bouts in the isokinetic mode at the individual pre-determined optimal sprinting cadence (group average: 120 ± 2 rpm). For each of the five submaximal bouts participants were instructed to work at a subjective ‘sense of effort’ of 4, 5, 6, 7 and 8 on the modified Borg CR10 ‘sense of effort scale’ (Christian et al. 2014), respectively, with 40 s of recovery interspersing each bout (15 s of passive rest and 25 s of cycling at ~ 100 W). Following the warm-up procedure, participants rested passively for 2 min. After an additional 3 min of recovery (2 min of passive rest and 1 min of cycling at ~ 100 W), two 4-s cycling bouts at a subjective “sense of effort” of 10 (*i.e.*, maximal) were completed, with each bout separated by 3 min of recovery (2 min of passive rest and 1 min of cycling at ~ 100 W).

The repeated sprint protocol consisted of 10×4 -s isokinetic cycle sprints, each at a maximal ‘sense of effort’, and interspersed with 30 s of recovery (15 s of passive rest and 15 s of cycling at ~ 100 W). Cycle sprints were completed in the isokinetic mode at the individual pre-determined optimal sprinting cadence (group average: 120 ± 2 rpm). The isokinetic mode allows the participant to pedal without resistance up to the fixed cadence, while resistance is automatically and proportionally increased when participants try to overcome it (Fernández-Peña et al. 2009). All bouts were initiated from a rolling start, with participants instructed to progressively increase to a cadence within 2-5 rpm of their optimal sprinting cadence 10 s prior to each bout. This procedure was used to ensure that all bouts began with the same kinetic energy, while minimizing any jolting sensation as participants reached their optimal sprint cadence and the breaking resistance of the ergometer was applied.

Participants were routinely provided (~ 15 s before each bout) with identical instructions to perform “all-out” exercise bouts. Heart rate, arterial oxygen saturation, sense of effort, as well as difficulty breathing, lower-limb heaviness and overall perceived exertion, were reported and recorded in an invariant order at exactly 10 s following each 4 s bout. Participants were instructed to reflect on their subjective perceptions during the preceding exercise bout.

Physiological Responses

Heart rate and arterial oxygen saturation were monitored and estimated, respectively, via a wireless monitoring system (Polar Electro Oy, Kempele, Finland) and non-invasive pulse oximetry using a finger probe (Palmsat 2500, NONIN Medical Inc., Plymouth, MI, USA). A capillary blood sample was taken from a fingertip and analysed immediately for lactate concentration ([La]) using an automated analyser (Lactate Pro LT-1710, Arkray, Japan) before the warm-up and 2 min after the RSA test.

Electromyography

Electromyographic (EMG) signals from superficial *rectus femoris*, *vastus lateralis*, and *vastus medialis* muscles of the right lower limb were recorded using pre-amplified bi-polar surface EMG (Delsys, Trigno Wireless, Boston, Massachusetts, USA) with an inter-electrode (center-to-center) distance of 20 mm and placed according to SENIAM's recommendations. Before electrode placement, the skin was lightly abraded and washed to remove surface layers of dead skin, hair, and oil. The ground electrode was attached to the pisiform bone of the right hand. The position of the EMG electrodes was marked with indelible ink (and pictures of the locations were taken) to ensure that they were placed in the same location during subsequent trials. The myoelectric signal was amplified (gain = 1000 ×) and filtered (bandwidth frequency = 12–500 Hz) to minimise extraneous noise and possible movement artifacts in the low-frequency region and to eliminate aliasing and other artifacts in the high-frequency region. EMG signals were recorded (sampling frequency = 2000 Hz) using a dedicated analysis system (Spike2 v3.21; Cambridge Electronic, Cambridge Design, Cambridge, UK).

Data Analysis

All power data were analysed using SRM torque analysis software (SRM Torque Win 1.1.0, SRM, Schoberer Rad MeQtechnik, Jülich, Germany) while all EMG data post-processing were performed in Spike2 (Version 3.21; Cambridge Electronic, Cambridge Design, Cambridge, UK). During the maximal 4-s cycle efforts, the mean power (MP) and RMS EMG activity for the 8 highest cycle revolutions was recorded for each muscle. Raw data for *rectus femoris*, *vastus lateralis* and *vastus medialis* and muscles were 0.119 ± 0.035 vs. 0.118 ± 0.028 mV, 0.246 ± 0.055 vs. 0.220 ± 0.050 mV and 0.223 ± 0.065 vs. 0.249 ± 0.051 mV for sprint 1 in NM and HY, respectively, displaying no significant difference ($P > 0.05$). The average sum of RMS EMG activity of the three muscles was therefore calculated (*i.e.*, quadriceps RMS EMG activity) to provide an index of overall quadriceps neural drive (Billau et al. 2011), and was expressed as a percentage of the maximal RMS EMG activity produced during the initial sprint bout achieved in each condition (Billaut et

al. 2013). The percentage decrement score for the entire exercise protocol was calculated as follows: $\{1 - (\text{cumulated MPO}) / (\text{best MPO} \times 10)\} \times 100$ (Girard et al. 2011). To prevent pacing effects occurring during the repeated-sprint exercise protocol, participants were required to achieve at least 95% of their criterion score (determined from the best of the two reference sprints at the end of the warm-up procedure). Mean power during the best of the reference sprints was 1066 ± 49 and 1053 ± 45 W for the NM vs. HY conditions, respectively). All participants satisfied the 95% criteria during the first sprint of the repeated-sprint exercise protocol for each testing session (NM and HY: 1122 ± 36 and 1075 ± 141 W), which suggests the participants did not adopt an anticipatory pacing prior to exercise in both trials.

Statistical Analysis

Values are expressed as means \pm SEM. Two-way repeated- measures ANOVAs were used to investigate the main effects of time, condition and possible interaction between these two factors for MPO, RMS EMG activity, physiological and perceptual data. To assess assumptions of variance, Mauchly's test of sphericity was performed using all ANOVA results. A Greenhouse-Geisser correction was performed to adjust the degree of freedom if an assumption was violated, while post hoc pairwise-comparisons with Bonferroni-adjusted P values were performed if a significant main effect was observed. For each ANOVA, partial eta-squared was calculated as measures of effect size. Values of 0.01, 0.06 and values above 0.14 were considered as small, medium and large, respectively (Cohen 2013). All statistical calculations were performed using SPSS statistical software V.21.0 (IBM Corp., Armonk, NY, USA). Statistical significance was set at $P < 0.05$.

RESULTS

RSA and Neural Drive

MPO during sprint 1 did not differ between NM and HY (1113 ± 41 vs. 1071 ± 45 W; $P = 0.085$; $ES = 0.33$; Figure 1-A). The MPO for all sprints was lower in HY compared to NM (841 ± 51 vs. 968 ± 52 W; $-13 \pm 8\%$; $P = 0.001$; $ES = 0.79$), and decrements in sprint performance were more pronounced (21.4 ± 3.7 vs. $13.2 \pm 2.7\%$; $P = 0.003$). When using sprint 1 as a reference, RMS EMG values decreased from sprint 2 onwards and declined by $\sim 17\%$ by sprint 10 ($P < 0.001$; $ES = 0.65$), with no difference between conditions ($P = 0.597$; $ES = 0.04$; Figure 1-B).

Physiological Responses

Heart rate increased from sprint 1 to sprint 10 ($P<0.001$; $ES=0.71$), with no difference between conditions ($P=0.238$; $ES=0.17$; Figure 2-A). Arterial oxygen saturation values were lower throughout the 10 sprints in HY compared to NM, (80.7 ± 0.9 vs. $95.6 \pm 0.6\%$; $P<0.001$; $ES=0.98$; Figure 2-B). Immediately after the RSA test, blood lactate concentrations were higher in HY compared to NM (11.9 ± 0.4 vs. 9.9 ± 0.9 mmol.L⁻¹; $P=0.05$; $ES=0.36$).

Sense of Effort and Perceptual Responses

After each of the ten sprints, all participants confirmed that all efforts were truly maximal. All perceptual responses increased across sprints 1 to 10 ($P<0.001$), with higher ratings of difficulty breathing (7.0 ± 0.4 vs. 5.6 ± 0.5 ; $ES=0.90$), lower-limb heaviness (7.9 ± 0.5 vs. 6.3 ± 0.7 ; $ES=0.86$) and overall perceived exertion (6.7 ± 0.6 vs. 5.3 ± 0.5 ; $ES=0.86$) for the average of sprints 1 to 10 in HY vs. NM (Figure 3).

DISCUSSION

Summary of Main Findings

Through the use of severe hypoxia exposure, exacerbating physiological perturbations, the aim of the present study was to describe the time course of changes in exercise-related sensations or perceptions and the regulation of neural drive during a set of repeated, brief, maximal cycle sprints. As expected, severe HY caused earlier and larger reductions in MPO compared to the sea-level control condition. However, despite both physiological responses and perceptions of fatigue being exacerbated under HY, our novel finding is that reductions in quadriceps neural drive were comparable between conditions. Our data therefore suggest that higher-than-normal exercise-related sensations or perceptions coincide with fatigue during repeated, short-duration cycle sprints. These sensations, however, appear to play a minimal role in regulating quadriceps neural drive, at least when the number of sprints to perform is known beforehand.

Repeated Sprint Performance

In accordance with previous cycle- (Balsom et al. 1994; Billaut and Smith 2010; Billaut and Aughey 2013) and run-based studies (Bowtell et al. 2014; Goods et al. 2014; Girard et al. 2015) that have explored fatigue-induced modification of RSA with severe hypoxia exposure (*i.e.*, $FiO_2 < 0.14$), earlier and more pronounced reductions in mechanical performance (MPO) clearly (*i.e.*, large effect size) occur in HY vs. NM. However, methodological differences between studies, which include differing work-to-rest ratios,

ergometers and/or participants physical capacities, and the nature/mode of hypoxic exposure, considerably limits the ability to compare previous findings.

Due to efficient blinding to the conditions, participants in the present study were not fully aware of the task requirements or the rate at which given subjective feelings would develop. As a consequence it appears they didn't adopt an anticipatory pacing strategy prior to exercise in both trials, as evidenced by the similar power output and RMS EMG activity during the initial sprint repetition (Figure 1). This is also reinforced by the observation that performance during the initial sprint was even slightly higher than during the reference (*i.e.*, warm-up) sprint. For single sprint efforts < 60 s, performance does not seem to be adversely affected by breathing normobaric hypoxic mixture (FiO_2 0.13; Weyand et al. 1999). It should be noted, however, that there was a tendency towards lower MPO values during sprint 1 was observed in HY *vs.* NM. The possible deleterious effect of a 30-min exposure to hypoxia (*i.e.*, 10 min wash in, constant-load warm-up cycling followed by sub-maximal and maximal efforts), potentially affecting effort perception at the commencement of the RSA protocol (Christian et al. 2014), shouldn't be overlooked.

Physiological Perturbations

The reduction in O_2 availability in HY was sufficient to exacerbate the physiological response during the present RSA test, as demonstrated by a higher heart rate (+2%; albeit not significantly different) and end-exercise blood lactate concentrations (+2 mmol.L⁻¹) when compared to NM. Although lower arterial oxygen saturation values are always associated with hypoxic exposure during RSA protocols, the extent to which heart rate responses and/or blood lactate concentrations are exaggerated compared to sea-level controls is more circumstantial. In the RSA literature, hypoxic exposure has been associated with similar (Billaut et al. 2013; Girard et al. 2015) or increased (Balsom et al. 1994; Goods et al. 2014) heart rate responses and/or blood lactate concentrations *vs.* sea-level. Such discrepancies between studies may relate to methodological differences (*i.e.*, different work-to-rest ratios) that may have a differing influence on the oxidative *vs.* glycolytic component when RSA protocols are conducted with or without hypoxic exposure.

The present study failed to observe any additional impact of exercise on arterial oxygen desaturation in both trials, which is known to be mediated via the effects of pH on the oxyhemoglobin dissociation curve and low FiO_2 (Wasserman et al. 1967). Previous RSA studies conducted at sea-level demonstrate time-dependent arterial oxygen desaturation (ranging 3-5%; Billaut and Smith 2010; Bowtell et al. 2014; Smith and Billaut 2010), while others have observed a pattern of desaturation in hypoxic conditions only (Billaut et

al. 2013; Girard et al. 2015). Compared to the aforementioned studies, a shorter sprint duration (4 s vs. 5-10 s) and a greater work-to-rest ratio (1:7 vs. 1:3-5) in the present study may not have been sufficient enough to challenge the blood bicarbonate buffering capacity through the excess accumulation of H^+ that would ultimately lower pH and impair the hemoglobin affinity to O_2 .

Perception of the Sensations of Fatigue

To the best of our knowledge, this is the first study to explore the sensations of fatigue during a RSA test, while differentiating between the “sense of effort” and “subjective perceptions”. As a consequence of the task requirements (*i.e.*, maximal sprints), the sense of effort was maximal from the initial sprint and remained maximal throughout the entire RSA protocol. Hence, after each of the ten sprints when asked the question: “*Was that a maximal effort?*” participants systematically scored 10 on the subjective scale. Despite this, overall perceived exertion during the initial sprints were quite low, presumably in accordance with low sensory inputs from peripheral sources such as the heart, lungs and active muscles. This corroborates observations of a dissociation between the sense of effort associated with various cycling tasks (*i.e.*, self-paced sub-maximal constant subjective-effort cycling and brief progressive sub-maximal and maximal cycling) and the accompanying subjective perceptions (Christian et al. 2013), and extends these observations to repeated sprints. In this study, we used Lickert scales with anchor points ranging from 0 or “nothing at all” to 10 or “maximal”. An alternative would have been to use a visual scale of 10 cm without any graduation (horizontal line ranging from nothing at the left to maximal at the right). Whether one scale is better than the other is a contentious issue, but it was proposed that Lickert responses are easier to administer and interpret (Bolognese et al. 2003). When evaluating the extent of delayed onset of muscle soreness in the plantar flexor muscles in response to a downhill walking exercise it is interesting to observe a statistical similarity in the responses of the two subjective scales (Racinais et al. 2008).

Globally, the reported values for difficulty breathing, lower-limb heaviness, and overall perceived exertion values were 31-36% higher in HY vs. NM, indicating exacerbated exercise-related sensations in an O_2 -deprived environment. Despite this, the repetition of “all out” efforts with incomplete recovery led to a progressive increase in perceptual scale values (*i.e.*, with a similar rate between conditions) and without reaching maximal scores during the last sprint (*i.e.*, values ranging 7 to 9, Figure 3). Contrastingly, when RPE was used as a single measure of the global “sense of effort” during cycle-based RSA protocols including the same number of sprints, smaller (*i.e.*, not significant) differences for the average of all sprints

and similar near-maximal end-exercise values were reported with or without hypoxic exposure (Billaut et al. 2013) and knowledge of sprint number (Billaut et al. 2011). This may indicate the presence of a conscious pacing strategy in which participants deliberately reduced their effort and subsequent muscle recruitment in anticipation of excessive and intolerable discomfort. However, while our participants experienced greater levels of perceived difficulty breathing, lower-limb heaviness, and overall perceived exertion in HY, the present protocol did not induce maximal levels for exercise-related sensations. Importantly, our exercising participants didn't receive any precise information about the number of sprints already completed or remaining, which may have prevented the use of a conscious pacing strategy. Therefore, participants may not have anticipated peripheral discomfort to exceed intolerable levels (Amann, 2011) and, therefore, chose not to reduce their involvement in the task as evidenced by the stable, maximal 'sense of effort' values that participants reported throughout the protocol.

Regulation of Neural Drive

In line with previous findings (Smith and Billaut 2010; Billaut et al. 2013; Bowtell et al. 2014; Girard et al. 2015), RMS EMG activity signals decreased significantly over time in both trials, indicating progressive suboptimal neural drive to the quadriceps across sprints. As maximal efforts are repeated, the surface EMG activity drops progressively because motoneurons become less responsive to synaptic input, receive decreased afferent feedback from muscle spindles, and/or receive insufficient descending drive due to supraspinal fatigue (Taylor and Gandevia 2008). An important ionotropic synaptic input received by the motoneuron signal under fatigue comes from the increased firing of group III/IV muscle afferents. This afferent signal not only interacts with the autonomic nervous system, but also contributes to conscious sensations of muscle discomfort and fatigue (Pollak et al. 2014). However, in the absence of any measure of sarcolemmal excitability for the purpose of RMS EMG activity normalization (*i.e.*, measuring the peak-to-peak amplitude of an electrically-evoked M-wave), the influence that potential peripheral alterations might have on changes in EMG activity amplitude cannot be ignored.

In the present study, a similar magnitude of EMG activity decrease occurred in HY *versus* NM. This is consistent with the data obtained during both cycling (Smith and Billaut 2010) and running (Bowtell et al. 2014; Girard et al. 2015) RSA tests conducted under similar hypoxic conditions. This contrasts, however, with observations made by Billaut et al. (2013) who reported greater reductions in EMG activity when ten male team-sport athletes performed fifteen 5-s sprints interspersed with 25 s of rest during acute moderate

hypoxia (FiO₂ 0.14) compared to sea level conditions. The authors proposed that the exacerbated decline in RMS EMG activity was likely due to “*muscle de-recruitment by the central nervous system in anticipation of the end of the exercise (in order to) limit the development of locomotor muscle fatigue*”. This down regulation of muscle recruitment in order to prevent excessive peripheral fatigue development is in line with the inhibitory afferent feedback model, which postulates that increased afferent feedback from fatigued muscles regulates the level of muscle recruitment in order to prevent a critical level of peripheral fatigue from being exceeded (Amann 2011). Recent RSA observations arising from the induction of pre-existing locomotor muscle fatigue (*i.e.*, following a 10 min neuromuscular electrical stimulation protocol of the quadriceps: Smith and Billaut 2010) and shorter recovery durations (*i.e.*, severe exercise-to-rest ratio: Hureau et al. 2016), leading to reductions in power and normalised quadriceps RMS EMG activity, support this assumption. In the present study, despite exaggerated physiological responses (and presumably greater afferent feedback), failure to observe a greater attenuation of muscle recruitment in HY vs. NM fails to support this contention. The difference in performance observed here may well be primarily attributed to perturbations within the skeletal muscles rather than the brain.

CONCLUSIONS

Despite severe hypoxia exposure exacerbating both physiological and perceptual responses, and producing a more pronounced performance decrement during brief maximal repeated cycle sprints, this was not accompanied by exaggerated down-regulation of neural drive of the quadriceps musculature. Our data therefore suggest that higher-than-normal exercise-related sensations or perceptions are signs of fatigue during repeated, short-duration cycle sprints. These sensations, however, appear to play a minimal role in regulating quadriceps neural drive, at least when the number of sprints to perform is known beforehand. There may be different responses when repeated sprints are performed on a cycle vs. a treadmill ergometer (Rampinini et al. 2016), or by team-sport players on the field (multi-directional involving decision-making actions), which implies that our conclusions must remain specific to the context of this study.

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FIGURE LEGENDS

Figure 1 – Mean power (A) and normalised quadriceps Root Mean Squared electromyography (EMG, B) during the ten repeated cycling sprints.

*Values are mean \pm SEM for 9 participants. Data is presented for normoxia (FiO₂ 0.21) and hypoxia (FiO₂ 0.13). C, T, and I, respectively refer to ANOVA main effects of condition, time and the interaction between these two factors, and are followed by the associated P-value and, in brackets, the effect size values. * significantly different from sprint 1 ($P < 0.05$). † significantly different from normoxia ($P < 0.05$).*

Figure 2 – Heart rate (A) and arterial oxygen saturation (B) during the ten repeated cycling sprints.

*Values are mean \pm SEM for 9 participants. Data is presented for normoxia (FiO₂ 0.21) and hypoxia (FiO₂ 0.13). C, T, and I, respectively refer to ANOVA main effects of condition, time and the interaction between these two factors, and are followed by the associated P-value and, in brackets, the effect size values. * significantly different from sprint 1 ($P < 0.05$). † significantly different from normoxia ($P < 0.05$).*

Figure 3 – Difficulty breathing (A), lower-limb heaviness (B) and overall perceived exertion (C) during the ten repeated cycling sprints.

*Values are mean \pm SEM for 9 participants. Data is presented for normoxia (FiO₂ 0.21) and hypoxia (FiO₂ 0.13). C, T, and I, respectively refer to ANOVA main effects of condition, time and the interaction between these two factors, and are followed by the associated P-value and, in brackets, the effect size values. * significantly different from sprint 1 ($P < 0.05$). † significantly different from normoxia ($P < 0.05$).*