


The effect of prior knowledge of test endpoint on non-local muscle fatigue

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Received: 9 October 2016 / Accepted: 23 December 2016 / Published online: 27 February 2017
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Abstract

Introduction If the work duration or volume is known, it is common for individuals to anticipate this challenge by pursuing a strategy that may decrease the initial force output and maintain a force level that ensures a force reserve towards the end of the task. However, it is unknown whether this is a global strategy that is transferred to a non-exercised muscle following fatigue of a contralateral homologous muscle.

Methods To clarify if prior knowledge of task endpoint has an effect on non-local muscle fatigue (NLMF), 15 male participants (22.4 ± 3.8 years) completed four conditions: (1) KNtest > fatigue (known endpoint after fatigue), (2) UNKtest > fatigue (unknown endpoint after fatigue), (3) KNtest > control (known endpoint without fatigue), (4) UNKtest > control (unknown endpoint without fatigue). For fatigue conditions, a maximal intensity, unilateral knee extension protocol was completed (two sets of 100 s maximal voluntary isometric contractions (MVIC) with 60 s rest between), whereas the control condition involved rest (260 s). The participants were either informed (known (KN) conditions) or not informed (unknown (UNK) conditions) of the duration of a post-intervention strength–endurance test (contralateral knee extension MVIC, ≥ 30 s).

Results During the strength–endurance test, the UNKtest > fatigue displayed meaningful decreases in force (UNKtest > fatigue 10–12% over first 30 s), which was

largest at the 25–30-s period (UNKtest > fatigue 7.4–41.1% from 25 to 1930s) compared to KNtest > fatigue and KNtest > control conditions, respectively.

Conclusion Prior knowledge of task endpoint can modify NLMF and affect pacing strategies.

Keywords Endurance · Force · Strength · Crossover fatigue · Pacing · Electromyography

Abbreviations

BF	Biceps femoris
CMEP	Cervicomedullary motor evoked potential
CNS	Central nervous system
EMG	Electromyography
ES	Effect size
KNtest > fatigue	Known test endpoint after fatigue protocol
KNtest > control	Known test endpoint without fatigue
MEP	Motor evoked potential
Mmax	Maximal compound muscle action potential
MVIC	Maximal voluntary isometric contraction
NLMF	Non-local muscle fatigue
RF	Rectus femoris
RMS	Root mean square
SD	Tandard deviation
TMS	Transcranial magnetic stimulation
TMEP	Horacic motor evoked potential
UNKtest > control	Unknown test endpoint without fatigue
UNKtest > fatigue	Unknown test endpoint after fatigue
VL	Vastus lateralis
VM	Vastus medialis

Communicated by Nicolas Place.

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Introduction

Crossover or non-local muscle fatigue (NLMF) occurs when fatiguing a muscle group produces fatigue effects in contralateral or other non-exercised muscles (Halperin et al. 2014b, d, 2015; Kawamoto et al. 2014; Martin and Rattey 2007; Paillard et al. 2010). NLMF effects have been primarily attributed to neural and psychological factors (Halperin et al. 2015). Central or neural fatigue refers to an activity-induced decline in the ability to activate a muscle voluntarily and results from a failure of the central nervous system to excite and drive motor neurons (Gandevia 2001). Psychologically, there is a natural response to cease activity when it becomes uncomfortable such as with muscle fatigue (Halperin et al. 2015). Fatigue-induced central nervous system deficits in muscle activation (Behm 2004; Behm et al. 2002, 2013) or the psychological effects of fatigue-induced discomfort, pain or sustained attention (Amann et al. 2013; Marcora et al. 2009; Pageaux et al. 2013, 2014) affect not only the fatigued exercised muscles but can also influence non-local or non-active muscle force output (Halperin et al. 2015). It is typically accepted that during short-duration, high-intensity activities where maximal efforts are necessary, the speed, force, and power output gradually decline as a function of the length of the activity (Chidnok et al. 2013). However, studies that manipulate participants' prior knowledge of task endpoint have shown that the extent or rate of decline can be altered (Billaut et al. 2011; Halperin et al. 2014a, c). Research tends to support a strong influence of task endpoint knowledge on the affected muscle's performance (i.e., force, power, speed). Studies where participants were deceived as to the true task endpoint revealed higher force and EMG outputs, with shorter than expected duration exercise periods (Billaut et al. 2011; Halperin et al. 2014a, c). While research has shown that prior knowledge of task endpoint affects localized (the exercised or fatigued muscle) force output, there are no studies that have examined whether task endpoint knowledge impacts NLMF similarly.

In their central governor model, St. Clair Gibson and Noakes (2004) proposed that before and continuously during exercise, the brain subconsciously calculates the metabolic cost required to complete an exercise task given prior experience, and under the influence of environmental conditions and the current physical state (St. Clair Gibson and Noakes 2004). Two strategies that are often used when confronted with performing a fatiguing exercise task are anticipatory force reductions and pacing (force reserve) (Halperin et al. 2014c, d; Reid et al. 2016). Although, participants' intent was to execute maximal voluntary isometric contractions (MVIC), MVIC force was consistently and significantly reduced in anticipation of a fatiguing protocol in adult

men (Halperin et al. 2014d), women (Halperin et al. 2014c) and adolescent girls (Reid et al. 2016). Pacing is a learned strategy employed during prolonged contractions or activities with a known endpoint (closed loop). Pacing strategy involves a "U"-shaped force or performance (e.g., running or cycling velocity) curve typified with an increased force or performance output at the end of the bout (force reserve) (Halperin et al. 2014c, d; Reid et al. 2016). Given the framework of St. Clair Gibson and Noakes (2004) theory, an inefficient pacing template is more likely in an unknown task endpoint situation, which would negatively impact performance. The typical force profile (pacing strategy) may be altered with NLMF when comparing known vs. unknown task endpoint conditions. However, these variables have not been examined with NLMF. With a fatigue-induced reduction in central drive (Gandevia 2001; Behm 2004), the knowledge or lack of knowledge of task endpoint could alter psychological motivation further exacerbating central factors involved with NLMF.

Thus, the objective of the present study was to determine the presence and extent of NLMF using known and unknown testing endpoints. It was hypothesized that an unknown test endpoint would interfere with the implementation of anticipatory and pacing (i.e., force reserve) strategies and lead to a greater expression of NLMF.

Methods

Participants

Fifteen recreationally trained male participants were recruited for this study (22.4 ± 3.8 years; 1.80 ± 0.052 m; 77.9 ± 10.4 kg). Recreationally trained was defined as participating in at least two activity sessions a week for the past 6 months. Prospective participants who reported neurological or musculoskeletal complications involving knee structures such as surgery or injury, or cardiovascular conditions such as high blood pressure were excused from the experiment. All participants filled out the Physical Activity Readiness Questionnaire+ (PAR-Q+) form (CSEP 2011) and provided written informed consent in accordance with ethics approval to further confirm suitability for the study. Ethical approval for the study was obtained from the Human Research Ethics Authority of the institution (#15.066). Participants were instructed to avoid strenuous activity and abstain from alcohol, caffeine or nicotine consumption for a 24-h period prior to participation. Leg dominance was determined by asking what foot the individual kicks a ball with (Oldfield 1971).

Study design

A randomized crossover study design was employed to examine the acute effects of unilateral knee extensor muscle fatigue on the performance of the contralateral homologous muscle. To determine if knowledge or lack of knowledge of task endpoint impacted NLMF in the contralateral homologous muscle, this study examined single and prolonged (30-s MVIC strength–endurance test) MVIC forces and electromyography (EMG) of the vastus lateralis (VL), vastus medialis (VM), rectus femoris (RF) and biceps femoris (BF), pre- and post-intervention (2 sets of 100-s maximal intensity unilateral knee extension fatigue protocol, or rest). Participants were scheduled for four separate testing sessions, each lasting approximately 45 min and separated by at least 48 h. Experimental conditions were presented randomly and included (1) KNtest > fatigue: known test endpoint (30 s) after pre-fatigue of contralateral knee extensors, (2) UNKtest > fatigue: unknown test

endpoint after pre-fatigue of contralateral knee extensors, (3) KNtest > control: known test endpoint (30 s) with no prior exercise of contralateral knee extensors and (4) UNKtest > control: unknown test endpoint with no prior exercise of contralateral knee extensors. A schematic diagram of the experimental design is illustrated in Fig. 1.

Protocol

Each session started with the placement of surface EMG electrodes on the VL, VM, RF, and BF muscles of the non-dominant leg. EMG activity was not monitored in dominant limb (the limb subjected to the fatigue intervention). Self-adhesive Ag/AgCl electrodes (Meditrace™ 130 ECG conductive adhesive electrodes) were placed according to previously supported protocols (Hermens et al. 2000; Paddock and Behm 2009; Kawamoto et al. 2014). The surface electrodes were placed at the mid-point of the anterior superior iliac spine and the

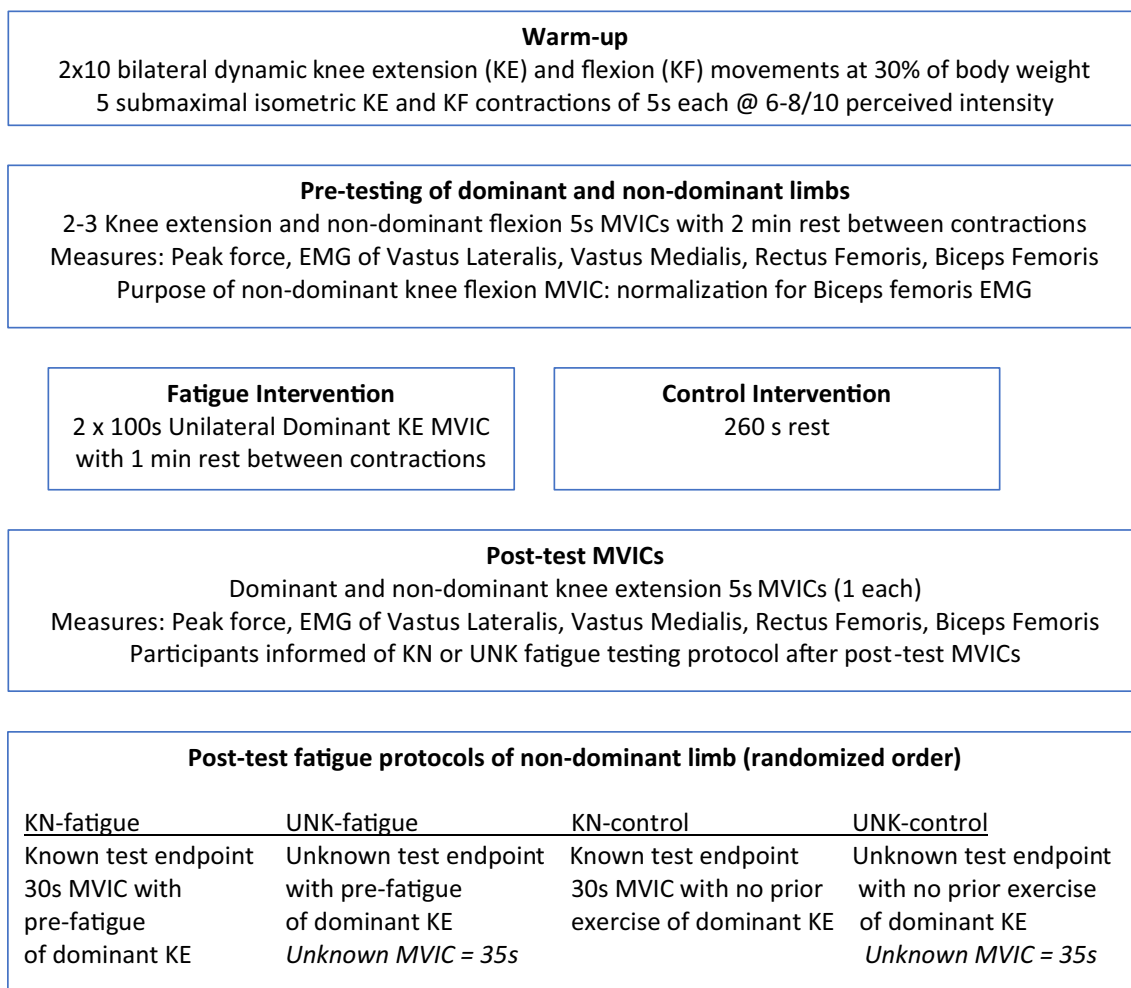


Fig. 1 Experimental protocol

patella for the RF, 80% along the line from anterior superior iliac spine to the joint space in front of the anterior border of the medial ligament for the VM, and 66% on the line between anterior superior iliac spine and lateral side of the patella for the VL. The mid-point between the gluteal fold and popliteal space was used for the BF. The electrodes were placed 2 cm apart (centre to centre) and parallel to the direction of the muscle fibers. The ground electrode was placed on the lateral femoral epicondyle. The skin was prepared prior to electrode placement by shaving the area, rubbing with sandpaper and cleansing with an isopropyl alcohol swab to ensure minimal skin resistance.

To ensure an adequate signal-to-noise ratio, an inter-electrode impedance of $<5\text{ k}\Omega$ was obtained prior to testing. The EMG signal acquisition system (Biopac System Inc., DA 100: analog–digital converter MP150WSW; Holliston, Massachusetts) recorded all signals at a sampling rate of 2000 Hz. All EMG signals were filtered with a Blackman -61 dB band-pass filter between 10 and 500 Hz, amplified (bi-polar differential amplifier, input impedance = $2\text{ M}\Omega$, common mode rejection ratio $>110\text{ dB}$ min (50/60 Hz), gain $\times 1000$, noise $>5\text{ }\mu\text{V}$), and analog-to-digitally converted (12 bit) for storage and analysis on a personal computer. A commercially designed software program (AcqKnowledge III, Biopac Systems Inc.) was used for the establishment of signal parameters and for data analysis.

Participants were then seated in a knee extension machine (Modular Leg Extension, Cybex International, Medway, MA, USA) with the hips and knees fixed at 90° and 83° , respectively. A knee flexion position of 83° was based on the constraints of the leg extension device. A five-point harness was placed around the waist and shoulders of the participants and they were instructed to cross their arms across their chest to minimize upper body involvement. The dominant and non-dominant ankles were inserted into padded ankle cuffs and attached to strain gauges (Omega Engineering Inc., LCCA 250, Don Mills, Ontario) with non-extensible straps. The straps and strain gauge were secured to the leg extension machine through a custom-built apparatus that allowed a 90° angle to be maintained between the straps and the participants' lower shin. Once properly positioned on the knee extension machine, subjects performed a warm-up consisting of two sets of ten dynamic bilateral knee extensions with a load approximately equal to 30% of the participant's total body mass. Following this procedure, they performed five submaximal unilateral isometric knee extensions lasting 5 s each, with both the right and left legs (at 83° ; 0° being full extension). The desired intensity for these isometric contractions was described as a force equal to 6–8/10 on a scale of one to ten, where 10/10 is maximal effort.

Immediately after this warm-up, participants performed a MVIC protocol with both legs. Each leg performed two unilateral isometric knee extension MVICs and if the difference between the two MVICs was more than 5%, a third MVIC was performed. Each MVIC was performed for 5 s with 2-min rest. Knee flexion MVICs (5 s) were then performed for the non-dominant leg for normalization of BF EMG. Following the MVIC pre-tests, a fatigue protocol or rest (260 s) was presented as an intervention depending on the experimental condition. The dominant leg was used for all fatiguing protocols and the participants were encouraged to keep the contralateral leg relaxed during leg contractions. The EMG of the contralateral leg was monitored throughout the fatigue protocol to ensure it was relaxed ($<5\%$ MVIC EMG). Data for both legs were saved throughout the fatigue protocol and tests for later analysis.

The fatigue protocol utilized for this study has been shown to elicit NLMF in contralateral knee extensors (Doix et al. 2013; Halperin et al. 2014b). With the same setup as for the MVIC testing, the dominant leg performed a continuous knee extension MVIC for two sets of 100 s each, separated by 1 min of rest.

Immediately upon completion of the fatigue protocol or rest period (control), the dominant and contralateral leg performed single 5-s knee extension MVIC in the same order as the pre-test. Dominant knee extensions were performed first as the fatigue intervention involved the dominant limb and thus it was more efficient to maintain the same strain gauge and cuff arrangement. The single MVICs were followed immediately by the known or unknown test endpoint, 30-s MVIC strength–endurance test dependent upon the experimental condition. Participants were informed of the intervention (KN or UNK) after the post-intervention single MVIC and prior to the start of the strength–endurance test. For the KN conditions, the participant was notified that they would have to hold a knee extension MVIC for 30 s. For the UNK conditions, the participant was instructed to hold a knee extension MVIC until they reached a certain point of fatigue, at which time they would be told by the researcher to stop. Unbeknownst to the participant, the point of fatigue where participants were stopped during the UNK test was set at 60% of the initial MVIC. If during the UNK condition, force decreased to 60% MVIC prior to 30 s, participants were not informed and they continued for 35 s so that equal contraction durations could be compared and analyzed. When the strength–endurance test was performed with a known endpoint (KN), a monitor was used to allow subjects to see the time, but they were blinded to their force output. When participants did not have prior knowledge of test endpoint (UNK), they were kept blinded to the test duration and force output. Participants were told to “go hard” at 5-s intervals throughout all fatigue protocols and tests to ensure

consistent encouragement. The hypotheses and expectations based on prior research were not explained to the subjects with the hope of reducing bias.

Measurements and data analysis

MVIC tests pre- and post-intervention were included to allow for comparisons of peak force production based on previous research (Hearn et al. 2009; Kawamoto et al. 2014). For each muscle, the mean amplitude of the root mean square (RMS) of the EMG signal was calculated over a 2-s period that included the peak force output (1 s prior, and 1 s after peak force). For condition comparisons, the peak force or EMG RMS from pre-MVICs were used to normalize values from post-MVICs (post/pre) and strength–endurance tests (5 s periods of mean MVIC/pre-MVIC).

The KN and UNK endpoint tests were directly compared over the initial 30 s of the strength–endurance test. EMG RMS signal and force output data from six sequential 5-s periods were used for analysis (mean from each period). The duration of the UNK endpoint test (time to 60% MVC) was monitored and compared between fatigue and rest conditions.

Statistical analyses

The inappropriateness of null-hypothesis significance testing (Hopkins 2004) for assessing clinical or practical importance has been noted in the fields of psychology (American Psychological Association 2001), medicine (Asai 2002), sports medicine (Hopkins et al. 2009), as well as statistics (Ludbrook and Dudley 1998). Hence, to expose meaningful differences in the present study, a magnitude-based approach for analysis and reporting of results was utilized. Effect sizes (ES) were reported along with the percent likelihood that the observed effect size was larger than a small effect size (meaningful difference). In accordance with previous research, Cohen's *d* values of 0.2, 0.6 and 1.2 were used as thresholds for small, medium and large effect sizes (Drinkwater et al. 2007). The percent likelihood that the observed effect size was larger than the smallest worthwhile change (ES: 0.2) was calculated based on previous methods (Drinkwater et al. 2007; Hopkins 2004, 2009; Page 2014). Chances of a meaningful difference were classified qualitatively as follows: <1%, almost certainly not; <5%, very unlikely; <25%, unlikely; 25–75%, possible; >75%, likely; >95%, very likely; >99% almost certain. The $\geq 75\%$, likely, classification was used as the threshold for a meaningful difference (Drinkwater et al. 2007). The normality and homogeneity of variances within the data were confirmed with the Shapiro–Wilk and Levene's tests, respectively.

Results

Normalized single (5 s) MVIC Peak force measures

Dominant limb: substantial localized muscle fatigue was demonstrated with greater MVIC forces in the control vs. fatigue conditions. The dominant leg demonstrated higher post-MVIC force in the KNtest>control condition compared to the two conditions with prior fatigue: 44.3% greater than KNtest>fatigue (100% likely; ES: 1.3), and 30.4% higher than UNKtest>fatigue (100% likely; ES: 1.1). The UNKtest>control condition also demonstrated higher peak normalized force measures compared to both fatigue conditions [34.1% higher than KNtest>fatigue (ES: 0.97); 21.1% greater than UNKtest>fatigue (ES: 0.89)] (100% likely) (Fig. 2).

Contralateral limb: demonstrating condition-specific crossover force differences, knee extension MVIC force of the non-exercised, non-dominant limb was “likely” to be higher during the UNKtest>fatigue condition compared to both control conditions (85% likely vs. KNtest>control, ES: 0.58; 93% likely vs. UNKtest>control, ES: 0.96) (Fig. 2).

Contralateral normalized single 5-s MVIC EMG measures

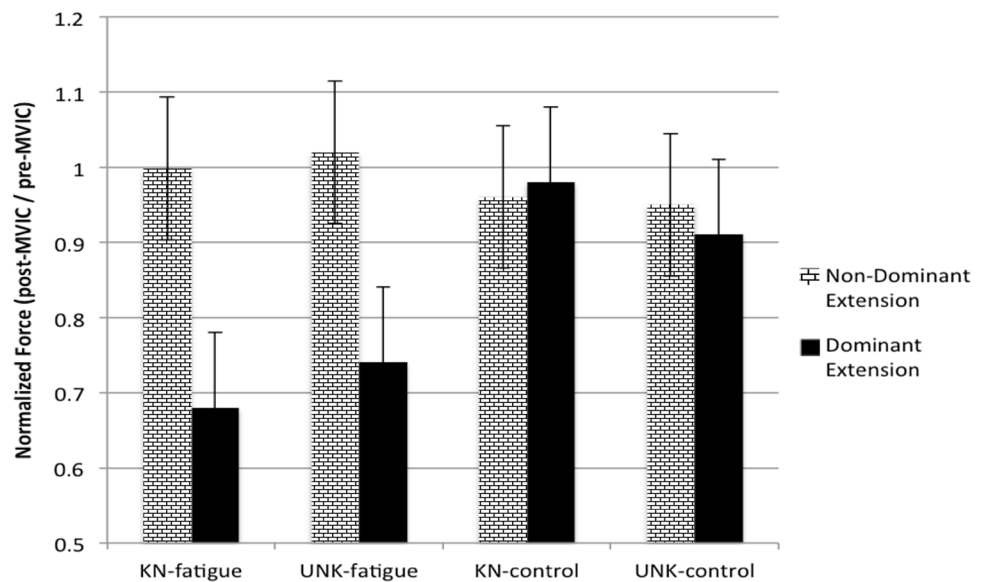
Similar to non-dominant knee extension MVIC force, UNKtest>fatigue demonstrated higher EMG activity compared to KNtest>control and UNKtest>control for all quadriceps muscles (80–95% likely). Although no meaningful difference in force was noted between KNtest>fatigue and KNtest>control, all muscle groups examined were 78–90% likely to exhibit higher EMG activity during KNtest>fatigue as well. It was also “likely” that the RF and VL muscles would be less active during KNtest>fatigue compared to UNKtest>fatigue (96 and 84%, respectively) (Table 1; Fig. 3).

Contralateral strength–endurance test measures

Normalized mean force

There was evidence for a prior knowledge effect. KNtest>fatigue was “likely” to generate higher forces in the final two periods (20–30s of strength–endurance test) compared to UNKtest>fatigue (75 and 80%, respectively) (Table 2). The strongest crossover fatigue effect was demonstrated when comparing the UNKtest>fatigue and KNtest>control conditions, as meaningful differences were “likely” or “very likely” from period 2 through 6 (86–97% likelihood) (Table 2). KNtest>control produced 12% higher force than UNKtest>fatigue during

Fig. 2 Mean and standard deviation of the MVIC force: peak normalized force across conditions for dominant and non-dominant (non-exercised) leg extensions



the strength–endurance test (average of first 30 s), which was greatest at period 6 (21.6%) (Table 2). The UNKtest>fatigue condition also exhibited lower forces in period 3 compared to UNKtest>control (75% likelihood) (Table 2), further supporting a crossover fatigue effect. There was no evidence of crossover fatigue with the known endpoint condition as no meaningful differences were shown between the KNtest>fatigue periods and the two control conditions (KNtest>control and UNKtest>control). Prior knowledge of endpoint did have an effect on pacing (force reserve) strategy as the KNtest>control condition demonstrated higher forces in the final period compared to UNKtest>control (90% likely) (Table 2; Fig. 4).

Both fatigue conditions demonstrated “very likely” or “almost certain” drops in force from period 1 to 2. THE CONTROL CONDITION did not exhibit the same initial drop in force (shaded areas of Table 2). In addition, KNtest>control was better able to recover force production at the end of the test (period 6: shaded area of Table 4) (Fig. 4).

Vastus medialis (VM) normalized RMS EMG

Analysis of VM RMS EMG data provided stronger indications that crossover fatigue had occurred. Similar to force, the VM EMG activity with KNtest>fatigue was “likely” to be higher during periods 4–6 compared to UNKtest>fatigue, lower activity than both control conditions at periods 2 and 3 as well as lower activity during periods 5 and 6 compared to KNtest>control. The UNKtest>fatigue condition was likely to have lower VM activity than KNtest>control throughout periods 2–6 as well as compared to UNKtest>control periods 1–6. Finally, in agreement with the force rebound

previously reported, KNtest>control was likely to have higher VM EMG activity than UNKtest>control during period 6 (Tables 3–5).

Comparisons of VM RMS EMG measures within each condition showed divergences with force comparisons (shaded areas of Tables 3–5). The first period of KNtest>fatigue was “likely” to have lower VM activity than period 6. KNtest>control demonstrated the greatest changes throughout the 30-s test with VM EMG activity during period 1 being lower than periods 4–6. No significant changes in VM activity throughout the strength–endurance test were revealed for UNKtest>fatigue or UNKtest>control conditions.

Vastus lateralis (VL) normalized RMS EMG

Demonstrating further evidence of a prior knowledge effect on a force and activation reserve, VL RMS EMG activity during KNtest>fatigue was “likely” to be higher than UNKtest>fatigue during period 6 and lower than KNtest>control from periods 2–6. KNtest>fatigue was “likely” to be lower than UNKtest>control at period 2, but higher at period 6. Similarly, KNtest>control was “likely” and “almost certain” to have higher VL activity than UNKtest>control during periods 5 and 6, respectively. UNKtest>fatigue was “likely” or “almost certain” to be lower than KNtest>control from periods 2–6 (Tables 3–5).

When considering each condition separately over the testing periods, the KNtest>fatigue condition was “likely” to produce higher activity in period 1 compared to 2, but lower activity compared to 6. Within KNtest>control, period 1 was “likely” to have lower activity than period 5 and “almost certain” to have lower activity than period 6.

Table 1 Contralateral limb anticipation effect: means are reported (with SD in brackets) for force and RMS EMG of the contralateral (non-exercised) leg from the pre-MVIC test and period 1 of strength–endurance test

	Pre-MVIC	Period 1	Percent difference (%)	Effect size
KN-fatigue				
Force (kg)	54.25 (14.12)	46.69 (12.52)	–13.9	–0.64*
VM EMG (mV)	0.72 (0.3)	0.56 (0.24)	–22.5	–0.67*
RF EMG (mV)	0.68 (0.2)	0.51 (0.22)	–25.4	–0.79*
VL EMG (mV)	0.84 (0.44)	0.6 (0.37)	–27.9	–0.63*
BF EMG (mV)	0.072 (0.038)	0.067 (0.04)	–7.5	–0.13
UNK-fatigue				
Force (kg)	51.79 (12.21)	41.99 (10.03)	–18.9	–0.98*
VM EMG (mV)	0.75 (0.25)	0.56 (0.19)	–25.6	–1.02*
RF EMG (mV)	0.66 (0.18)	0.52 (0.16)	–21.7	–0.87*
VL EMG (mV)	0.75 (0.44)	0.58 (0.33)	–22.7	–0.52*
BF EMG (mV)	0.067 (0.028)	0.054 (0.019)	–18.9	–0.68*
KN-control				
Force (kg)	55.21 (13.76)	47.51 (12.7)	–13.9	–0.62*
VM EMG (mV)	0.76 (0.33)	0.58 (0.23)	–23.3	–0.77*
RF EMG (mV)	0.7 (0.22)	0.51 (0.2)	–27.5	–1*
VL EMG (mV)	0.76 (0.34)	0.56 (0.28)	–33.6	0.68*
BF EMG (mV)	0.076 (0.031)	0.061 (0.031)	–20.1	–0.49*
UNK-control				
Force (kg)	55.13 (12.73)	46.87 (14.45)	–15	–0.54*
VM EMG (mV)	0.72 (0.3)	0.6 (0.25)	–16.8	–0.48*
RF EMG (mV)	0.71 (0.21)	0.56 (0.19)	–20.5	–0.75*
VL EMG (mV)	0.85 (0.45)	0.65 (0.4)	–23.7	–0.51*
BF EMG (mV)	0.069 (0.024)	0.063 (0.032)	–8.6	–0.03

Percent difference and effect size comparisons between pre-MVIC and period 1 values are also provided. Asterisks (*) denote a $\geq 75\%$ likelihood to demonstrate a meaningful difference

Regardless of crossover fatigue, both unknown conditions failed to show significant changes (UNKtest > fatigue and UNKtest > control).

Rectus femoris (RF) normalized RMS EMG

Once again, higher activity was seen during KNtest > fatigue compared to UNKtest > fatigue at period 6. There was limited evidence of crossover fatigue as KNtest > fatigue was lower than UNKtest > control at period 2 only and UNKtest > fatigue was only “likely” to be lower than KNtest > control during period 6. Overall, performance of the RF appeared to be more stable across conditions than the other muscle groups (Tables 3–5).

Within conditions, crossover fatigue did appear to have a noticeable effect. The control conditions did not show any significant changes in activity throughout the test. Meanwhile, RF EMG activity during period 1 was “likely” to be greater than period 2 for KNtest > fatigue, and periods 4 and 6 for UNKtest > fatigue.

Biceps femoris (BF) normalized RMS EMG

Consistent with other muscle groups, BF activity was lower during UNKtest > fatigue compared to KNtest > control at periods 5 and 6 as well as the KNtest > fatigue and UNKtest > control conditions during periods 1–6 (Tables 3–5).

Within the UNKtest > fatigue condition, only period 2 was “likely” to be lower than period 1. Periods 5 and 6 were “likely” to be higher than period 1 within KNtest > control. Finally, UNKtest > control period 4 was higher than period 1.

Endurance time

There was a trivial (effect size = 0.15), unlikely (32%) chance for a difference in the mean endurance time for the UNKtest > fatigue condition (42.05 ± 5.3 s), compared to the UNKtest > control (43.83 ± 11.5 s) (Tables 4, 5).

Discussion

The findings demonstrate that prior knowledge of test endpoint had an impact on NLMF. Evidence for NLMF was apparent with force and muscle activation deficits with the strength–endurance test, which contrasted with the force potentiation that occurred with the initial, single MVIC post-test. During the strength–endurance test the UNKtest > fatigue condition progressively produced lower force (periods 5 and 6) and muscle activity (VM: periods 4–6; RF and VL: period 6) compared to KNtest > fatigue. In addition, UNKtest > fatigue

Fig. 3 Mean and standard deviation of the contralateral MVIC EMG RMS: normalized EMG measures (post-MVIC RMS/pre-MVIC RMS) for each muscle group across all conditions

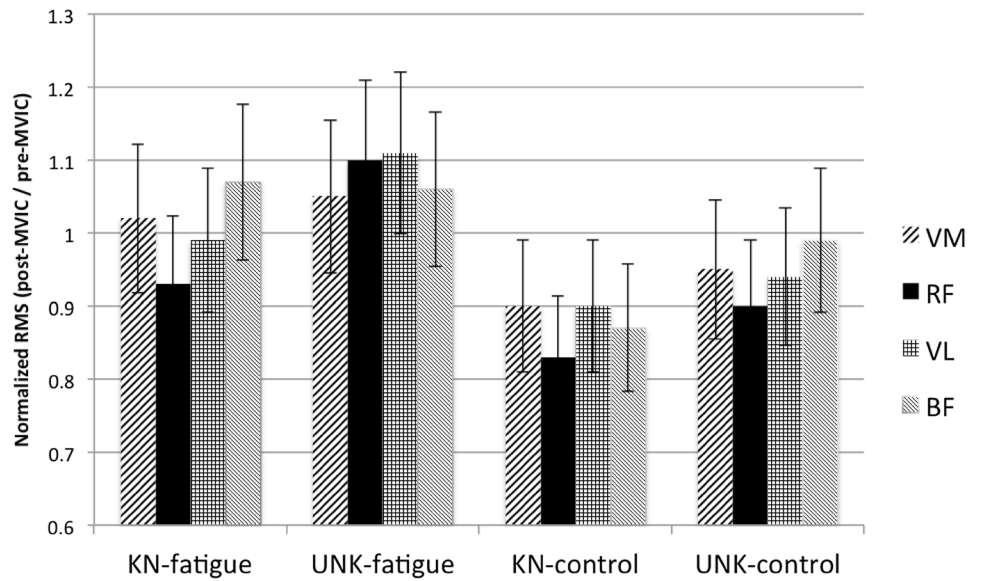


Table 2 Contralateral EMG activity with single 5-s MVIC pre- to post-intervention

	KN-fatigue vs. UNK-fatigue	KN-fatigue vs. KN-control	KN-fatigue vs. UNK-control	UNK-fatigue vs. KN-control	UNK-fatigue vs. UNK-control	KN-control vs. UNK-control
Vastus lateralis	-0.69*	0.50	0.27	0.61*	0.52*	-0.35*
Vastus medialis	0.07	0.53*	0.41	0.57*	0.52*	-0.58*
Rectus femoris	-0.81*	0.59*	0.17	0.92*	0.68*	0.29
Biceps femoris	0.11	0.65*	0.28	0.64*	0.22	-0.71*

Effect size comparisons between conditions at each fatigue protocol period. Asterisks (*) denote a $\geq 75\%$ likelihood to demonstrate a meaningful difference

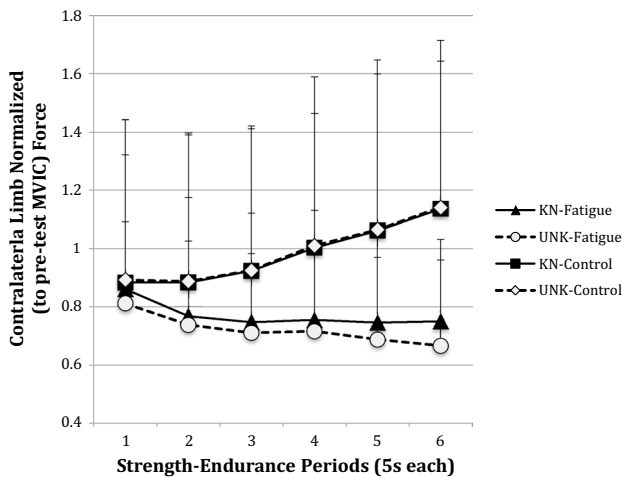


Fig. 4 Contralateral limb normalized force over six 5-s periods of the strength–endurance post-test. Check Tables 3 and 4 for respective effect size and meaningful differences between and within conditions

consistently demonstrated lower force and muscle activity (12% lower overall, 41.1% at period 6) than KNtest > control. A representative tracing of this effect is illustrated in Fig. 5.

Consistent with previous research (Halperin et al. 2014a, b; Kawamoto et al. 2014), the longer duration strength–endurance test demonstrated significant NLMF effects vs. a single MVIC. At various points throughout the strength–endurance test of the contralateral, non-exercised limb, both fatigue conditions produced less force and muscle activity than the control conditions. In contrast, the single MVIC following the fatigue intervention produced a potentiation of force (UNKtest > fatigue) and muscle activity (UNK- and KNtest > fatigue). When participants knew they only had a 5-s MVIC, they were able to maintain or even increase their effort in response to contralateral fatigue. When participants were expecting to perform a longer duration test (30 s for KN, or unknown duration for UNK), reductions in muscle activation were more evident. This is the first study to demonstrate non-local muscle potentiation effects.

Table 3 Contralateral limb between conditions × time MVIC force interaction: effect sizes comparisons between conditions at each fatigue protocol period

	KN-fatigue vs. UNK-fatigue	KN-fatigue vs. KN-control	KN-Fatigue vs. UNK-control	UNK-fatigue vs. KN-control	UNK-fatigue vs. UNK-control	KN-control vs. UNK-control
Period 1: 0–5 s	–0.21	0	–0.04	0.29	0.25	–0.04
Period 2: 5–10 s	–0.18	0.18	0.14	0.42*	0.4*	–0.04
Period 3: 10–15 s	–0.23	0.22	0.16	0.54*	0.44*	–0.06
Period 4: 15–20 s	–0.19	0.13	0	0.45*	0.26	–0.13
Period 5: 20–25 s	–0.33*	0.18	–0.05	0.67*	0.34	–0.25
Period 6: 25–30 s	–0.4*	0.17	–0.07	0.86*	0.29	–0.41*

Asterisks (*) and bolded numbers denote a ≥75% likelihood to demonstrate a meaningful difference. Negative signs indicate the second variable decreased in comparison to the first variable. See Fig. 3 for normalized means and standard deviations

Table 4 Contralateral limb within conditions × time MVIC force interaction: percentage chance that the mean force in the identified period was meaningfully (>75% likely) worse than the first period (0–5 s)

	Period 2: 5–10 s	Period 3: 10–15 s	Period 4: 15–20 s	Period 5: 20–25 s	Period 6: 25–30 s
KN-fatigue	98	100	99	100	99
UNK-fatigue	95	99	98	100	100
KN-control	51#	76	85	80	70#
UNK-control	35#	73#	98	99	100

Number signs (#) indicate where differences were NOT meaningful (<75% likely). See Fig. 3 for normalized means and standard deviations. Asterisks (*) and bolded numbers denote a ≥75% likelihood to demonstrate a meaningful difference

Post-activation potentiation (PAP) is commonly induced in the affected muscle following a non-fatiguing conditioning activity (e.g., 5–10-s MVICs or 3–5 high-intensity squat actions) (Behm et al. 2004; Sale 2002). In contrast, in the present study, potentiation was induced in a non-active muscle following fatiguing activity in the contralateral limb. PAP increases muscle force through muscular (i.e., myosin phosphorylation) and neural mechanisms (Behm 2004; Sale 2002). Since the muscle PAP occurred in a non-active muscle, muscle phosphorylation was very unlikely to have played a role. Rattey et al. (2006) showed that crossover fatigue in their study was not related to peripheral responses such as excitation–contraction coupling since there were no alterations in M-waves or resting twitch forces in the contralateral muscle. Hence, the force and activation potentiation must have been induced by neural mechanisms including the possibility of increased motor unit recruitment, rate coding, and synchronization (Behm et al. 2004; Behm 2004; Sale 2002). Specifically, other NLMF studies have demonstrated increased spinal (Aboodarda et al. 2015a; Šambaher et al. 2016) and cortical (Aboodarda et al. 2015b) excitability with the

non-exercised muscle following the contralateral exercise intervention.

The lack of a known task endpoint (UNKtest > fatigue) produced lower forces (periods 5–6) and muscle activity (VM: periods 4–6; RF and VL: period 6). It is likely that the known endpoint conditions provided higher motivation, which has been shown to enhance self-control and help overcome performance impairments due to fatigue (Hagger et al. 2010). A prevailing view of self-control is that it is a finite resource like strength or energy, and becomes less effective when depleted (Baumeister 2002; Hagger et al. 2010). This ‘strength model’ of self-control places great emphasis on prior task performance and fatigue on our ability to exercise self-control (Hagger et al. 2010). For example, Paterson and Marino (2004) illustrated that deceiving cyclists as to the distance cycled (actual distances: 24–36 km vs. perceived distance: 30 km) in a time trial could either increase or decrease the subsequent cycling time in a 30-km time trial. Although ethical issues would need to be addressed, deception of the individual could be used to increase force, power, or other performance parameters when performing an activity with a predictable endpoint (closed loop). The effects of fatigue and motivation on self-control and task performance are largely suggested to be interactive (Muraven and Baumeister 2000), which appears to be the case in the present study.

Participants’ pacing strategies through the strength–endurance test were comparable to the Halperin et al. studies (2014a, c). Halperin et al. (2014a) noted a more marked force decrease during the first 6 MVICs (13%) across all conditions, and a plateau in force over the last 6 MVICs (3% decrease). Both fatigue conditions in the present study reduced force output into period 2 before plateauing. The control conditions displayed a more gradual loss of force output into periods 3 or 4 (UNKtest > control) before plateauing, and even rebounding in the case of KNtest > control. Muscle activity remained fairly stable throughout the strength–endurance test, with most

Table 5 Contralateral limb between conditions \times time RMS EMG interaction: effect size comparisons between conditions at each period for muscle groups of the contralateral (non-exercised) leg during the strength–endurance test

	KN-fatigue vs. UNK-fatigue	KN-fatigue vs. KN-control	KN-fatigue vs. UNK-control	UNK-fatigue vs. KN-control	UNK-fatigue vs. UNK-control	KN-control vs. UNK- control
Period 1: 0–5 s	VM -0.23 RF 0.1 VL 0.1 *BF -0.47	VM 0.04 RF 0.04 VL 0.38 BF -0.41	VM 0.37 RF 0.3 VL 0.04 BF -0.13	VM 0.29 RF -0.11 VL 0.42 BF 0.02	*VM 0.52 RF 0.18 VL 0.08 *BF 1.02	*VM 0.53 RF 0.21 VL -0.29 BF 0.2
Period 2: 5–10 s	VM -0.28 RF 0.17 VL 0.13 *BF -0.53	*VM 0.5 RF 0.35 *VL 0.78 BF -0.2	*VM 0.94 *RF 0.64 *VL 0.52 BF 0.08	*VM 0.5 RF 0.17 *VL 0.62 BF 0.36	*VM 0.76 RF 0.38 *VL 0.38 *BF 0.87	VM 0.22 RF 0.2 VL -0.16 BF 0.33
Period 3: 10–15 s	VM -0.3 RF 0.02 VL -0.02 *BF -0.44	*VM 0.47 RF 0.22 *VL 0.77 BF -0.07	*VM 0.86 RF 0.45 VL 0.45 BF 0.16	*VM 0.57 RF 0.25 *VL 0.8 BF 0.33	*VM 0.72 RF 0.26 VL 0.41 *BF 0.59	VM 0.33 RF 0.17 VL -0.22 BF 0.25
Period 4: 15–20 s	*VM -0.45 RF -0.12 VL 0.05 *BF -0.41	VM 0.2 RF -0.01 *VL 0.75 BF -0.14	*VM 0.54 RF 0.27 VL 0.23 BF -0.01	*VM 0.78 RF 0.24 *VL 0.73 BF 0.42	*VM 0.68 RF 0.28 VL 0.2 *BF 0.57	VM 0.17 RF 0.18 VL -0.26 BF 0.15
Period 5: 20–25 s	*VM -0.59 RF -0.17 VL -0.32 *BF -0.53	*VM 0.63 RF 0.14 *VL 0.8 BF -0.05	VM 0.36 RF 0.14 VL 0.13 BF 0.1	*VM 0.95 RF 0.32 *VL 1.08 *BF 0.61	*VM 0.6 RF 0.1 VL 0.31 *BF 0.73	VM -0.02 RF 0 *VL -0.41 BF 0.14
Period 6: 25–30 s	*VM -0.62 *RF -0.39 *VL -0.47 *BF -0.65	*VM 0.53 RF -0.03 *VL 0.84 BF -0.08	VM -0.24 RF -0.31 *VL -0.5 BF -0.29	*VM 1.34 *RF 0.6 *VL 1.4 *BF 0.94	*VM 0.43 RF 0.07 VL 0.1 *BF 0.56	*VM -0.47 RF -0.25 *VL -0.94 BF -0.24

Asterisks (*) denote a $\geq 75\%$ likelihood to demonstrate a meaningful difference for that muscle. Bold cells illustrate a $\geq 75\%$ likelihood of a meaningful difference in force (comparison with meaningful force differences detailed and shaded in Table 1a). Negative signs indicate the second variable decreased in comparison to the first variable

significant differences occurring when comparing period 1 and 6 for the known endpoint conditions (VM and VL). Thus, based on the present study as well as other studies, the knowledge or lack of knowledge of a task endpoint would have the most substantial effects upon the initial and final segments of a task (Billaut et al. 2011; Halperin et al. 2014c, d; St. Clair Gibson and; Noakes 2004).

Given the maximal and fairly brief (~30 s) nature of the strength–endurance test, it can be argued there was limited opportunity for centrally mediated pacing strategies to be employed (Shephard 2009; Weir et al. 2006). Peripheral feedback has been noted in previous research to be the key mediator of performance impairments at maximal intensities or shorter durations (Amann et al. 2013; Shephard 2009; Weir et al. 2006). However, our findings of test duration and known vs. unknown endpoint effects demonstrate that psychological or perceptual factors can also provide an impact at this intensity and duration. Similar to our study, Halperin et al. (2014a, c) examined pacing while manipulating the participants' prior knowledge of test endpoint. They found MVIC forces in trained females (Halperin et al. 2014a) and a recreationally active group of both sexes (Halperin et al. 2014c) were significantly higher

in a deception condition (told to perform six repetitions but then did 12) during the first 6 MVICs, compared to known (12 repetitions) and unknown conditions. Both studies found no differences between conditions over the last 6 MVICs of the 12 MVIC protocol. Their findings, consistent with those of the present study, reveal that even when every effort is intended to be maximal, higher forces can be produced when an individual is aware of a more immediate and known endpoint.

The similar endurance profiles between KNtest > fatigue and control groups for the strength–endurance test is in agreement with Kawamoto et al. (2014), who found no difference in time to task failure between control and contralateral pre-fatigue conditions (40 and 70% MVIC fatigue protocols used). They followed a similar isometric knee extension endurance test, but task failure was set at 70% pre-test MVIC as opposed to 60% in our study. Although, Amann et al. (2013) noted a decrease in endurance time following a contralateral knee extensor fatigue protocol, they used a longer duration cycling test (5–10 min) for their endurance protocol. In contrast to UNKtest > fatigue, the knowledge of test endpoint (KNtest > fatigue) allowed the individual to offset the NLMF influences.

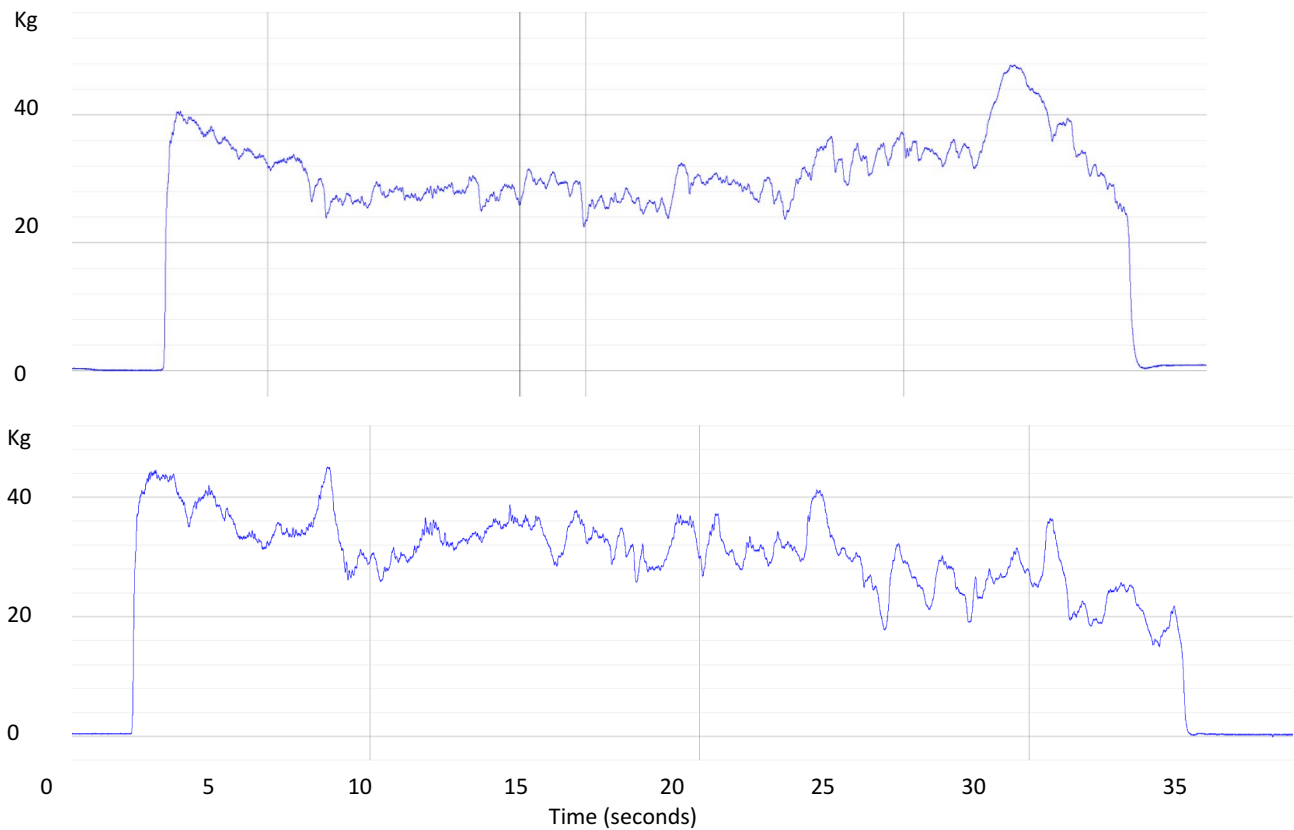


Fig. 5 Representative tracings of a **a** known test endpoint (KNtest > fatigue) and an **b** unknown test endpoint strength–endurance test (UNK-test > fatigue) following contralateral fatigue

When examining the influence of different muscle groups, the VM and VL contributed most notably to the aforementioned force changes. Both the VM and VL displayed significant changes that regularly paralleled and in some cases preceded significant changes in force. The greater sensitivity to change observed in muscle activity compared to force suggests that changes in central drive and excitability mitigated other performance impairments. Research has demonstrated that central excitability changes occur in response to developing fatigue, even in the absence of visible performance deficits such as loss of force (Aboodarda et al. 2015a, b; Behm 2004).

While the known vs. unknown endpoints were designed to investigate pacing strategies, it is difficult to specify what physiological mechanisms were predominant. Aboodarda et al. (2015a) did not observe any change in MVIC force following bilateral elbow flexor fatigue, but normalized VL EMG did decrease significantly. Using thoracic motor evoked potentials (TMEPs) and maximal compound muscle action potentials (Mmax), they concluded that supraspinal motor output had decreased given that spinal motoneuronal responses (TMEP/Mmax) were higher and peripheral excitability (compound muscle action potential) did not change.

In another study, Aboodarda et al. (2015b) again did not find changes in force or EMG of contralateral elbow flexors in response to a unilateral elbow flexion protocol. Their analysis of motor evoked potentials (MEPs) and cervicomedullary motor evoked potentials (CMEPs) indicated an increase in supraspinal responsiveness (higher MEP/CMEP ratio) might have mitigated performance impairments. Although there was evidence for NLMF of the elbow flexors following knee extensor fatigue, Šambaher et al. (2016) reported an enhanced spinal excitability (CMEP) countered by reduced supraspinal excitability (lower MEP/CMEP ratio) of the biceps brachii. Hence, based on the limited conflicting studies in this area, the specific central nervous system changes cannot yet be identified.

Peripheral fatigue and its accompanying afferent feedback can modify central neural excitability affecting the previously non-exercised limb through various mechanisms (Amann et al. 2013). Inhibition by Renshaw cells, Golgi tendon organs, and type III and IV afferents, or decreased excitation of Ia afferents have all been shown to stimulate changes in central excitability and performance (Behm 2004). Peripheral feedback mechanisms are likely working in conjunction with psychological influences of mental

fatigue and prior knowledge of endpoint to modulate performance in this study.

Conclusion

In accordance with previous research in this area (Doix et al. 2013; Halperin et al. 2015), this study demonstrated that a high-intensity and high-volume fatigue protocol could produce NLMF effects. Additionally, the longer duration strength–endurance test provided clearer indications of performance decrements than a single MVIC. In fact, the single MVIC demonstrated potentiation following contralateral fatigue. Most importantly, the present study revealed that prior knowledge of test endpoint could modify NLMF expression.

Acknowledgements We wish to thank Dr. Thamir Alkanani for his technical assistance as well as acknowledge and thank the participants for their time and patience. This study was partially funded by the Natural Science and Engineering Research Council of Canada (Grant No. 238792-2012).

Compliance with ethical standards

Conflict of interest This study was conducted without any funding from companies or manufacturers or outside organizations. There are no conflicts of interest.

Ethical approval All procedures performed in the study involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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