European Journal of Applied Physiology (2019) 119:2065–2073
https://doi.org/10.1007/s00421-019-04195-6

ORIGINAL ARTICLE

# Experimental knee pain impairs joint torque and rate of force development in isometric and isokinetic muscle activation

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Received: 19 March 2019 / Accepted: 16 July 2019 / Published online: 23 July 2019


## Abstract

**Purpose** To investigate the effects of acute experimental knee joint pain on maximum force generation and rate of force development (RFD) of the quadriceps muscle during isometric and dynamic muscle activations.

**Methods** The right knee of 20 healthy people was injected with hypertonic saline to create an acute pain experience. Measurements of maximum knee extensor torque during isometric, concentric, and eccentric contractions were undertaken using a Biodex dynamometer. The RFD was also examined during the isometric contractions. Quadriceps muscle activity was obtained using electromyography (EMG). The outcome measures were obtained at baseline, during pain, and after knee pain had resolved.

**Results** Maximum joint torque and peak EMG were significantly reduced during pain, but there were no differences across the three types of contraction. The maximum RFD and rate of EMG rise were also reduced during pain, primarily at 50–100 ms post-contraction onset. The RFD and EMG rise were largely unaffected at later time periods following contraction onset (150–200 ms).

**Conclusions** Acute joint pain has a similar impact on isometric and isokinetic contractions despite differences in neural control strategies. Joint pain also impairs rapid muscle activation and the RFD. These findings are important for people with musculoskeletal pain as it likely contributes to impairments in joint function in these populations.

**Keywords** Acute pain · Knee extensors · Maximum voluntary contraction · Rate of force development

## Abbreviations

| | |
|---|---|
| ANOVA | Analysis of variance |
| EMG | Electromyography |
| MVC | Maximum voluntary contraction |
| RFD | Rate of force development |

## Introduction

There is extensive literature demonstrating that pain reduces the strength of muscle contraction (for reviews see Graven-Nielsen and Arendt-Nielsen 2009, and Hodges and Tucker 2011). This is evident in both chronic pain conditions and in experimental pain models, where the influence of pain can be examined more specifically without the confounding effects of behavioral adaptations to chronic conditions or changes in the integrity of the musculoskeletal system. There can be marked clinical implications of reduced muscle strength, particularly in the lower limb. At the knee, the joint most commonly affected by osteoarthritis, quadriceps weakness has been associated with impaired stability (Felson et al. 2007), reduced function (Liikavainio et al. 2008), and falls (Moreland et al. 2003).

The majority of studies have investigated the effects of chronic and experimental pain during maximal concentric and isometric muscle activation, despite the fact that there are notable differences in force generation capacity, fatigue properties, and neural control of eccentric

Communicated by Toshio Moritani.

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muscle activations (Enoka 1996). At the knee specifically, eccentric activation is critical for weight acceptance while walking downstairs or downhill, transitioning from standing to sitting, or landing from jumps or falls. The few studies that have investigated the effect of pain on eccentric muscle activation have reported findings that challenge the notion of generalized impairment in muscle strength with pain. Three studies have reported a relative loss of concentric compared to eccentric strength in trunk muscles of people with low back pain (Dvir and Keating 2003; Marshall et al. 2010; Shirado et al. 1995). No studies have examined the impact of experimental pain on the relative strength of eccentric, concentric and isometric muscle activations. This would enable any differences in the effect of pain on the different types of activation and a clearer picture of how pain may influence function to be determined.

An additional relevant characteristic of muscle contraction is the rate of force development (RFD). In comparison to maximal strength, the RFD is more strongly related to function (Maffiuletti et al. 2010; Tillin et al. 2013) and is more sensitive to changes in the integrity of the neuromuscular system (Angelozzi et al. 2012; Crameri et al. 2007; Jenkins et al. 2014; Penailillo et al. 2015). Maximal RFD is dependent on muscle size and strength as well as neural drive, with the early component (0–75 ms) primarily determined by neural drive to the muscle and the later component more dependent on structural contractile properties of the muscle (Andersen and Aagaard 2006; Andersen et al. 2010; Folland et al. 2014). Several studies have shown that maximal RFD is reduced in chronic musculoskeletal pain conditions (Andersen et al. 2008; Chourasia et al. 2012; Gapeyeva et al. 2007; Vahtrik et al. 2012; Winters et al. 2014). Given the aforementioned issues when chronic pain is present, it would be more ideal to examine the effects of pain on the RFD in a controlled acute pain paradigm to enable a clearer delineation of the effect of pain on the ability to rapidly develop muscle force.

The aim of this study was to investigate the effects of acute experimental knee joint pain on maximum force generation of the quadriceps muscle during isometric and dynamic muscle activations. We hypothesized that acute pain would differentially impact the maximum force developed during the different types of muscle activation. A further goal was to determine the effects of acute pain on the RFD during isometric muscle activation. We hypothesized that acute pain would impair the early component of RFD more than the later component and that pain-induced changes in early RFD would be related to impaired muscle activation, whereas the pain-induced changes in the later component of RFD would be more strongly related to changes in peak torque.

## Methods

### Participants

Twenty healthy people (11 female, age 18–49 years) participated in the study. Participants were excluded if they reported a history of significant trauma to the lower limbs and/or trunk with any current musculoskeletal or neural symptoms, any diagnosed neurological or spinal disorders, use of pain medications on the day or day prior to testing, pain > 2/10 anywhere in the body on the day of testing, or a score > 30 on the Pain Catastrophizing Scale (Osman et al. 2000; Sullivan et al. 2009). All participants gave written informed consent and the study received ethical approval from the local institution.

### Isokinetic dynamometer

Knee extensor torque was measured using isokinetic dynamometry (Biodex system 3, Biodex Medical Systems, New York). Participants were positioned in the dynamometer with adjustable straps placed around their shoulders, waist, and right thigh to restrain movement. The center of the right knee joint was aligned with the axis of rotation of the dynamometer and the shank restrained by a resistance cuff. For isometric contractions, the knee joint was set to 65° (0° = full extension). For concentric and eccentric contractions, the velocity was 30°/s and the range of movement was from 0 to 100° (Henriksen et al. 2011). To enable the effects of gravity to be corrected, prior to testing the weight of the limb was obtained by asking participants to relax their leg with the limb held stationary. Joint position and torque data from the dynamometer were sampled at 2 kHz (LabChart v7.2.1, AD Instruments Pty Ltd, NSW).

### Electromyography

Muscle activity from the vastus lateralis (VL), vastus medialis (VM), and rectus femoris (RF) muscles was recorded using surface electromyography (EMG). Standard skin preparation was implemented prior to positioning the electrodes. Electrodes were placed according to SENIAM guidelines, with a ground positioned over the anterior tibia. EMG data were amplified (AMT-8, Bortec Biomedical, Canada), filtered (10–1000 Hz), and sampled at 2 kHz (LabChart v7.2.1, AD Instruments Pty Ltd, NSW).

### Experimental pain

With the participant positioned in the dynamometer, an injection of 1.0 ml of 5.8% hypertonic saline was

administered using a 27-G needle. The injection was given into the anterior medial aspect of the right infrapatellar fat pad at a 45° angle, with a needle insertion depth of 1 cm. A 0–10 numerical rating scale was used to rate pain intensity at 30 s following needle withdrawal, and then every 90 s until pain resolved (approximately 15 min).

### Procedure

Participants attended a practice session that involved familiarization with the equipment and tasks. At least 1 week later, they attended a data collection session. Maximum voluntary contractions (MVCs) were recorded during isometric, eccentric and concentric muscle activation. For each type of muscle activation, five MVCs were performed. Participants were instructed to perform all MVCs as "hard and fast as possible" and were verbally encouraged throughout. Each MVC was sustained for 3 s, with a 3–5-s rest between each of the 5 MVCs. After the 5 MVCs for one type of muscle activation were completed, a 30-s rest was given before completing MVCs in the next type of muscle activation. To control for order effects, the order of muscle activation types was randomized among participants using a counterbalanced design.

MVCs were collected at three time points: baseline, during experimental knee pain (pain), and after the experimental knee pain had resolved (post-pain). A 5-min rest was given after completion of the baseline MVCs before experimental knee pain was induced. The MVCs during knee pain were initiated once a numerical pain rating of $\geq$ 3/10 was reached. When the numerical pain rating returned to 0, participants waited a further 2 min before completing the post-pain MVCs.

### Data processing

Peak torque was determined as the maximum torque value during any of the 5 MVCs in each type of muscle activation (isometric, concentric, eccentric). RFD was analyzed in the isometric muscle activations only. RFD data were obtained from the trial that showed the highest peak slope of the torque–time curve and was determined as the average slope of the curve over time intervals of 0–50, 50–100, 100–150, and 150–200 ms (Tillin et al. 2010), relative to the onset of contraction. The onset of contraction was defined as the time point at which torque exceeded 7.5 Nm (Aagaard et al. 2002).

EMG data were filtered using a moving root mean square (rms) filter with a time constant of 50 ms (Aagaard et al. 2002). For all types of muscle activation, the peak EMG amplitude was determined from the trial in which peak torque was identified. For the isometric muscle activation trials, the rate of EMG rise was analyzed in the trial that was used to obtain RFD data. Integrated EMG was determined in time intervals of 0–50, 50–100, 100–150, and 150–200 ms relative to onset of EMG, with the onset of EMG defined as 70 ms prior to the onset of contraction (Aagaard et al. 2002). For all EMG analyses, data from the three muscles were averaged to provide a single measure of quadriceps muscle activation (Ruiter et al. 2004).

For all torque and EMG outcome measures, change scores were also determined to measure the impact of pain. Change scores were determined by subtracting the baseline measure from the measure obtained during pain, which was then expressed as a percentage of the baseline value.

### Statistical analysis

To compare the effect of pain on maximum force generation across the three forms muscle activation, peak torque and EMG peak amplitude were analyzed using a two-way repeated measures ANOVA with factors of muscle activation type (isometric, concentric, eccentric) and time (baseline, pain, post-pain). To investigate the effect of pain on RFD, torque slope and integrated EMG from the isometric trials were analyzed using a one-way ANOVA with the factor of time (baseline, pain, post-pain). Separate ANOVAs were completed for each time interval (0–50, 50–100, 100–150, 150–200 ms). A Huynh–Feldt correction was used to adjust $P$ values when Epsilon $< 1$. Significant main effects were further investigated using paired $T$ tests.

All statistical analyses were performed using SPSS (v22, IBM Corp., Armonk, NY). Statistical significance was set at $P < 0.05$. Data are presented as mean$\pm$standard deviation unless otherwise indicated.

## Results

### Experimental pain duration

One participant did not complete data collection due to an adverse reaction to the injection of hypertonic saline. Pain ratings following injection of hypertonic saline were not recorded in one participant. The average time from injection of hypertonic saline until the numerical pain rating returned to 0 was $18 \pm 4$ min. The average peak pain rating was $5.8 \pm 1.9$ out of 10.

### Maximum force generation: peak torque

Figure 1a shows peak torque values during the three types of muscle activation. There was a main effect of activation type ($F_{2,36} = 77.7$; $P < 0.001$) and time ($F_{2,36} = 5.9$; $P = 0.008$), but the interaction was not significant ($F_{4,72} = 0.5$; $P = 0.8$). Further analysis indicated that peak torque was significantly

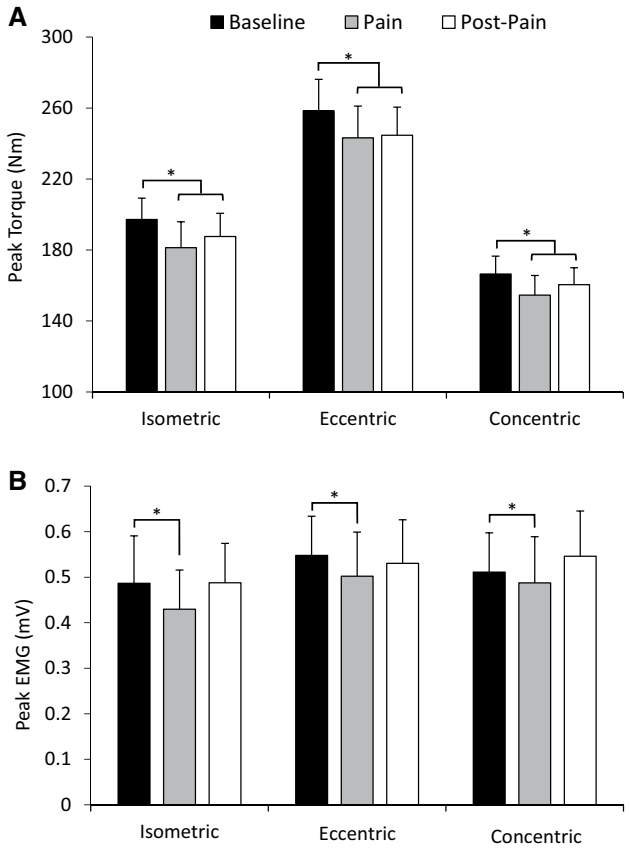


**Fig. 1** Group results showing mean peak torque (**a**) and peak EMG (**b**) before, during, and after pain for the three muscle activation types. Bars are 1 standard error

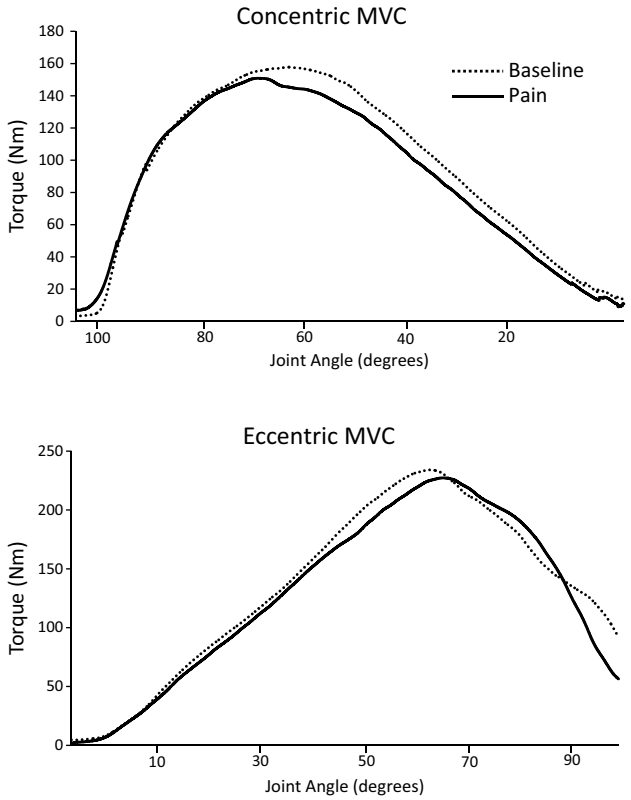


**Fig. 2** Average torque profiles during eccentric and concentric maximum voluntary contractions (MVCs). Data are shown for baseline and during pain

greater at baseline than during pain ($P = 0.01$) and at the post-pain period ($P = 0.008$). The difference between pain and post-pain was not significant ($P = 0.3$). Across all participants, peak torque during pain decreased by 9.7 ± 16.9, 5.8 ± 15.9, and 7.4 ± 15.4% during isometric, eccentric, and concentric muscle activation, respectively. Figure 2 shows the average torque profiles at baseline and during pain for the CON and ECC trials. In both ECC and CON contractions, torque is primarily inhibited in the midrange from 40 to 65°, which encompasses the point of peak torque.

As expected, analysis of the main effect of activation type revealed that peak torque was significantly greater during eccentric activation compared to concentric ($P < 0.001$) and isometric ($P < 0.001$) activation, and was greater during isometric compared to concentric activation ($P < 0.001$).

## Maximum force generation: peak EMG

Figure 1b shows peak EMG data during the three types of muscle activation. Analysis of peak EMG revealed a main effect of time ($F_{2,36} = 4.1$; $P = 0.03$) but not activation type ($F_{2,36} = 1.1$; $P = 0.3$). The interaction also was not significant ($F_{4,72} = 1.0$; $P = 0.4$). Peak EMG was reduced during pain compared to baseline ($P = 0.01$), but was not different at post-pain compared to baseline ($P = 0.3$). In the isometric condition, the change in peak torque during pain was strongly correlated with the change in EMG peak during pain (Pearson $r = 0.75$; $P < 0.001$).

## RFD: torque

Figure 3a shows the group torque slope data over the four time periods analyzed, while Fig. 3b shows the average baseline and pain torque profile across participants. At baseline, there was a strong positive correlation between peak torque and peak torque slope (Pearson's $r = 0.79$; $P < 0.001$).

The main effect of time was significant for peak torque slope ($F_{2,36} = 5.0$; $P = 0.02$) and the slope measured over 50–100 ms ($F_{2,36} = 6.8$; $P = 0.003$). For both of these variables, the torque slope during pain was less than baseline (both $P < 0.005$). While peak torque slope increased post-pain and was no different to baseline ($P = 0.6$), torque slope over 50–100 ms was still reduced from baseline at post-pain ($P = 0.03$). The main effect of time was not significant for torque slope over 0–50 ms ($F_{2,36} = 2.5$; $P = 0.1$), 100–150 ms ($F_{2,36} = 0.8$; $P = 0.4$), or 150–200 ms ($F_{2,36} = 2.7$; $P = 0.08$).

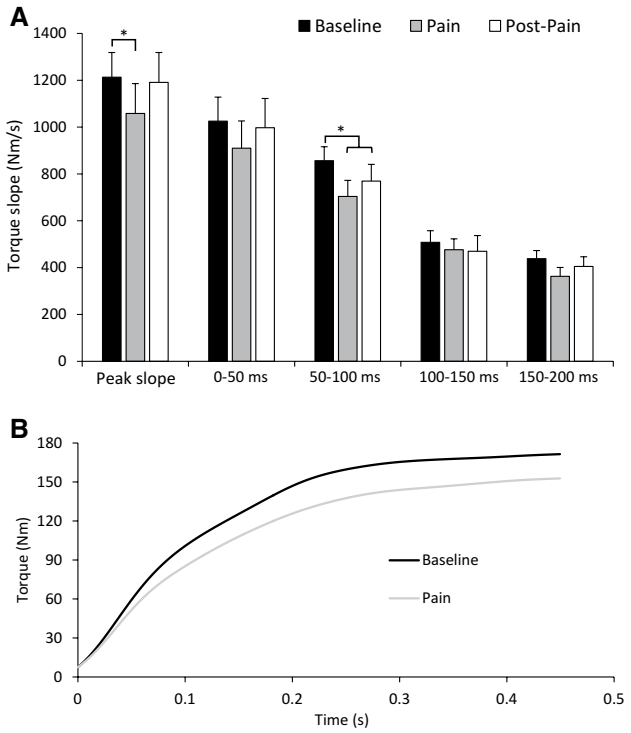


**Fig. 3** **a** Rate of force development group results showing the torque slope before, during, and after pain for isometric contractions. Data for the peak slope are shown as well as the four discrete time periods. *$P < 0.05$. Bars are 1 standard error. **b** Group averaged torque profile showing the initial 450 ms following contraction onset at baseline and during pain

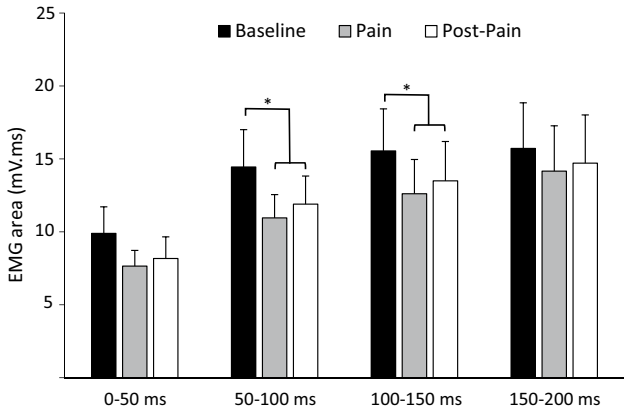


**Fig. 4** Group results showing the EMG rise data before, during, and after pain. *$P < 0.05$. Bars are 1 standard error

To test the hypothesis that the change in peak torque with pain would be more strongly related to the change in the later components of RFD, we correlated the change scores between these variables. There were moderate-strong correlations between the change in peak torque during pain and the change in torque slope across all time periods, with the strongest relationship evident in the 150–200-ms time period (0–50 ms: $r = 0.62$, $P = 0.005$; 50–100 ms: $r = 0.55$, $P = 0.014$; 100–150 ms: $r = 0.57$, $P = 0.013$; 150–200 ms: $r = 0.70$; $P = 0.001$).

### RFD: rate of EMG rise

The rate of EMG rise was measured using EMG area. Figure 4 shows the group results over the four time periods analyzed, while Fig. 5 shows the average EMG profiles for the three muscles. There was a main effect of time in the time periods of 50–100 ms ($F_{2,36} = 5.8$; $P = 0.009$) and 100–150 ms ($F_{2,36} = 5.0$; $P = 0.01$). At both of these time periods, EMG area was significantly lower than baseline during pain (both $P < 0.015$) and at post-pain (both $P < 0.05$). The main effect of time was not significant for EMG area over 0–50 ms ($F_{2,36} = 3.3$; $P = 0.053$) or 150–200 ms ($F_{2,36} = 1.3$; $P = 0.3$).

To test the hypothesis that the early component of RFD would be more affected by changes in EMG during pain than the later component, we correlated the change in EMG area at each time period with the changes in torque slope over the equivalent time period. The correlation between these variables was significant at all time periods (all $P < 0.03$) but was stronger at 0–50 ms ($r = 0.71$), 50–100 ms ($r = 0.67$), and 100–150 ms ($r = 0.67$) than the final time period of 150–200 ms ($r = 0.52$).

## Discussion

The first aim of this study was to investigate the effect of experimental joint pain on maximal knee extension torque during isometric and isokinetic activation, and to determine the impact on maximal RFD. We found that peak torque and EMG were equally impaired during isometric, concentric, and eccentric muscle activation in the presence of joint pain. We also provide novel evidence that the RFD is reduced in the presence of joint pain, and that this is accompanied by a reduction in the rate of EMG rise. Notably, the impact of pain on the RFD appeared to differ across the time periods examined. Thus, our findings indicate that not only does knee joint pain reduce the ability to maximally activate the quadriceps muscle, it also impairs the maximal rate of activation.

The reduction in isometric and concentric MVC was a similar magnitude to comparable studies of acute experimental pain on knee extensor torque (Henriksen et al. 2011; Salomoni et al. 2016), although it was modest in comparison to others (Asaki et al. 2018; Graven-Nielsen et al. 2002; Park and Hopkins 2013). Supporting Salomoni et al. (2016), the effects of pain were quite variable among participants,

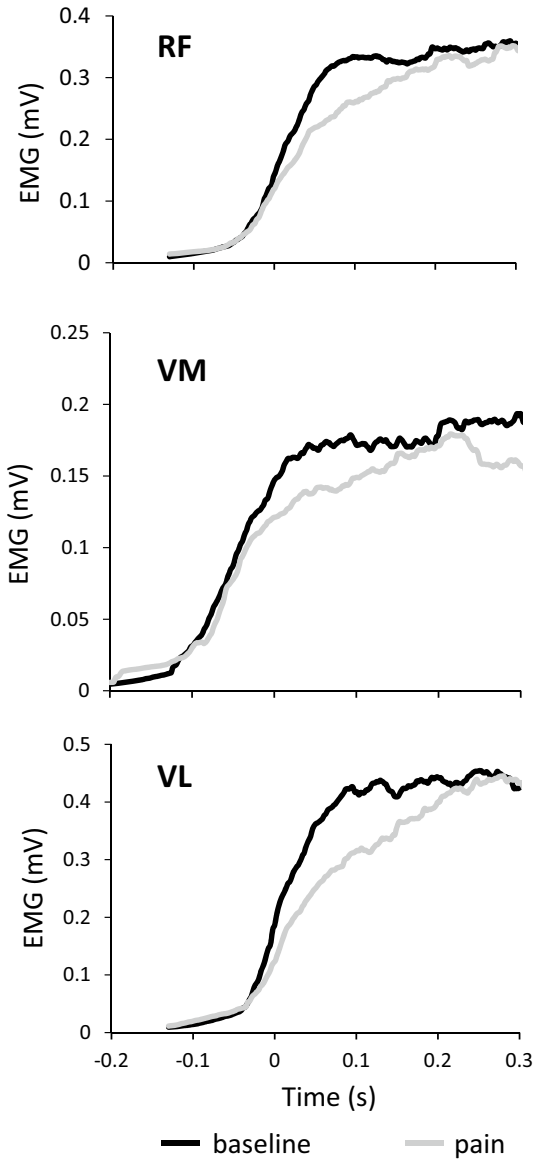


**Fig. 5** Group averaged profiles of the initial 300 ms of EMG activity for the quadriceps muscles at baseline and during pain. *RF* rectus femoris, *VM* vastus medialis, *VL* vastus lateralis

with only 10 participants (50%) showing greater than 5% reduction in peak torque in the isometric condition. Despite previous research indicating an altered eccentric/concentric strength ratio in people with chronic pain (Dvir and Keating 2003; Marshall et al. 2010; Shirado et al. 1995), we found no evidence that eccentric muscle activation is impacted any differently by acute pain. Compared to concentric activation, eccentric muscle actions are served by different neural control strategies (Enoka 1996; McHugh et al. 2002), have greater resistance to fatigue (Enoka 1996), and are capable of generating more force (Lindstedt et al. 2001). Thus, there is certainly potential for nociceptive input to have a differing effect on eccentric actions. It is possible that the effect of acute pain on eccentric and concentric actions is different from that of chronic pain, where fear of movement (Karayannis et al. 2013; Nederhand et al. 2006) or altered patterns of activation (Arendt-Nielsen et al. 1996; Hodges and Richardson 1996) may be influential.

The exact mechanisms of joint torque inhibition during pain are unknown; however, it is widely postulated that it is due to central adaptations (Henriksen et al. 2009, 2011; Salomoni et al. 2016; Slater et al. 2003). Supporting this view, the strong correlation between changes in peak EMG and peak torque suggest the impaired force was related to reduced neural drive. Based on findings showing a relationship between voluntary activation and force impairment, Salomoni et al. (2016) speculated that this was at least in part due to impaired descending voluntary drive. In the upper limb, studies using transcranial magnetic stimulation have provided good evidence that acute pain is associated with reduced corticomotor excitability (Burns et al. 2016), but there is evidence for the opposite in the quadriceps muscle (Rice et al. 2015). Thus, impaired activation of the quadriceps cannot be solely explained by changes in corticomotor excitability.

The maximum EMG recovered to baseline levels following the cessation of pain but the maximum torque did not. Other studies have also shown a sustained impairment in maximum isometric or concentric peak torque following the resolution of acute pain (Ervilha et al. 2004; Henriksen et al. 2007, 2009, 2011; Salomoni et al. 2016; Shakespeare et al. 1985; Slater et al. 2003) and ongoing suppression of corticomotor excitability (Burns et al. 2016). Together, these findings suggest that the impact of nociception on motor output persists beyond the period of subjective pain experience.

This is the first study to show that the rate of force development and EMG rise is impaired in the presence of acute experimental pain. Aagaard and colleagues (2002) were among the first to delineate different temporal components of RFD and speculate different contributing factors. Subsequent cross-sectional studies have used various techniques (e.g., twitch force comparisons, muscle or fiber types, comparison to clinical populations) to more clearly identify the limiting factors across the early and late components (reviewed in Maffiuletti et al. 2016). There is now good evidence that maximal motor unit discharge rate is critical for initial force development (< 75 ms) (Del Vecchio et al. 2019), while the later component is more strongly influenced by the contractile properties of muscle and the peak MVC force itself. It is known that acute nociceptive activation can reduce the firing rate of motor units recruited during submaximal tasks (Farina et al. 2004, 2005, 2008; Sohn et al. 2000; Tucker et al. 2009). There is also evidence that muscle contractile properties are not impacted by experimentally induced acute muscle pain (Farina et al. 2004; Graven-Nielsen et al. 2002). Therefore, it was hypothesized

that acute pain would preferentially impair the earlier components of the RFD, and that this would be associated with impaired EMG rise. Our hypotheses are somewhat supported by the finding that peak torque slope and slope from 50 to 100 ms were the only periods to be significantly inhibited during pain. EMG area was also significantly inhibited from 50 to 100 and 100–150 ms, with a trend for inhibition over 0–50 ms ($P = 0.053$). The strength of the correlations between the changes in torque slope and EMG area during pain also decreased from the early to late components, while the relationship between change in torque with pain and change in RFD was strongest for the late component.

Two studies have used a delayed onset muscle soreness (DOMS) model to induce quadriceps muscle pain and examine the impact on RFD (Molina and Denadai 2012; Vila-Chã et al. 2012). In the presence of DOMS-induced pain, both studies reported a reduction in MVC and extensor torque slope, while Vila-Cha et al. (2012) also reported reduced quadriceps EMG activity. In DOMS, the muscular nociceptive pain has been related to inflammation following tissue damage (MacIntyre et al. 1995). While acknowledging this muscle damage and the potential impact on force development, the reduced EMG activity during ballistic activation indicates there is also neural contribution to impaired RFD. Similar to our findings, Molina and Denadai (2012) reported that the change in peak torque correlated with the change in RFD, at least at 24 h following DOMS inducement. The benefit of our acute experimental pain model was that we manipulated MVC capacity in a single session where the effects of electrode placement, body position, and changes in muscle properties were eliminated. Our study findings, therefore, more clearly show that acute joint pain impairs the maximal RFD, and that it primarily affects the early component.

## Clinical relevance

Given the documented relationship between RFD in the quadriceps and function (Maffiuletti et al. 2010; Tillin et al. 2013), our finding of impaired RFD during acute pain is important and relevant for people with long-term pain conditions, such as osteoarthritis. Indeed, other studies have provided evidence that the RFD is lower in people with knee osteoarthritis (Gapeyeva et al. 2007; Vahtrik et al. 2012; Winters et al. 2014) as well as in chronic upper limb musculoskeletal pain conditions (Andersen et al. 2008; Chourasia et al. 2012). In these clinical populations, the RFD has been correlated with both pain (Andersen et al. 2008; Chourasia et al. 2012) and function (Andersen et al. 2008; Chourasia et al. 2012; Winters et al. 2014) measures. Along with our findings, the correlations with pain evident support an impact of pain on RFD in these populations, although Anderson et al. (2008) also acknowledge psychological effects associated with fear of movement that are likely to be influential in people with long-term pain. The finding that quadriceps RFD remains impaired following total knee joint replacement (Chourasia et al. 2012; Winters et al. 2014) highlights the importance of addressing pain control and targeting rapid torque generation through explosive/power training in clinical populations.

## Limitations

There were several limitations to the current study. First, maintaining the desired pain intensity for a period long enough to complete the tasks involved is difficult with a single injection of hypertonic saline, and three participants reached a pain rating of 0/10 before they had completed all the pain condition measures. We did not include a control (isotonic saline) injection in the study due to the potential risk to the participants. Therefore, the changes in MVC and RFD during pain may have been due to fatigue; however, this is unlikely given the rest periods provided and the return to baseline of some measures in the post-pain period. We also did not record EMG activity from the hamstring muscles and are, therefore, unable to determine any changes in co-activation during pain. However, a reduction in antagonist activation during pain has been shown in other studies (Ciubotariu et al. 2007; Salomoni et al. 2016). Finally, there was a large age range of the participants in the study and there is evidence that speed-related force development properties are reduced with ageing, which may have influenced the variability of our findings (Klass et al. 2008).

**Author contributions** DR, PM, and JM conceived and designed the research. LF and DR collected the data. LF and GL analyzed the data. GL and LF wrote the manuscript. All the authors read and approved the manuscript.

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

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