

# The effect of experimental pain during motor skill training on acquisition and motor cortical maps



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# The effect of experimental pain during motor skill training on acquisition and motor cortical maps

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**ABSTRACT:** The aim of the study was to determine if acute experimental pain in the soleus (SOL) affects the learning of a novel trace-tracking task involving fine control of dorsi flexion of the ankle joint, and the associated motor cortical maps of the tibialis (TA) and SOL, as assessed preand post-training. Twenty-four healthy participants (20 men, 4 women, age 24.79  $\pm$  0.57 years, height 1.81  $\pm$  0.02 m, weight 80.67  $\pm$  2.49 kg, sporting activity per week 6.63  $\pm$  0.58 hours) were semi-randomly divided into a pain and control group. Multiple injections of either hypertonic saline (0.5 mL, 0.6 mL, 0.8 mL, 1 mL, 5.8%) or isotonic saline (0.5 mL, 0.6, mL, 0.8 mL, 1 mL, 0.9%) as a control, was administered to the dominant soleus muscle throughout a 24-minute trace-tracking training period. Before and after the training, transcranial magnetic stimulation (TMS) was applied to the primary motor cortex (MI) to generate motor cortical maps of TA and SOL. Pain and control groups improved equally in motor skill performance, and pain did not interfere with performance improvement. Furthermore, no alterations were found in the motor maps for either TA or SOL for either group.

**PERSPECTIVE:** Since the present study shows that pain does not interfere with motor skill acquisition or alter the motor cortical maps. This can be useful in rehabilitation since patients can acquire new or modified motor skills without the influence of pain which might help them to move in a more beneficial way.

*Key words*: motor cortical mapping, motor skill performance, motor skill acquisition, experimental pain, TMS.

#### **1. INTRODUCTION**

The influence of pain on learning is an essential problem in the area of sports since many athletes experience painful injuries at some point during their training (Hopkins, Marshall, Quarrie, & Hume, 2007). Leg injuries caused by exercise and overuse is reported as a common problem (Burrus et al., 2015; among athletes Rajasekaran, Kvinlaug, & Finnoff, 2012). This is critical since adaptations to pain, such as changes in motor strategy and reduction in movement, can lead to longterm detrimental impairments such as overload of other joints and/or muscles (Hodges & Tucker, 2011).

During motor skill learning plastic changes in the primary motor cortex (MI), such as the expansion and excitability of the cortical areas related to the specific muscles involved in the motor training task are shown to increase; especially during the early stages of motor skill acquisition (Pascual-Leone et al., 1995a; Pascual-Leone, Grafman, & Hallett, 1994; Perez, Lungholt, Nyborg, & Nielsen, 2004; Sanes & Donoghue, 2000). In addition to novel motor skill training, acute experimental pain has been shown to modulate the MI cortical maps both at rest (Dubé & Mercier, 2011) and during motor training (Boudreau al.. 2007). et Acute experimental pain within or proximal to the specific muscles assessed has been reduce the shown to corticospinal excitability (Dubé & Mercier, 2011; Farina et al., 2001; Kofler et al., 1998; Le Pera et al., 2001). In the early stages of motor learning Boudreau et al. (2007) showed that experimental pain suppressed the training-induced plasticity while a study by Ingham, Tucker, Tsao & Hodges (2011) did not. However, the discrepancies of results can be due to the difference in pain models (capsaicin vs. saline), task (novel tongue protrusion vs. finger tapping sequence)



and location of pain (tongue vs. finger). Clarifying whether the inhibition of plastic changes is caused by pain is important since it might impair the ability to learn or relearn a motor task, by obstructing the plastic processes but also impairing the immediate motor performance (Ingham et al., 2011; Lamothe et al., 2014). Additionally, the presence of experimental pain during motor skill acquisition has also been shown to reduce retention performance of the novel motor task, suggesting that the consolidation phases and/or retrieval of the newly learned motor task is influenced by pain (Bouffard, Bouyer, Roy, & Mercier, 2014).

In the area of research examining painful motor skill trainings effect on the motor cortical changes, prior research performed input/output curves to examine the cortical effects of pain on motor training (Boudreau et al., 2007; Ingham et al., 2011). The mapping of motor cortical representations as assessed for the m. tibialis anterior (TA) and m. soleus (SOL) in studv. allows for the present an investigation of the interaction between related muscles and may help reveal MI reorganization after painful motor training exploring the underlying neural by mechanisms that mediate this effect. This might help to get a better understanding of the motor strategies that are employed during painful motor skill training, and create knowledge for rehabilitation in order to create a training approach that might reduce pain, disability and the risk of reinjury (Tsao, Galea, & Hodges, 2010).

The aim of this study was to examine determine if acute experimental pain in the SOL affects the learning of a novel tracetracking task involving fine control of the TA by dorsi flexion of the ankle joint, and how the motor cortical maps of the TA and SOL are affected.



# 2. METHODS

#### Participants

Twenty-four healthy participants (20 males and 4 females, age  $24.79 \pm 0.57$ years, height  $1.81 \pm 0.02$  m, weight  $80.67 \pm$ 2.49 kg, sporting activity per week 6.63 ± 0.58 hours) were semi-randomly divided into two groups (pain and control). The pain and control groups were counterbalanced for age, height, weight and gender. Participants had to be engaged in sports at a minimum of 3 hours per week. Throughout the training of the novel motor skill, the pain group received injections of hypertonic saline in SOL (10 males and 2 females, age  $25.08 \pm 1.01$ years, height  $1.81 \pm 0.03$  m, weight  $79.17 \pm$ 4.07 kg, participations in sports per week  $6.83 \pm 0.94$  hours), and the control group received injections of isotonic saline in the SOL (10 males and 2 females, age 24.5 ± 0.56 years, height 1.82 ± 0.02 m, weight 82.17 ± 2.99 kg, participations in sports per week 6.42 0.72 hours). ± Semirandomization was performed by random.org, where a participant was randomized into one of the groups and when a match was found, this participant was allocated to the opposite group. The group allocation was blinded to the participants. Furthermore, participants completed а transcranial magnetic stimulation (TMS) safety screening and provided written informed consent. Participants had no known neurological or muscular diseases, had no present or previous ankle injuries and were pain free. The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Northern Jutland (N-20140041).

# Procedures

The participants volunteered for a blinded placebo controlled 2-hr single session study. Participants were seated in an armchair during the entire experiment. Participants were prepared for EMG (see



section 'Electromyography (EMG)') and TMS recordings (BrainSight version 2.2.1.4). Motor cortical mapping with TMS was performed before and after the motor skill session, consisting of a pre test, a 24minute training period, and a posttest. Earplugs were provided to the participants during TMS in attempt to dampen noise and maintain a consistent state of mind. For further details of the progress of the protocol see figure 1.

Pain ratings were assessed using a verbal numeric rating scale (VNRS) every minute continuously through the whole motor skill session. The post-training motor skill performance and TMS motor cortical maps were assessed when the participants were pain free again. This was accomplished by asking the participants to rate their pain every 30 seconds until it reached 0 and hereafter an additional 5 minutes was added to make sure the participants were pain free. Finally, a TMS retention test were performed 30-minutes post training (see section '*Motor cortical mapping'*).



**Figure 1.** Illustration of the experimental protocol. The type of injections given were either hypertonic (5.8 %) or isotonic (0.9%) saline and depended on which group the participant was randomized into. Each training-block lasted 4 minutes and a 2-minute period in between was used for reinjection and rest.

# Electromyography (EMG)

Surface EMG was used to record motor evoked potentials (MEP) induced by TMS. Pairs of surface electromyographic (sEMG) electrodes (Neuroline 720 silver/silver chloride, AMBU) were placed on TA and SOL according to SENIAM recommendations and a reference electrode was placed on the tibia bone.

EMG data was collected in the bandwidth of 16-470Hz by the amplifier (BrainSight 2/model 3/SENS-003-001/almond) with a sampling rate of 1 KHz.

# Motor cortical mapping

TMS was applied over the MI before and after the motor skill session by a Magstim stimulator (Magstim Bistim 2). A figure of eight coned coil (Magstim, diameter 7.5 cm) and BrainSight version 2.2.1.4 was used for stimulation over the cortical area of TA. The hotspot, defined as the optimal coil position to evoke the highest peak-topeak MEP's for a given TMS intensity, and the resting threshold (RTh), defined as the lowest stimulation intensity at the hotspot which produced five out of ten stimuli with a minimum peak-to-peak of at least 50  $\mu$ V, were determined for TA. In order to create the motor maps of TA and SOL a grid of 9 by 9 and a spacing of 15 mm, surrounding the hotspot of TA, were performed to evoke TMS-MEPs at each site. Each point in the grid was stimulated three times with an intensity of 110% of the individuals RTh, and the mean of the three TMS-MEP's for each site was assessed offline and used for further data analysis. MEPs for SOL were recorded simultaneously using the same stimulation sites and intensity. Furthermore, five TMS stimuli were performed at the cortical hotspot of TA 30minutes post training and compared to the pre measurements of hotspot excitability of TA. the cortical to assess motor components of retention of the motor skill.



#### Motor skill training and performance

During the motor skill session the participants were seated in an armchair with the legs flexed at 120° at the hip, 160° at the knee, and a plantar flexion of 110° at the ankle (figure 2A). The dominant leg of the participants was established by asking them which leg they would use to kick a ball (Mcgrath et al., 2016). The foot of the dominant leg was strapped to a footplate recording the force applied by the foot. A computer screen was placed 1.5 meters in front of the participant (figure 2A), and a custom made Labview program (Follow Me, version 1.11, Knud Larsen, Aalborg University, Denmark) was used to display and record the trace-tracking of a series of six different and randomized traces of force to be followed (fig. 2B; as previously used by Perez et al., (2004)).



Figure 2. A: Illustration of the experimental setup. The dominant foot of the participant was strapped to the footplate. B: The six randomized traces to be followed during the motor skill session.

The traces were presented as different figures sketching different combinations of movements of dorsi flexion and relaxing of the ankle (figure 2B). This was controlled by the force, produced by TA, applied to the footplate and displayed by a cursor. A countdown of three seconds was visually shown and on the word 'GO', the force-level of the participant's foot on the plate was



displayed in real time overlaying the respective trace. The participants were instructed to observe the traces on the screen in front of them and track, to the best of their ability, each of the 6 different traces (figure 2B), by performing voluntary dorsi flexion and relaxing of the ankle. The traces were displayed one at time and in random order. When performing dorsi flexion the cursor moved towards the top of the screen and when relaxing the ankle the cursor moved back down towards the bottom of the screen. The cursor moved automatically from the left to the right at a pace of 4.4 seconds with randomized 3-4.5 seconds between each trace.

A single training block lasted four minutes (28 traces) followed by a 2-minute period for reinjection and rest. The training period consisted of four training blocks, giving a total training time of 24 minutes (figure 1). The motor skill performance pre and posttest (pre and post motor skill training) consisted of quantifying the accuracy of the tracing to evaluate the motor skill performance and consisted of a 4-minute block of trace-tracking (28 traces). Before the pretest, each participant performed eight traces to get familiar with the task. The motor skill performance tests were performed pain free. Before the motor skill session participants performed three maximal voluntary contractions (MVC) of the TA by a dorsi flexion with the ankle of dominant leg. Participants were the instructed to perform a fast and forceful dorsi flexion to reach the highest possible force within three seconds. During MVC measurements experimenters shouted to motivate the participant. Each MVC was separated by a 1-minute rest period. The Labview program used 20% of the highest force produced in the MVC to set the peak height for each trace.

Trace data was collected at 2 KHz.



# Acute experimental pain

Four intramuscular sterile injections with increasing amounts of isotonic (0.5 mL, 0.6 mL 0.8 mL, 1 mL, 0.9%) or painful hypertonic saline (0.5 mL 0.6 mL 0.8 mL, 1 mL, 5.8%) were injected into the dominant SOL. Injections were located right next to the EMG electrodes on the SOL. One injection was given every five minutes in the two-minute break between the motor skill training blocks (figure 1).

# Subjective assessments of pain

A verbal numeric rating scale (VNRS) was used to assess the pain intensity, ranging from 0 (no pain) to 10 (worst imaginably pain).

The intensity was evaluated by asking the participants to rate their pain intensity in the SOL from 0 to 10. Pain intensity was assessed every minute during the motor skill session, at the time of the countdown in between traces. Furthermore, pain was assessed once prior to the pre and the posttest, ending with a total of 33 measurements. A print of the VNRS was located in front of the participants so the scale was viewable at all times (figure 2A).

# Data analysis

Data was imported and processed using Matlab® version R2015b and Microsoft Excel 2010.

Matlab® was used to extract TMS-MEP's to quantify the motor map area, and to calculate the correlation of trace data (Rvalue). R-value shows the degree of the linear correlation between subject performance and the actual trace and was used to quantify the motor skill performance by the accuracy of the tracing. Excel was used to plot and calculate the motor maps' COG, total excitability of the active sites, VNRS scores and retention data. The active sites were found to calculate area of the excitability. A site was considered active if the mean peak-to-peak amplitude of two out of three MEPS' were



above 50  $\mu$ V. However, the mean value of all three amplitudes had to be above 50  $\mu$ V to be included in the statistics. The mean values of the MEP amplitude are used for further analysis.

Using an established procedure (S. M. Schabrun, Christensen, Mrachacz-Kersting, & Graven-Nielsen, 2016; S. M. Schabrun, Jones, Elgueta Cancino, & Hodges, 2014) discrete peaks were defined if all of the following criteria were met: 1) the MEP amplitude at a grid site was above 50% of the maximum MEP value from the pre test 2) 7 out of 8 of the surrounding grid sites had a reduction in amplitude of at least 5% of the peak MEP amplitude 3) the peak was separated by at least one grid site from another peak that satisfied the first two criteria. For each motor map of each muscle the COG, defined as the amplitudeweighted center of the map (Wassermann, 2012), was calculated using the formula:

$$X = \sum_{i=1}^{N} aixi / \sum_{i=1}^{N} ai$$
$$Y = \sum_{i=1}^{N} aiyi / \sum_{i=1}^{N} ai$$

where ai represents the MEP size at each location, xi represents the x coordinate and yi represents the y coordinate (Wassermann, 2012). The COG position (x,y) of each muscle was compared between the pre- and post-motor skill training for each group to evaluate the change in COG position.

# Statistical analysis

Data was analyzed using SPSS version 23. All data was tested for normality by a Shapiro-Wilk test. With the exception of map COG all data were non-normally distributed. The pretest results of VNRS, trace data, map excitability, map area, map discrete peaks and retention data were tested by a Mann Whitney U test and map COG were tested by a Student's T-test to the make sure that groups were comparable. A repeated measures analysis of variance (RM-ANOVA) was used to analyze the mean change (%) in the map COG position between the factors. The first



factor was groups with two levels (pain and control) and the second factor was time with two levels (pre and post). A Friedman test followed by a Mann-Whitney U post hoc analysis was used to analyze the mean VNRS scores, trace data, map area, map excitability, map discrete peaks and retention data. Map excitability, map area, map COG and retention data were all normalized to their mean value from the pretest to remove baseline variance.

For all tests the significance was set at P < 0.05.

Data are presented as mean values along with standard error of the mean, unless specified otherwise.



# 3. RESULTS

Pretest results for all data (VNRS, trace data, map excitability, map area, map discrete peaks, map COG and retention data) were not significantly different between the two groups (P<0.05).

#### Pain

All participants in both groups scored their VNRS pain rating to be 0 in the pre- and posttest. The mean VNRS pain rating during the motor skill training period was 2.19 ± 0.14 for the pain group and 0.49 ± 0.09 for the control group, which is significantly different from the pre- and posttest (Fig 3; Friedman  $\chi 2(2) = 46$ , P < 0.001; Fig 3; Friedman  $\chi^2(2) = 42$ , P < 0.001). The mean VNRS pain rating for the pain group was significantly higher than the control group (Fig 3; Mann – Whitney U = 12.5, n<sub>1</sub> = n<sub>2</sub> = 23, P <

0.001). The mean VNRS pain rating for the pain group was 2.63  $\pm$  0.22 in 1<sup>st</sup> block of training, 2.6  $\pm$  0.26 in 2<sup>nd</sup> block, 2.22  $\pm$  0.21 in the 3<sup>rd</sup> block and 1.72  $\pm$  0.23 in the 4<sup>th</sup> block. The mean VNRS pain rating for the control group was 0.28  $\pm$  0.1 in 1<sup>st</sup> block of training, 0.45  $\pm$  0.18 in 2<sup>nd</sup> block, 0.6  $\pm$  0.21 in the 3<sup>rd</sup> block and 0.83  $\pm$  0.22 in the 4<sup>th</sup> block(Fig.3).





**Figure 3.** Pain rating scores for the control group (grey diamond-shapes, n=12) and the pain group (black square-shaped, n=12) as collected during the motor skill session. The first and last five measurements were obtained during the pre- and posttest. Grey areas indicate the motor skill training blocks and arrows indicate the timing of injections.

#### Motor skill performance

The motor skill performance increased for both the pain group and the control group from the pre- to the posttest (Fig. 6; Friedman  $\chi 2(1) = 24$ , P < 0.001). No significant difference in the motor skill performance was found between the two groups (Fig 6; Mann – Whitney U = 95 n<sub>1</sub> = n<sub>2</sub> = 12, P = 0.198).

Figure 4 and 5 illustrates representative examples of the improvement that occurs in the motor skill performance trace data from the pre- and posttest, for one participant from each group. For the representative participant of the control group (figure 4A, 4B) the correlation between shown traces and actual participant performance increased from 0.627 to 0.771. For the representative participant of the pain group (figure 4C, 4D) the correlation between shown traces and actual participant performance increased from 0.6 0.817. to





**Figure 4.** Representative examples of the relationship between the trace-target level ( $\mu V$ ) and participant performance ( $\mu V$ ) for all data points for one participant from the control group (figure 4A, 4B) and one from the pain group (4C, 4D).



**Figure 5.** Representative examples of the participant performance on trace-tracking pre- and post training, for one participant from the control group (figure A and B) and one from the pain group (figure C and D).

The mean R-value for the pain group was  $0.69 \pm 0.03$  for the pretest and  $0.84 \pm 0.02$  for the posttest (Fig. 6).

The mean R-value for the control group

was 0.72  $\pm$  0.02 for the pretest and 0.85  $\pm$ 

0.02 for the posttest (Fig 6).



**Figure 6.** The mean trace correlation scores (*R*-values) for the control group (n=12) and the pain group (n=12), for the pre- and post-training tests. (\**P*< 0.05)

# Motor cortical mapping

Table 1 shows the mean change in percentage for the map COG lateral-medial (LM), map COG posterior-anterior (PA), map excitability and map area from the pre- to the posttest for both groups. The change in the map COG PA and LM was not significant for either group from the pre- to posttest for TA (RM – ANOVA: F(1,22) =



0.009, P = 0.924; RM - ANOVA: F(1,22) = 0.059, P = 0.81) or SOL (RM - ANOVA: F(1,22) = 0.052, P =

0.822; RM – ANOVA: F(1,22) = 0.021, P = 0.886).

The change in the map area was not significant for either group from the pre- to posttest for ТΑ (Friedman  $\chi^2(1) =$ 0.168, P = 0.863) or SOL (Friedman  $\chi^2(1) = 2.13, P = 0.144$ ). The change in the map area was not significant between groups for TA (Table 1; Mann-Whitney U =89,  $n_1 = n_2 = 12$ , P = 0.347) or SOL (Table 1; Mann-Whitney U =98,  $n_1 = n_2 = 12$ , P = 0.143). The change in the map excitability was not significant from the pre to posttest for either group for TA (Friedman  $\chi^2(1) = 0.000$ , P = 1) or SOL (Friedman  $\chi^2(1) = 0.167$ , P = 0.683). The change in the map excitability was not significant between groups for TA (Table 1; Mann-Whitney U =81  $n_1 = n_2 = 12$ , P = 0.63) or SOL (Table 1; Mann-Whitney U =63,  $n_1 = n_2 = 12$ , P = 0.63). The change in the map discrete peaks was not significant from the pre- to posttest for either group for TA (Friedman  $\chi^2(1) = 3$ , P = 0.083) or SOL (Friedman  $\chi^2(1) = 0.091$ , P = 0.763). The change in the map area was not significant between groups for TA (Table 1; Mann-Whitney U =83.5  $n_1 = n_2 = 12$ , P = 0.514) or SOL (Table 1; Mann-Whitney U =67  $n_1 = n_2 = 12$ , P = 0.799).

**Table 1.** The TMS neurophysiological measures for both the TA and the SOL muscle, for the control (n=12) and the pain (n =12) group. Map COG LM, map COG PA, map excitability and map area are normalized and presented as the mean change in percent from the pre- to the posttest, and the map discrete peaks are presented as the mean number of peaks, for the pre- and posttest.

	Control		Pain	
	ТА	SOL	ТА	SOL
Map COG LM (%)	4.37 ± 0.98	$4.27 \pm 0.8$	$3.28 \pm 0.73$	$3.37 \pm 0.84$
Map COG PA (%)	22.94 ± 5.15	19.44 ± 4.15	33.14 ± 6.57	37.39 ± 8.39
Map excitability (%)	5.97 ± 6.16	$0.69 \pm 10.78$	$0.65 \pm 6.42$	$13.18 \pm 12.78$
Map area (%)	50.89 ± 16.36	123.59 ± 55.37	26.19 ± 4.09	86.68 ± 47.31
Map discrete peaks pre (number)	$1.42 \pm 0.18$	$1.58 \pm 0.14$	$1.58 \pm 0.25$	1.75 ± 0.39
Map discrete peaks post (number)	1.83 ± 0.31	1.67 ± 0.36	1.67 ± 0.32	1.83 ± 0.37



**Figure 7.** An example of the motor cortical maps for TA and SOL for the pre- and posttest for a participant in the control group (n = 1). The colored scale represents the mean MEP amplitude relative to the highest MEP value presented as 1.





Figure 8. An example of the motor cortical maps for TA and SOL for the pre- and posttest for a participant in the pain group (n =1). The colored scale represents the mean MEP amplitude relative to the highest MEP value presented as 1.

For both TA and SOL, figure 7 and 8 shows representative examples of the motor cortical map from the pre- to the posttest.

#### Retention

All retention data (mean hotspot excitability) are presented in % relative to the highest value in the pretest. For the control group, the change in the mean hotspot excitability for the post 30-minute test was 108 % ± 11.2 for TA and 107.32 % ± 15.26 for SOL for the posttest (Fig. 9), and 90.04 % ± 9.68 for TA and 110.34 % ± 12.61 for SOL (Fig. 9). For the pain group, the change in the mean hotspot excitability for the post 30-minute test was 126.25 % ± 32.22 for TA and 123.79 % ± 25.89 for SOL for the posttest (Fig. 9), and 93.43 % ± 22.58 for TA and 137.71 % ± 40.14 for SOL (Fig. 9). No significant changes in the mean



hotspot excitability was shown between the pre-test, posttest and the post 30 minutes for either group for either TA (Fig. 9; Friedman  $\chi^2$  (2) = 3.083, P = 0.214) or SOL (Fig. 9; Friedman  $\chi^2$  (2) = 0.583, P=0.747).



**Figure 9.** Diagram of the mean hotspot excitability (%) for the pretest, posttest and post 30 minutes after the motor skill training period, normalized to the mean hotspot value in the pretest.

#### 4. DISCUSSION

The aim was to determine if acute experimental pain in the SOL affects the learning of a novel trace-tracking task involving fine control of TA by dorsi flexion of the ankle joint, and determine if the motor cortical maps of TA and SOL were affected. The acquisition of the novel motor skill was unaffected by the presence of pain during training. Furthermore, the motor skill training did not affect the cortical maps of TA and SOL and the presence of pain did not exert any additional modulations on these cortical maps.

#### Pain

The pain group experienced a significantly higher level of pain than the control group. Even though the control group rated a mean pain of  $0.49 \pm 0.09$  it can still be



considered a pain free group since the value is below 1 and therefore categorizes as 'no pain' according to the pain intensity numerical rating scale (Farrar, Young, Lamoreaux, Werth, & Poole, 2001; Hines, Urman, & Vadivelu, 2011).

The mean pain ratings were comparable with a previous study using a similar pain model (1.7  $\pm$  SD 1.0 for pain and 0.2  $\pm$  SD 0.4 for control; Ingham et al., 2011). For clinical pain conditions, the effect of pain on motor control may involve many confounding factors that can make it difficult to distinguish the separate effects of nociceptive input on the motor system. This could be physiological impairments like musculoskeletal etiologies (Rajasekaran et al., 2012) and/or cognitive impairments as catastrophizing where an increased attentional capture and disruption is experienced by pain (Heathcote et al., 2015). By using experimental acute pain in healthy participants in the present study, the motor

consequences of acute pain are thereby isolated (Bank, Peper, Marinus, Beek, & van Hilten, 2013).

The effect of pain on motor performance The motor skill performance was significantly improved regardless of the presence of pain, and furthermore, the extent of performance improvement was not different between the groups. Firstly, the control group demonstrated that the quality and quantity of training was sufficient to ensure an improvement in the motor skill performance and secondly, the presence of pain did not interfere with the improvement. These results are in line with previous studies showing that the acquisition of a motor skill was unaffected by experimental pain (Bilodeau, Roosink, & Mercier, 2016; Bouffard et al., 2014; Ingham et al., 2011). However, other studies have shown the improvement in motor skill performance to be significantly lower for the group training with pain



compared to the group training without pain (Boudreau et al., 2007; Lamothe et al., 2014). Multiple factors might explain these discrepancies, such as the location and type of pain, and the type and complexity of the task. When pain was induced directly onto the working muscle in the study by Boudreau et al. (2007) the execution of the training may have been more painful as previously shown by Kothari, Svensson, Huo, Ghovanloo, & Baad-hansen (2012). It is well known that changes in motor control occur as an adaptation to pain, where a change in motor strategy acts as the body's immediate strategy to take care of the injured limb (Hodges & Tucker, 2011). In contrast to the present study, which did not show pain in the antagonist muscle to impair the execution of the motor performance, the participants in the study by Boudreau et al. (2007) may have obtained a disadvantageous motor strategy to minimize the pain, which may be an explanation of their impaired acquisition of the motor skill (Lamothe et al., 2014). By inducing pain in the antagonist muscle the present study may have avoided this along with the influence of biomechanical changes in the working muscle, such as volume effects and changes in muscle stiffness. Furthermore, the fairly low pain rating of the pain group could have been a limitation of the present study, since it categorizes as mild pain (Hines et al., 2011) and may have been too low to inhibit the motor performance. Additionally, since the participants were active in sports and therefore used to experiencing painful events during their training (Hopkins et al., 2007), the mild pain may not have been a determining factor on their performance. Moreover, the fluctuating pain ratings that were observed throughout the training period could have had an influence, since participants did not experience the consistent pain throughout the duration of the training. This is supported by the results of Ingham et al. (2011) who found



a similar pain rating of the local muscle to have no effect on the motor skill performance, and by Seeley, Park, King, & Hopkins (2013) who showed a consistent pain rating throughout the experiment to impair the motor performance.

#### Motor cortical maps

Motor skill training is known to increase motor cortical excitability (Liepert, Terborg, & Weiller, 1999; Pascual-Leone et al., 1995b; Pascual-Leone et al., 1994; Perez et al., 2004). However, even though the motor skill performance was significantly improved for both groups, no changes in motor cortical maps were found. As demonstrated by the control group the motor skill training may not have been sufficient to elicit any motor cortical changes. Since a study by Perez et al. (2004) showed significant changes in excitability following a longer duration of training (32 minutes) of the same motor task as in the present study, the duration of training in this study (24 minutes) may have been inadequate to elicit these Furthermore, since previous changes. studies have shown that different pain modalities can induce an inhibitory effect on motor cortical excitability (Dubé & Mercier, 2011; Farina et al., 2001; Kofler et al., 1998; Le Pera et al., 2001), the fact that pain did not elicit any changes in excitability suggests that the saline injections in the antagonist muscle may have been inadequate to have an effect on the motor cortical maps. The previous studies examine experimental pain in the target muscle of the upper extremities (Dubé & Mercier, 2011; Farina et al., 2001; Kofler et al., 1998; Le Pera et al., 2001), indicating that the type and location of pain may have an influence.

No prior studies have analyzed changes in map discrete peaks in relation to the acquisition of a novel motor skill. However, few studies have looked at discrete peaks

relation based in to pain on an experimental model with injections of nerve growth factor (NGF) in the lower arm (S. M. Schabrun et al., 2016) or on chronic elbow pain (M. Schabrun S., Hodges, Vicenzino, Jones, & Chipchase, 2015), and both have linked an increase in discrete peaks to the presence of pain. An explanation as to why the present study did not show an increase in discrete peaks might be found in the duration of the pain. The increase in peaks seen by S. M. Schabrun et al. (2016) was not observed until pain had persisted for four days, hereby linking the number of peaks to the chronic components of pain.

Furthermore, the present study did not show map COG to be influenced by pain, which is opposite to the study of Tsao et al. (2010) who found a change in COG position for chronic low back pain patients following motor skill training.

#### Retention

The present study did not find pain to affect the MI excitability at the post 30minute retention, which was in contrast to the study by Bouffard et al. (2014) who showed that experimental pain at the ankle impairs the motor performance retention. However, study investigates the retention performance and not MI excitability as in the present study, and furthermore, this was assessed 24 hours post training. The time between the training and retention test could therefore be a limitation for the study since 30 minutes may be inadequate for the motor consolidate (Brashers-Krug, skill to Shadmehr, & Bizzi, 1996).

#### Perspective

The influence of pain on motor learning is an essential in the area of sports where painful leg injuries are reported as a common complaint among athletes (Burrus et al., 2015; Rajasekaran et al., 2012).



Musculoskeletal injuries of the lower extremities have been shown to increase the reaction time, which has been associated with a higher prevalence of injuries (Taimela & Kujala, 1992), and this is critical since an athlete training in pain will be at an increased risk of reinjuring or acquiring other injuries (Taimela & Kujala, 1992).

However, since the present study showed that pain did not affect the ability to acquire a new motor skill, this knowledge can be useful in rehabilitation since patients can acquire new or modified motor skills without any influence of pain. This could help them move or perform in a more beneficial manner, and help to resolve the maladaptive changes in motor strategy that occurs during pain (Brindle, Mattacola, & McCrory, 2003; Cowan, Bennell, Hodges, Crossley, & McConnell, 2001). Failure of such changes to resolve is it is associated with critical since

recurrence of pain or long-term detrimental impairments of other tissues (Hodges & Moseley, 2003; Hodges & Tucker, 2011).

# Conclusion

The novel trace-tracking acquisition was not impaired by the presence of pain training. during Motor skill training significantly improved motor skill performance equally for both groups. Furthermore, the motor skill training did not affect the cortical maps of TA and SOL and the presence of pain did not exert any additional modulations on these cortical maps. Thus, the present study indicates that painful motor training does not always interfere with motor skill acquisition or motor cortical maps and highly depends on the pain model and task to be learned.



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