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Original Research Article

Accelerated Muscle Fatigability of Latent Myofascial Trigger Points in Humans

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Abstract

Objective. Muscle fatigue is prevalent in acute and chronic musculoskeletal pain conditions in which myofascial trigger points (MTPs) are involved. The aim of this study was to investigate the association of latent MTPs with muscle fatigue.

Design. Intramuscular electromyographic (EMG) recordings were obtained from latent MTPs and non-MTPs together with surface EMG recordings from the upper trapezius muscles during sustained isometric muscle contractions in 12 healthy subjects.

Outcome Measures. Normalized root mean square (RMS) EMG amplitude and mean power frequency (MNF) were analyzed. The rate of perceived exertion and pain intensity from MTP side and non-MTP side were recorded.

Results. Pain intensity on the MTP side was significantly higher than the non-MTP side ($P < 0.05$). Intramuscular EMG from latent MTPs showed an early onset of decrease in MNF and a significant decrease at the end of fatiguing contraction as compared with non-MTPs ($P < 0.05$). Surface EMG from muscle fibers close to latent MTPs presented with an early onset of the increase in RMS amplitude and the increase was significantly higher than that from non-MTPs at the end of sustained isometric contraction ($P < 0.05$).

Conclusions. A latent MTP is associated with an accelerated development of muscle fatigue and simultaneously overloading active motor units close to an MTP. Elimination of latent MTPs and inactivation of active MTPs may effectively reduce accelerated muscle fatigue and prevent overload spreading within a muscle.

Key Words. Electromyography; Fatigue; Motor Control; Myofascial Trigger Point; Pain

Introduction

Prolonged dynamic exercise and sustained isometric contractions induce muscle fatigue, as manifested by task failure and/or a reduction in the maximum voluntary contraction force [1,2]. Lower muscle strength and accelerated fatigue development are commonly reported in patients with chronic musculoskeletal pain [3,4], where both latent and active myofascial trigger points (MTPs) contribute significantly to the generation of pain and motor dysfunction [5–7]. However, the relationship between accelerated fatigability and MTPs has not been investigated.

Surface electromyographic (EMG) recordings are often performed to monitor the development of muscle fatigue during sustained contraction. Muscle fatigue development is characterized by increased EMG amplitude and concomitantly decreased frequency contents [8,9], underlining changes in the population of active motor units and their firing patterns. Unlike surface EMG, intramuscular EMG enables high spatial selectivity and was thus used in the present study to record motor unit activity directly from an isolated MTP in the muscle in an effort to observe its fatigue characteristics.

Muscle fatigue may arise not only from peripheral changes at the level of the muscle, but also from the central nervous system [10]. Central fatigue may be affected

by psychological factors, such as perceived effort or physiological factors, such as inhibition of pathways that prevent efficient activation of motor neuron pools [10]. At the peripheral level, limitations in energy supply, including energy available from phosphocreatine hydrolysis, anaerobic glycolysis and oxidative metabolism, as well as the intramuscular accumulation of metabolic by-products, such as hydrogen ions, emerge as key factors responsible for fatigue [11]. MTPs have been reported to show an increased concentration of algesic substances, including hydrogen ions [12] and muscle ischemia [13] associated with sympathetic hyperactivity [14]. Apart from the sensory hypersensitivity at MTPs, latent MTPs have been found to be associated with abnormal muscle activation pattern [15] and increased resting motoneuron excitability [16]. These mechanisms may predispose taut muscle fibers harboring MTPs to an early onset of muscle fatigue in sustained isometric contractions.

The aim of this study was to determine muscle fatigue characteristics derived from surface and intramuscular EMG recordings at latent MTPs as compared with non-MTPs during sustained isometric contractions in healthy subjects. We therefore hypothesized that MTPs would contribute to accelerated muscle fatigue. Confirmation of this hypothesis may provide an explanation to the prevalent muscle fatigue symptoms in musculoskeletal pain conditions.

Methods

Subjects

Twelve healthy subjects (eight males and four females, mean age: 27.4 ± 3.6 years), with no signs or symptoms of musculoskeletal pain, volunteered for this study following advertisement on the university campus. Each subject had at least one latent MTP bilaterally in the upper trapezius muscle. The study was approved by the local Ethics Committee (20080018) and conducted in accordance with the Helsinki Declaration. Informed consent was obtained from all subjects.

Experimental Protocol

Each subject was seated in a straight-backed chair against a wall with both knees and hips flexed at 90°and arms held relaxed along the lateral sides of the trunk during electrodes placement. Following skin preparation, bipolar surface EMG electrodes (Ambu, 720-01-K, Ballerup, Denmark) were placed 20 mm laterally from the mid-distance between C7 and acromion [17] in the direction of the muscle fibers of the upper trapezius. The reference electrodes were positioned over the skin surface of medial corner of scapular and of the wrist (right side). A latent MTP (most tender spot along a muscle taut band) 20–30 mm medial to the surface electrodes was identified in each subject and marked in the upper trapezius muscle on either the dominant or nondominant side. Then a non-MTP (nontender, out of taut muscle band) was marked symmetrically on the contralateral side. The selection of the dominant or nondominant side to identify a latent MTP was balanced and randomized according to a random numbers table in order to exclude the effect of shoulder dominance on the level of EMG activity during muscle contraction. Following skin preparation, an intramuscular concentric EMG needle (Ambu, 25 × 0.3 mm) was inserted slowly into the latent MTP at the marked point until the presence of spontaneous electrical activity (SEA) in the needle electrodes and the absence of surface EMG activity in the surface electrodes were achieved as shown elsewhere [16]. Another identical intramuscular concentric EMG needle was inserted into the muscle at the marked non-MTP with no intramuscular SEA and no surface EMG activity on the monitor. With both intramuscular needles in place, subjects were asked to slowly abduct both arms to 90° and hold the position until task failure (detailed later). The rate of perceived exertion and pain intensity were recorded at the end of isometric contraction (detailed later). Surface and intramuscular EMG data from both MTPs and non-MTPs were collected and stored for further analysis (detailed later).

This study is a one session experiment with one episode of sustained contraction. The rationale for this experimental design is to minimize the effect of supraspinal factors on muscle fatigue characteristics as EMG activities from MTPs and non-MTPs were recorded under the same exertion level in each subject, thus the differences in EMG fatigue characteristics between MTPs and non-MTPs were mainly due to the factors from the spinal and muscle levels.

Sustained Isometric Shoulder Abduction, Rate of Perceived Exertion, and Pain Intensity

The subjects were asked to hold both arms in 90° abduction until exhaustion (task failure), with elbows fully extended and forearms pronated with palms facing toward the ground. During the sustained isometric contraction, subjects were asked to make contact with the two levers horizontally over their forearms with the head in an untwisted and upright position. The volunteers were asked to stop the shoulder abduction as soon as the forearms failed to make the contact with the levers. Immediately after the shoulder abduction, the overall rate of perceived exertion recorded on a 10-cm visual analog scale (VAS), in which 0 corresponded to “no perceived exertion” and 10 to “maximal perceived exertion,” defined as the point where subjects were unable to maintain their arms in the initial position.

Pain intensity on each side of the trapezius was recorded on a 10 cm VAS scale as described elsewhere [18].

Intramuscular and Surface EMG Recordings

The EMG signals were amplified 2,000 times and band-pass filtered at (5–500 Hz). Intramuscular needle/wire was placed at the MTP and non-MTP (usually at the middle point on the line between C7 and the acromion) of the upper trapezius. The intramuscular EMG signals were amplified 2,000 times and band-pass filtered at (10–

2,000 Hz), which is used for detecting SEA at MTPs [19]. Surface EMG and intramuscular EMG were recorded (DANTEC Counterpoint Electromyograph, Dantec Medical A/S, Skovlunde, Denmark) synchronously at 5 kHz, converted in digital form by a 12-bit A/D converter and stored on disk.

Data Analysis

The absolute root mean square (RMS) and the mean power frequency (MNF) values of the surface EMG and intramuscular EMG signals were computed for the entire contraction period. The power spectral density of the surface EMG and intramuscular EMG signals were estimated by using Welch's averaged periodogram method [20]. The surface EMG and intramuscular EMG signals were divided into overlapping sections of 1 second. Each of the sections were windowed (Hanning window of 8,192 samples), and zero-padded to length (8,192 samples). Absolute RMS and MNF values were normalized to the initial RMS (time domain) and MNF value (frequency domain) to account for intersubject variability of temporal and spectral estimators due, for example, to skin fold thickness or muscle size [21].

To reduce the amount of data obtained during the continuous contraction, the first 20 computed values of the above mentioned parameters were averaged to obtain the first mean values at time 0% of the time to exhaustion. The 10 computed values before and after 25% of the time to exhaustion were averaged to obtain the mean values at time 25% of the time to exhaustion. This procedure was repeated at 50% and 75% of the time to exhaustion. The mean values at time 100% of the time to exhaustion were obtained by averaging the last 20 values computed. All in all, five mean values (0%, 25%, 50%, 75%, and 100% of the time to exhaustion) were used to describe changes in the temporal and spectral domains during sustained isometric contraction.

Statistical Analysis

Paired *t*-test was used to compare the difference in the rate of perceived exertion and pain intensity between the MTP side and non-MTP side. Two-way repeated measure analysis of variance (ANOVA) was applied to detect differences in surface and intramuscular EMG normalized RMS and MNF at different time intervals (0-25-50-75-100% of contraction time) between MTPs and non-MTPs. Student-Newman-Keuls (SNK) method was followed for post hoc comparisons. The data are presented as mean ± standard error of the mean and the significance level was set to $P < 0.05$.

Results

The Rate of Perceived Exertion and Pain Intensity

The total time length of sustained isometric contraction until exhaustion was 7.42 ± 0.26 minutes. The rate of perceived exertion was 8.68 ± 1.36 cm following the sustained isometric contraction, being same for both MTP side and non-MTP side. On the contrary, pain intensity was significantly higher ($t = 2.19$, $P = 0.03$) during sustained isometric contraction on the MTP side than non-MTP side (Figure 1).

Differences in Surface EMG RMS and MNF Between MTP Side and Non-MTP Side

There was a significant increase in surface EMG RMS over time (ANOVA, $F = 4.36$, $P = 0.005$). There was no interaction between time interval and point type (ANOVA, $F = 2.18$, $P = 0.08$) due to the similar trend of the increased RMS over time. Post hoc analysis showed that at surface EMG RMS on the MTP side was significantly higher the non-MTP side at 100% of the contraction time

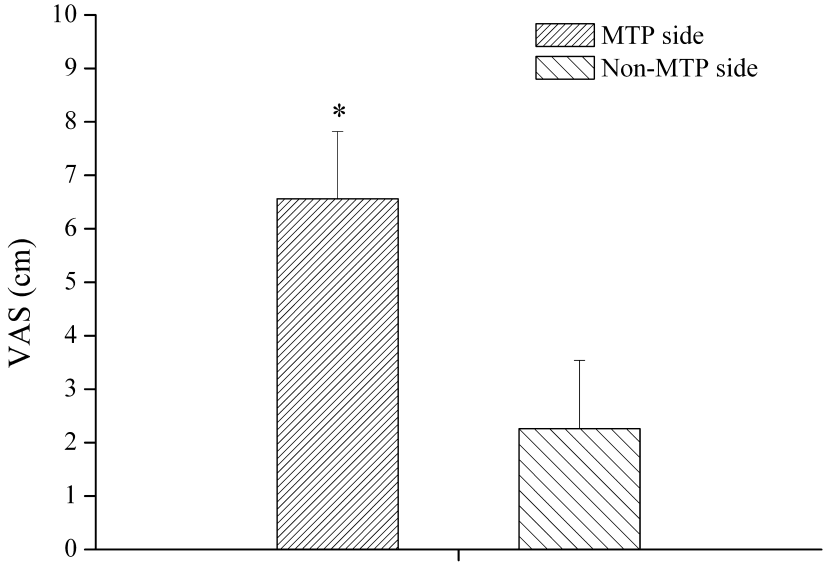

Figure 1 Pain intensity following sustained isometric contractions. Pain intensity (VAS) is significantly higher on the latent myofascial trigger point (MTP) side than on the non-MTP side (*$P < 0.05$).

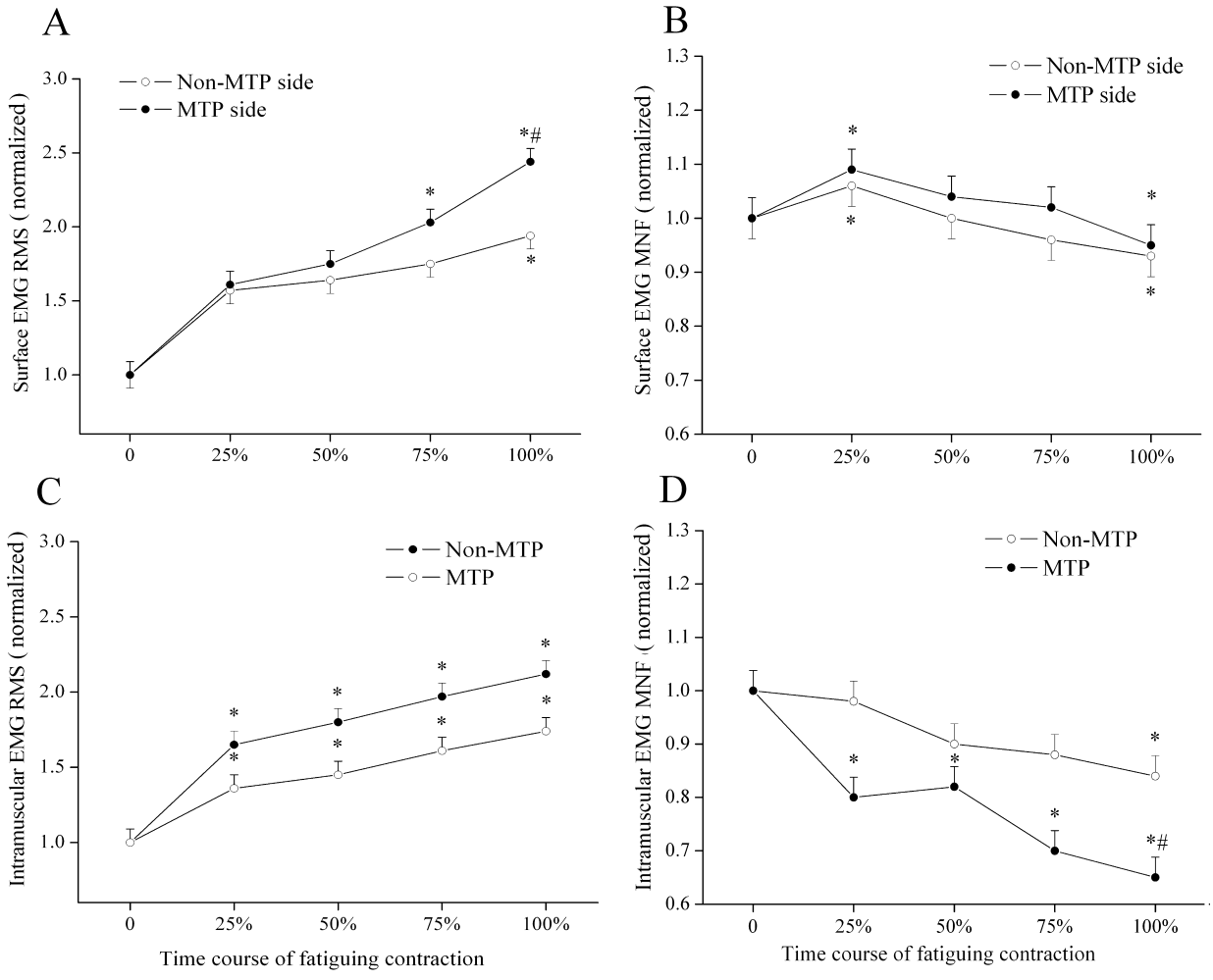

Figure 2 Surface electromyographic (EMG) changes in normalized root mean square (RMS, A) and normalized mean power frequency (MNF, B) on myofascial trigger point (MTP) side and non-MTP side in the upper trapezius during sustained isometric contractions. Surface EMG on the MTP side reflects the activity of active motor units close to an MTP. Intramuscular EMG changes in normalized RMS (C) and normalized MNF (D) from latent MTPs and non-MTPs in the upper trapezius during sustained isometric contractions. * represents significant difference as compared with that at the start of the sustained isometric contraction. # represents significant group difference.

(SNK, $P < 0.05$); Within the MTP side, surface EMG RMS at 75% and 100% contraction time was significantly higher than that at 0% contraction time (SNK, both: $P < 0.05$), but within the non-MTP side, surface EMG RMS at 100% contraction time only was significantly higher than that at 0% contraction time (SNK, $P < 0.05$, Figure 2A).

There was a significant difference in surface EMG MNF over time (ANOVA, $F = 8.99$, $P < 0.001$). There was neither significant difference in surface EMG MNF between MTPs and non-MTPs (ANOVA, $F = 3.41$, $P = 0.09$) nor significant interaction between time interval and point type (ANOVA, $F = 2.19$, $P = 0.08$) due to the similar changes of the MNF over time. Post hoc analysis showed that surface EMG MNF at 25% contraction time was significantly higher than that at 0% contraction time (SNK, $P < 0.05$) and surface EMG MNF at 100% contraction time was significantly lower than that at 0% contraction time within either the MTP side or the non-MTP side (SNK, both: $P < 0.05$, Figure 2B).

Difference in Intramuscular EMG RMS and MNF between MTPs and Non-MTPs

There was a significant increase in intramuscular EMG RMS over time (ANOVA, $F = 6.95$, $P = < 0.001$). Neither difference in intramuscular EMG RMS between MTPs and

non-MTPs (ANOVA, $F = 1.94$, $P = 0.19$) nor interaction between time interval and point type (ANOVA, $F = 1.55$, $P = 0.20$) were found. Post hoc analysis showed that intramuscular EMG RMS at the 25%, 50%, 75%, and 100% of the contraction time was significantly higher than 0% contraction time within both groups (SNK, all, $P < 0.05$, Figure 2C).

There was a significant decrease in intramuscular EMG MNF over time (ANOVA, $F = 11.62$, $P < 0.001$). There was a significant interaction between time interval and point type (ANOVA, $F = 2.69$, $P = 0.04$). Post hoc analysis showed intramuscular EMG MNF was significantly lower in the MTP group than the non-MTP group at 100% of the contraction time (SNK, $P < 0.05$). Within the MTP group, intramuscular EMG MNF at 25%, 50%, 75%, and 100% contraction time was significantly lower than that at 0% contraction time (SNK, all: $P < 0.05$, Figure 2D). Within the non-MTP group, intramuscular EMG MNF at 100% contraction time only was significantly lower than that at 0% contraction time (SNK, $P < 0.05$, Figure 2D).

Discussion

In the present study, muscle fatigue developed as shown by an increase in EMG amplitude and a decrease in frequency contents of surface and intramuscular EMG. The major new finding of the current study is that latent MTPs are associated with an accelerated muscle fatigue as depicted by the early decrease in intramuscular EMG mean power frequency compared with non-MTPs. Also noteworthy is the increased motor unit activity of muscle fibers close to latent MTPs as shown by the early and significant increase in surface EMG RMS amplitude compared with non-MTPs.

Latent MTPs Are Associated with Accelerated Development of Muscle Fatigue

Intramuscular EMG recordings in the current study represent electrical activities from focal muscle motor units at latent MTPs and non-MTPs. While both latent and non-MTPs show similar increases in intramuscular EMG RMS, intramuscular EMG MNF showed an early significant decrease from latent MTPs as compared with non-MTPs (at 25% and 100% contraction time for latent MTPs and non-MTPs, respectively). Further, the decrease in intramuscular EMG MNF was more significant for latent MTPs than non-MTPs at task failure (Figure 2D). These results suggest that the development of muscle fatigue at latent MTPs is approximately four times faster than at non-MTPs.

An increase in the RMS amplitude reflects additional recruitment of motor units and/or motor units substitution to maintain the same force level [8,22]. The decrease in MNF reflects changes in the firing properties of the active motor units, a slowing in muscle fiber conduction velocity and/or a change in the synchronization of motor unit firing [8,9,23,24]. Peripheral fatigue relates to factors within the muscle that cause impaired contractile function during strenuous exercise [1,25]. The current study design permits the equal amount of central command to latent and non-MTPs, allowing us to decompose the mechanisms of the accelerated fatigability of MTPs largely into the spinal and muscular levels. At the muscle level, sustained physical activity affects the biochemical equilibrium within the exercising muscle cells. Among others, inorganic phosphate, protons, lactate, and free Mg2+ accumulate within these cells and appear to inhibit cross-bridge formation and activation [26,27]. In addition to the increased concentration of metabolites during muscle fatigue, decreased EMG MNF is reported to be positively associated with muscle ischemia [28]. Based on these mechanisms of muscle fatigue at muscle fiber level, it is not surprising to observe the result in the current study that MTPs are associated with accelerated fatigability.

At the muscle level, sustained focal muscle fiber activity and/or muscle cramps at latent MTPs [7,29] may induce focal accumulation of metabolites and algesic substances, such as bradykinin, substance P, norepinephrine, protons (lower pH), etc [12]. The higher pain intensity reported from latent MTPs compared with non-MTPs during sustained muscle contraction (from needle insertion to task failure) suggest that local muscle pain may impair motor unit function and the development of muscle fatigue may in turn enhance muscle hyperalgesia [2,30,31]. Muscle ischemia has also been consistently reported to be associated with MTPs across a number of studies [13,14,32] and reduced muscle oxygenation may dose dependently reduce muscle twitch force [33].

At the spinal level, the efficiency of the reciprocal inhibition evoked by spinal inhibitory interneurons is decreased when their target motoneurons are active, even in the absence of supraspinal influence [34]; motoneurons innervating motor endplates of MTPs are spontaneously active and may thus contribute to the decreased reciprocal inhibition at spinal level [35].

Although the detailed mechanisms of accelerated development of muscle fatigue at MTPs are not fully understood, the finding of accelerated development of muscle fatigue at MTPs in the current study has important clinical significance as fatigue is prevalent in musculoskeletal pain conditions [2]. Elimination of latent MTPs and inactivation of active MTPs may effectively reduce muscle fatigue.

Increased Motor Unit Activities of Muscle Fibers Close to Latent MTPs

In the current study, surface EMG recordings from motor units close to latent MTP in the upper trapezius (2 cm lateral to the latent MTP) showed an earlier increase in RMS (starting from 75% contraction time) as compared with 100% contraction time for the non-MTP side. Further, the level of activation of the upper trapezius, i.e., surface EMG RMS was higher for MTP side than non-MTP side at task failure (Figure 2A). These results indicate that the

activity of active motor units close to latent MTPs is increased to compensate for the reduced force output (fatigue) from latent MTPs during sustained muscle contractions. Similarly, a transient increase in surface EMG MNF at 25% contraction time for both MTP and non-MTP sides (Figure 2B) may suggest an increase in firing rate of motor units, even though small changes are reported in the upper trapezius [36]. It is also interesting to note that intramuscular EMG RMS from latent MTPs is slightly higher (though not significant) as compared with non-MTPs (Figure 2C), in addition to the increased surface EMG RMS amplitude from active motor units close to latent MTPs compared with non-MTP side (Figure 2A). These results may suggest a specific sensory motor control strategy in MTP-related pain, i.e., MTP pain is associated with increased motor unit activity and/or motor unit excitability. In line with this notion, we have shown that muscle pain induced by needle insertion into latent MTPs is positively associated with the amplitude and duration of SEA [37] and that painful activation of latent MTPs with algesic substance glutamate induces muscle cramps [38]. Thus, there is a reciprocal interaction between MTP pain and motor unit activity, which may provide part of the explanation to the increased motor unit activity within a painful muscle in experimental studies [39] and in chronic pain patients [2,4,40]. Increased motor unit activity associated with MTPs may also contribute to the decreased shifts in activity between motor unit subpopulations in a single muscle during monotonous contractions [41,42] and lead to early development of muscle fatigue [43]. It is our speculation that when a muscle contains a latent MTP, the surrounding normal muscle fibers would be overloaded to perform the same amount of force; the overloaded muscle fibers may in turn become taut and lead to the formation of new latent MTPs, predisposing further muscle dysfunction and local pain propagation. It would be interesting to see if the rapid fatigue and the recruitment of compensatory muscle fibers could be rapidly reversed with inactivation of the latent MTPs.

The current study investigates latent MTPs in healthy subjects but not active MTPs in patients, thus the results may not reflect the complex nature of muscle fatigue characteristics in chronic pain patients. Moreover, it should be emphasized that the transition from muscle fatigue to musculoskeletal pain is still under debate [2]. The small sample size may limit the validity of the current results. Further studies investigating active MTPs in patients suffering from neck-shoulder musculoskeletal disorders are warranted.

Conclusions

The current study shows that latent MTPs are associated with accelerated development of muscle fatigue, which may overload active motor units close to an MTP. Elimination of latent MTPs and inactivation of active MTPs may effectively reduce muscle fatigue and prevent overload spreading within a single muscle in musculoskeletal pain conditions.

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