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One hour jaw muscle training does not evoke plasticity in the corticomotor control of the masseter muscle

Shengyi Lu [a,b,*], *Lene Baad-Hansen* [b], *Zhenting Zhang* [a], *Peter Svensson* [b,c]

[a] *Department of Oral Implantology, School of Stomatology, Capital Medical University, Dongcheng District, Beijing 100050, PR China*

[b] *Section of Clinical Oral Physiology, Department of Dentistry, Aarhus University, Aarhus, Denmark*

[c] *Center for Functionally Integrative Neuroscience (CFIN), MindLab, Aarhus University Hospital, Aarhus, Denmark*

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ABSTRACT

Transcranial magnetic stimulation (TMS) has been used to assess corticomotor control of jaw muscles, but few studies have examined cortical plasticity of the masticatory system and potential modification by jaw muscle training.

Objectives: To determine if a 1-h jaw muscle training task would be sufficient to induce signs of neuroplastic changes in the corticomotor excitability of the masseter muscle.

Materials and methods: Corticomotor excitability was assessed by changes in electromyographic activity evoked by TMS in 15 healthy participants. Motor evoked potentials (MEPs) recorded in the masseter and the first dorsal interosseos (FDI – as a control) muscle were assessed at four time points: at baseline, immediately after the 1-h training, 1 h after training and 1 day follow-up ($n = 7$). MEPs were assessed by stimulus–response curves and corticomotor mapping.

Results: All participants successfully performed the task (mean success rate: $47.0 \pm 4.1\%$) which increased significantly during the 1-h training. However, no significant effect of jaw muscle training on masseter and FDI MEPs or corticomotor maps were observed.

Conclusion: The present finding showed that 1-h jaw muscle training is insufficient to evoke neuroplastic changes in corticomotor excitability. The potential for training-induced neuroplasticity may vary among different cranial muscles which may have therapeutic consequences.

1. Introduction

The trigeminally innervated masticatory muscles are involved in voluntary biting, chewing, swallowing and speech. The motor cortex participates in the initiation and control of movements of the mandible through descending corticobulbar projections to the trigeminal motor nuclei and brainstem reticular formation.[1]

Jaw muscles are characterized by small motor unit territories with remarkable directional specificity of force generation; one jaw motor task could be achieved by different muscle co-activation strategies.[2] This is important when neuromuscular disorders necessitate a change in motor pattern if the same task is to be achieved.[3] It has been suggested that skilled motor training, but not strength training, results in cortical reorganization and the adaptation of the behaviour of motor units.[4–6]

* *Corresponding author at*: School of Stomatology, Capital Medical University, Dongcheng District, Beijing 100050, PR China. Tel.: +86 13810064564.
E-mail address: eflsy@hotmail.com (S. Lu).

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Transcranial magnetic stimulation (TMS) is a painless, non-invasive technique which has been widely used to assess the corticomotor projection to a number of target muscles.[7,8] Changes in responses to TMS during different voluntary tasks can reveal information about the involvement of corticobulbar neurons in the motor act[9] and the amplitude of the motor evoked potentials (MEP) can reflect the neuronal excitability in the motor cortex.[6] Several studies have, indeed, shown an expansion of the motor cortex maps associated with different types of task training.[10–14]

The corticotrigeminal projection from motor cortex (MI) to the pons near the trigeminal motor nuclei, is a relatively direct path for voluntary activation of jaw muscles and is likely to be important for fine voluntary control of bite force.[1,6] Corticomotoneuronal cells are thought to be important for fine control of individual muscles, and single motor unit studies have provided strong evidence for corticomotoneuronal cell projections to trigeminal motor nuclei in humans.[15]

The neuroplasticity evoked by task training may vary between muscle groups and be dependent on the specific applied motor task. Previous studies have revealed neuroplasticity in the cortical control of trunk muscles, tongue and masseter muscles. [11,12,16,17] However, the effects of a repeated submaximal biting task with fine voluntary control of the bite force have not been reported. The objective of this study was to determine if a 1-h jaw muscle training task would be sufficient to induce signs of neuroplastic changes in the corticomotor pathways related to the masseter muscle. The rationale for this duration and intensity of the jaw muscle training was that we have previously and consistently observed significant degrees of neuroplasticity in the corticomotor pathways related to the tongue muscles after low intensity, 1-h tongue protrusion training.[10,18,19]

2. Materials and methods

2.1. Participants

Seven male and eight female volunteers with a mean (±SEM) age of 22.5 ± 0.9 years were recruited by posting an advertisement on 'www.forsoegsperson.dk' webpage and at Aarhus University. The study protocol was approved by the Local Ethics Committee (Central Denmark Region, Denmark) and written informed consent was obtained from all participants. The inclusion criterion was good health with no orofacial pain complaints or temporomandibular disorders. Exclusion criteria included a history of neurological disease, implanted electronic devices and skull defects. The study was conducted in accordance with the Declaration of Helsinki.

2.2. Study design

This study consisted of four sessions: TMS and MEP recordings, maximum bite force and subject-based reports of evoked sensations (fatigue, pain) were assessed before 1-h jaw muscle training, immediately after completion of training, 1 h follow-up and 1 day follow-up (in only 7 out of the original 15 participants).[13]

2.3. Transcranial magnetic stimulation

TMS and MEP recordings and analysis have been described in detail[10] and will only be briefly described here. The participants were in a relaxed and supine position on a physiotherapy couch with the head supported by a headrest. Electromyography (EMG) (acquisition rate: 4 kHz) was recorded from the right masseter muscle by self-adhesive, bipolar electrodes (Neuroline 720, Ambu, DK) positioned parallel to the main direction of the muscle fibres over the lower anterior part of the main belly of the muscle determined by palpation, about 3 cm above and anterior to the mandibular angle.[20,21] EMG was also recorded, as an internal control, from the first dorsal interosseous (FDI) (muscle belly – caput metatarsale I).[13] The participants were grounded by a common electrode placed on the left hand. The EMG signals were amplified, filtered (10 Hz to 5 kHz), and stored on a Viking (Viasys Healthcare, Madison, WI, USA) for analysis. TMS was performed using a Magstim 200 stimulator (Magstim Company Limited, UK) and a focal Fig. 8 stimulating coil. A snugly fitting, flexible cap was placed over the head in a standardized way based on anatomical markers. The coil of the stimulator was oriented 45° oblique to the sagittal midline with the handle pointing posteriorly,[22] so that the induced current flowed in a plane perpendicular to the estimated alignment of the central sulcus. Three markings on the coil helped to identify the position in relation to the scalp sites.

The active motor threshold (AMT) for the masseter muscle was measured during a constant background contraction of the jaw-closing muscles (incisor biting on a 10 N predetermined biting device) because masseter MEPs cannot be recorded in the relaxed muscle.[12] In contrast, the motor threshold (MT) for FDI was measured in a relaxed state. Both types of MT were determined with the use of an ascending and descending method and were defined as the minimum stimulus intensity that produced five discrete MEPs clearly discernible from the background EMG activity.

Stimulus–response curves were constructed in steps of 10% of stimulator output, from the AMT/MT up to a maximum of 90% TMS output. Twelve stimuli were presented at each stimulus level with an inter-stimulus interval of 10–15 s. An example of the MEPs evoked in one of the subjects is shown in Fig. 2.

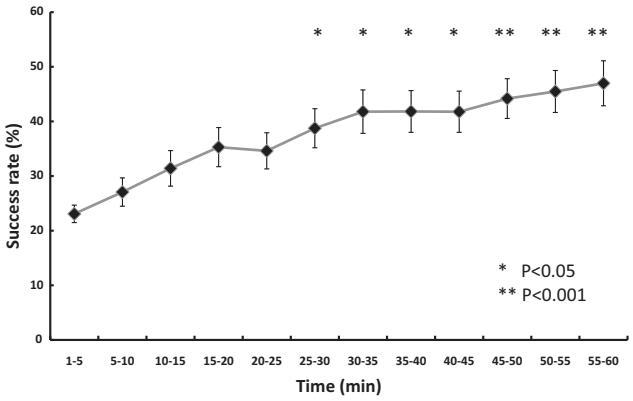

Fig. 1 – Mean (±SEM) success rate (%) during the 60 min training session. Success rate increased over time. */Significant difference from baseline ($P < 0.05/0.001$) ($n = 15$).**