O45-Modulation of cortical oscillatory suppression is associated with decreased activation of antagonist muscles in strength-trained athletes

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The adaptations induced by strength-training throughout all levels of the neuraxis increase efficiency of muscle contraction during force production task. This study investigated the modulations of cortical oscillations suppression associated with an "optimized" agonist-antagonist co-activation pattern. The electro-encephalogram, the electromyogram and the net joint torque were recorded while ten strength-trained athletes and eleven untrained participants performed an isometric force production task at different levels of their maximum voluntary contraction. Our results confirm the importance of the supraspinal level for regulation of antagonist muscles coactivation, and further suggest that the modulation of the central command observed in strength-trained athletes could convey a specific encoding of antagonist muscles activation, responsible for more efficient muscular contraction.
Keywords: motor cortex adaptations; agonist-antagonist co-activation; cortical oscillations; strength-training; submaximal isometric contractions

INTRODUCTION

Adaptations in response to strength-training contribute to increase efficiency of muscle contraction through a decrease of antagonist muscles activity (Tillin et al., 2011). At the supraspinal level, Falvo et al. (2010) showed that strength-training involves adaptations in term of motor-related evoked potentials recorded by electroencephalography (EEG). The present study compared the modulations of cortical oscillatory activity in strength-trained athletes (ST) and untrained participants (UT) during an isometric force production task, with the aim to investigate whether modulation of oscillatory cortical suppression could be associated with an "optimized" agonist-antagonist co-activation pattern. Given that voluntary contractions lead to the suppression of cortical oscillations ~ 20 Hz (Mima et al., 1999), and that activation of contralateral motor cortex is higher in high-skilled participants (Lotze et al., 2003), we hypothesized increased suppression of cortical oscillations in the ~ 20 Hz frequency band associated with decreased activation of antagonist muscles in strength-trained athletes.

METHODS

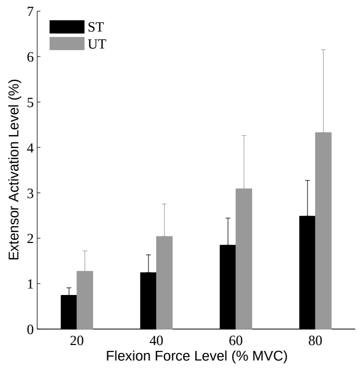
Ten ST and eleven UT males participated in this study. EEG was recorded using a 64-channel Biosemi Active Two system at 1024 Hz. Surface electromyography (EMG) was recorded at 1000 Hz with Delsys Bagnoli-8 from representative flexor and extensor knee muscles. The net knee joint moment was measured by a BIODEX S4 Pro dynamometer at 1000 Hz. Participants performed a total of 20 knee isometric flexion contractions from 20 to 80 % of their previously determined maximum voluntary contraction (MVC), in steps of 20 %. The EEG task-related modulations of cortical oscillations of the Cz electrode were computed using Morlet-wavelet time-frequency transformation in the ~ 20 Hz frequency band. For each muscle, the EMG activation level was computed as the mean of the EMG linear envelop normalized to the EMG maximal value obtained from MVC contractions. A two factors repeated measures ANOVA (*Force level* × *Expertise*) was carried out ($p < .05$). Linear regression was tested between the EEG task-related cortical oscillations and EMG activation level of extensor and flexor muscles.

RESULTS

ST and UT produced similar MVC values (110.1 ± 28.5 and 108.3 ± 24.7 Nm, respectively; $t_{19} = 0.16$; $p > .05$). Statistical analyses showed a *Force level* effect ($F_{1,19} = 9.79$; $p < .05$), an *Expertise* effect ($F_{3,57} = 83.10$; $p < .05$) and a *Force level* × *Expertise* interaction ($F_{3.57} = 6.32$; $p < .05$) on the level of EMG activity in antagonist muscles (Fig. 1). A *Force level* effect ($F_{3.57} = 3.35$; $p < .05$) and a *Force level* × *Expertise* interaction ($F_{3.57} = 3.67$; $p < .05$) were also found on the ~ 20 Hz cortical oscillations suppression (Fig 2). A significant correlation was found between ~ 20 Hz cortical oscillations suppression and the level of EMG activity of antagonist muscles in ST (r = -0.51; p < .05) and UT (r = 0.3; p < .05).

Figure 1 Antagonist EMG activation at each force level in ST and UT (mean □ std)

Figure 2~ 20 Hz EEG oscillations suppression at each force level in ST and UT (mean □ std)



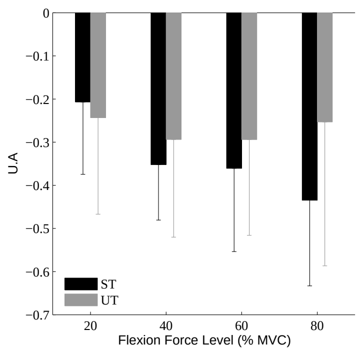


Fig. 1: Antagonist EMG activation at each force level in ST and UT (mean □ std)
Fig. 2: ~ 20 Hz EEG oscillations suppression at each force level in ST and UT (mean □ std)

DISCUSSION

Despite ST and UT produced equivalent MVC values, probably due to the aspecificity of the experimental task for ST, our results confirm previous findings that strength-training involves lower antagonist muscle activation (Tillin et al., 2011), leading to a form of energetic optimization of muscular contraction. The linear correlation between cortical oscillatory suppression and EMG activity of antagonist muscles confirms the important role of supraspinal control for regulation of antagonist muscles coactivation in both ST and UT. However, the suppression of ~ 20 Hz cortical oscillations with increase in *Force Level* revealed specific effects of strength-training. Considering that increase in motor cortex activation could reflect the control of muscles other than the prime mover muscles (Dettmers et al., 1995), our findings suggest that the central command could exert a specific encoding of antagonist muscles activation in ST, responsible for more efficient muscular contraction.

REFERENCES

Dettmers, C., Fink, G.R., Lemon, R.N., Stephan, K.M., Passingham, R.E., Silbersweig, D., Holmes, A., Ridding, M.C., Brooks, D.J., Frackowiak, R.S. (1995). *J. Neurophysiol., 2,* 802-815.

Falvo, M.J., Sirevaag, E.J., Rohrbaugh, J.W., Earhart, G.M. (2010). *Eur. J. Appl. Physiol., 109,* 923-33.

Lotze, M., Scheler, G., Tan, H.R.M., Braun, C., Birbaumer, N. (2003). *NeuroImage, 20*, 1817-1829.

Mima, T., Simpkins, N., Oluwatimilehin, T., Hallett, M., (1999). *Neurosci. Lett.*, 275, 77-80.

Tillin, N.A., Pain, M.T., Folland, J.P. (2011). *Muscle Nerve*, 43, 375-84.