Title:
The role of the autonomic nervous system in the development of exercise induced muscle damage: results of a RCT

Authors:
Johannes Fleckenstein1,2, Philipp Bormuth1, Fabio Comes1, Winfried Banzer1
1Department of Sports Medicine, Institute of Sports Science, Goethe-University Frankfurt, Germany
*Department of TCM/Acupuncture, Institute of Complementary Medicine, University of Bern, Switzerland

Abstract:
Introduction: Experimentally induced muscle damage (EIMD) is a common model in sports medicine, causing dysfunction as well as pain. Whereas primary dull pain is often referred to as delayed-onset muscle soreness (DOMS), the appearance of severe dysfunction and deep tissue pain refers to an increased trauma on a cellular and fascial level [1]. The sympathetic part of the autonomic nervous system has particularly been associated to induce neurogenic inflammation via vasodilation and plasma extravasation and the release of pro-inflammatory neuropeptides from its own nerve fibers [2,3]). Thus we assume, the sympathetic nerves to be a key mechanism mediating the late phase of EIMD.

Methods: Randomised controlled study to detect effects of a blockage of the sympathetic stellate ganglion (4-5 ml procaine 1%, [3,4]) compared to a non-invasive sham treatment on experimentally induced EIMD of the non-dominant arm in healthy participants over 96 hours. Main outcome was the detection of the pressure pain threshold (PPT), secondary outcomes included the mechanical pain threshold (MPT), pain intensity (VAS), and the maximum isometric voluntary force (MIVF).

Results: Fifteen out of 30 subjects (25.1±7.8 years, BMI 23.2±2.2 kg/m2) received stellatum blockage. The induction of EIMD caused a decrease in PPT and MIVF, and an increase in VAS in the control group. TimeXgroup analysis (4x2) revealed significant differences of the PPT at the distant belly of the biceps muscle (p = 0.034), as well as perceived pain intensity (p = 0.048) favouring the intervention group. Post-hoc test indicated a trend that the changes occur in the late phase of the EIMD (48-96 h; p < 0.01).

Discussion: Data suggest that a reduced sympathetic tone (i.e. intervention) reduces the risk of high threshold mechanoreceptor sensitisation (as assessed by PPT), which is the predominant nociceptor of the myofascial unit [1]. We could not detect changes of superficial nociceptors indicating irritations of superficial connective tissues. We hypothesize that sports related microtrauma are mainly limited to the myofascial unit, and that the extent of damage is mediated by sympathetically induced neurogenic inflammation. To what extent hands-on fascial techniques are able to reduce the level of sympathetic activation should be subject to future studies.

References: