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LETTER TO THE EDITOR

Motor unit recruitment cannot be inferred from surface EMG amplitude and basic reporting standards must be adhered to

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Abstract The study by Jenkins et al. attempted to elucidate the mechanisms behind the findings of Mitchell et al. (J Appl Physiol 113(1):71–77, 2012). However, we believe the work of Jenkins et al. contains methodological issues, does not meet electromyographic reporting standards, and deduces conclusions beyond which can be interpreted from the data provided.

Keywords Electromyography · Motor unit recruitment

Abbreviations

EMG	Electromyography
iEMG	Integrated electromyography
MPF	Mean power frequency
MU	Motor unit

Dear Editor,

We read with great interest the study by Jenkins et al. (2015), which attempted to better understand the mechanisms associated with the quadriceps hypertrophy observed by Mitchell et al. (2012). Unfortunately, several measures have been misreported and the conclusions drawn by the authors are, in our view, unsubstantiated.

Surface electromyography (EMG) measures the electrical potential of many elements that are often thought to be representative of motor unit (MU) recruitment, rate coding, and possibly synchronization. However, multiple other peripheral constituents—that is, muscle fiber propagation velocity and intracellular action potentials—are also included in the signal. As the EMG signal represents electrical potential, its units are to be presented in the form of volts, viz. micro- or millivolts (μV or mV). In addition, integrated EMG (iEMG) is the area under the EMG–time curve (usually full-wave rectified), and should thus be reported as μV·s or mV·s. Jenkins et al. (2015) reported the standard error of measurement of EMG and iEMG using μV/s and μV, respectively. These units are incorrect, as the former represents the rate of change of EMG amplitude and the latter is simply the unit for EMG amplitude. In addition, they are different from the units in which the data are presented (normalized), which make the reliability of the presented data difficult to interpret. However, the coefficients of variation of ~20 % for the EMG amplitudes are indicative that, when both the amplitude in the exercise and normalization amplitude are combined, the error has the potential to be quite large. A more compendious method of presenting the reliability data would be to present it in terms of how the results are presented; that is, normalized. The authors' failure to comply with basic reporting standards greatly increases the chances of misleading readers.

In EMG studies that require the reapplication of electrodes, normalization is a technique often utilized. In the study by Jenkins et al. (2015), participants visited the laboratory four times. The first two times consisted of maximum voluntary isometric contraction (MVIC) testing; the second of which was utilized as the denominator by which the signals on visits 3 and 4 (low- and high-load trials) were divided for normalization. By moving or reapplying an electrode, one risks changing the spatial filtering characteristics between active fibers and detecting different innervation zones, which will then be reflected by the

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EMG signal. Thus, normalization needs to be repeated when electrodes are reapplied. The normalization procedure employed by Jenkins et al. (2015) is the mathematical equivalent of comparing raw signals between days, as the same number served as the denominator for both the low- and high-load trials, from which conclusions cannot be drawn for the aforementioned reasons.

In their discussion, Jenkins et al. (2015) note that their data are not congruent with the thesis put forward by Mitchell et al. (2012), which stated that relatively lighter loads lifted to muscular fatigue may induce similar muscle fiber activation. This disagreement stems from Jenkins and colleagues' misinterpretation of the EMG amplitude measured in their study, as 80 % 1RM elicited greater EMG amplitude than the 30 % condition. Specifically, Jenkins et al. (2015) are inferring that EMG amplitude directly represents MU recruitment. The fallacy of deducing MU recruitment from EMG amplitude during fatiguing contractions has been described in a number of papers by prominent EMG researchers. Furthermore, the conclusions of Jenkins et al. (2015) ignore the contribution of MU cycling, wherein fatigued MUs may be momentarily de-recruited to reduce fatigue. Accordingly, in fatiguing conditions that require less force output, such as the low-load conditions utilized by Jenkins et al. (2015), the full spectrum of MUs may ultimately be recruited, albeit not simultaneously. In order to truly measure MU recruitment, more advanced methods are needed, such as spike-triggered averaging or initial wavelet analysis followed by principal component classification of major frequency properties and optimization to tune wavelets to these frequencies.

At present, there is no such evidence to support that greater simultaneous activation of the MU pool confers any hypertrophic advantage over conditions that recruit a comparable complement of the MU population, albeit with less cumulative activity at any one point in time. Although a number of studies have now demonstrated greater EMG amplitude with high- versus low-load training, the data from Mitchell et al. (2012) demonstrate comparable growth of both type I and II fibers between conditions, and these findings are congruent with other studies. If anything, this leads to the conclusion that consideration of the differential in EMG amplitude between high- and low-loads confers little information regarding the subsequent hypertrophic response to training. We appreciate that the authors have acknowledged that, in the future, other experimental techniques will be required to better address any differential muscle fiber response to varying training intensities.

Jenkins et al. (2015) utilized EMG mean power frequency (MPF) in attempt to surmise conclusions regarding the biochemical environment during both the low- and high-load trials. As a greater decrease in MPF was observed in the low-load condition, it was presumed that intramuscular pH decreased, inorganic phosphate (P_i) levels increased, and sarcolemmal ion gradients were altered to a greater extent than with high-load training. Alterations in the EMG power spectrum have been attributed to changes in action potential conduction velocity, action potential shape, and muscle relaxation rates, which have been attributed to the aforementioned biochemical changes. However, direct experimental evidence associating MPF with these biochemical outcomes is equivocal. The most relevant study, which examined the vastus lateralis during dynamic contractions, found no association between intramuscular pH and MPF (Bouissou et al. 1989). Thus, MPF in these conditions represents an ambiguous measure and is subject to differing interpretation.

We would like to thank Jenkins et al. (2015) for their contribution to the literature and eagerness to understand the mechanisms behind the results of Mitchell et al. (2012). Notwithstanding, it is of the utmost importance to adhere to proper reporting standards and not attach mechanisms to EMG signals beyond what is reasonably acknowledged to be interpretable from such data.

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