



Longing for a Longitudinal Proxy: Acutely Measured Surface EMG Amplitude is not a Validated Predictor of Muscle Hypertrophy

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Abstract

Surface electromyography amplitudes are commonly measured in acute sports and exercise science studies to make inferences about muscular strength, performance, and hypertrophic adaptations that may result from different exercises or exercise-related variables. Here, we discuss the presumptive logic and assumptions underlying these inferences, focusing on hypertrophic adaptations for simplicity's sake. We present counter-evidence for each of its premises and discuss evidence both for and against the logical conclusion. Given the limited evidence validating the amplitude of surface electromyograms as a predictor of longitudinal hypertrophic adaptations, coupled with its weak mechanistic foundation, we suggest that acute comparative studies that wish to assess stimulus potency be met with scrutiny.

1 Background

In view of the growing popularity of surface electromyography (sEMG) studies in sports science, we voiced our concerns in our 2018 review paper on sEMG's use and misuse [1]. Since its publication 4 years ago, the paper has garnered over 200 citations, indicating that it has attracted much attention and the field may indeed be receptive to our concerns. However, over time we learned that many of these citations treated our concerns as minor, inconsequential limitations of sEMG amplitudes rather than serious flaws that undermine authors' conclusions. In retrospect, this may not be overly surprising as, admittedly, parts of the review were technically dense and required background knowledge of neurophysiology. Given these mis-citations and the continued,

pervasive extrapolations of sEMG amplitudes,¹ we wish to clarify and simplify our primary concerns regarding the interpretation of sEMG amplitudes in applied sports science studies, which are complementary to previous critical reviews on sEMG's use and misuse.

To achieve this goal, in this commentary, we solely focus on one study design which is especially popular and problematic in sports science: using sEMG amplitudes from acute studies to make inferences about longitudinal adaptations, primarily concerning hypertrophy. Studies of this type are ubiquitous and easy to identify. They commonly involve placing surface electrodes on muscles of interest and comparing the sEMG amplitudes that result from two or more exercise variations, loading schemes, or some other exercise-related variable (e.g., exercise order) performed within a single or over a few sessions (i.e., without performing a longitudinal study). For example, researchers may place electrodes on the biceps brachii and have participants perform dumbbell preacher curls and dumbbell incline curls during a single session. Authors then make exercise recommendations based on their findings, indeliberately attributing predictive power to the acutely observed differences in sEMG amplitude; for instance, higher sEMG amplitudes observed during one of the exercises implies that the exercise will elicit greater hypertrophy in the long term. We note that other uses of sEMG, such as investigating coordination and timing of

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¹ By sEMG amplitudes, we refer to statistics summarizing the average degree of variation of the amplitude of the raw sEMG over a given period into a single value, with the most popular estimate being the root mean square amplitude.

Key Points

Researchers commonly make exercise recommendations based on acute surface electromyography (sEMG) studies, under the assumption that exercises that elicit the greatest amplitudes will also confer the most favorable long-term adaptations.

We break down the logic behind this assumption as it relates to muscle hypertrophy into three premises and a conclusion. None of the three premises can be heavily relied upon, rendering the conclusion to be logically tenuous.

Researchers have not attempted to assess the ability of acutely measured sEMG amplitudes to predict long-term hypertrophic adaptations. Thus, both the predictive validity and its boundary conditions remain unclear.

Acutely measured sEMG amplitude has not been validated as a predictor of hypertrophy (and other long-term outcomes), and the predictive validity stands on weak theoretical grounds. As such, acute comparative exercise studies attributing predictive power to sEMG amplitude deserve greater scrutiny.

muscle excitation, are unrelated to our discussion and thus will not be covered, nor will we discuss what information is contained in the sEMG signal. For information regarding the latter, we direct readers to reviews and texts on the topic (see Table 2 in [1]). Instead, this article aims to break down the assertion that sEMG amplitudes can predict hypertrophic adaptations into three premises and a conclusion. Importantly, all three premises that we will cover are necessary to conclude that sEMG is a valid predictor of hypertrophy. Yet, only one is dependent on sEMG amplitudes. We argue that all three premises are weak, rendering the conclusion that sEMG amplitudes can be used to predict hypertrophic adaptations tenuous.

Definitions Box 1

In this commentary, we simplify some of the jargon commonly used in this research area. Although some of these terms require knowledge of muscle physiology, a deep understanding of them is not required to grasp the key message of this article.^a

- **Neuromuscular excitation**—the electrical signal that causes calcium to be released into the sarcoplasm to enable activation. This includes the depolarization of the α -motoneuron (neural excitation) and subsequent depolarization of the sarcolemma (muscle excitation). Neural excitation may vary in terms of the number of α -motoneurons excited (motor unit recruitment) and their discharge rate (rate coding), while muscle excitation is affected by changes in the peripheral muscle environment (e.g., $[Ca^{2+}]$ changes).

- **Muscle activation**—relative binding of calcium to troponin to facilitate cross-bridge attachment.
- **Cross-bridge attachment**—myosin binding to actin to produce active muscle force.
- **Muscle state changes**—muscle activation and subsequent cross-bridge attachment.

^aWe lump ‘neural excitation’ and ‘muscle excitation’ into ‘neuromuscular excitation’, which is the electrochemical phase. Similarly, we sometimes lump ‘muscle activation’ and ‘cross-bridge attachment’ into ‘muscle state changes’, which is the chemomechanical phase. This lumping is justified because the former in each is commonly—though not always—the rate-limiting step [2], and thus, the relationships are close to one-to-one for the purposes of this commentary.

2 The Logic of sEMG as a Predictor of Hypertrophic Adaptations

Similar to our 2018 review paper [1], here, we will briefly cover both mechanistic and applied interpretations of sEMG amplitudes. Although seemingly unrelated, these interpretations are closely linked—the latter follows directly from the former. That is, inferences that greater sEMG amplitudes are associated with more favorable hypertrophic adaptations stem from various mechanistic assumptions. The full, often implicit, logical argument is presented below and in Fig. 1.

1. sEMG amplitudes reflect the neuromuscular excitation of the target muscle(s).
 2. Neuromuscular excitation is directly related to the state of the muscle.
 3. Changes in muscle state drive hypertrophic adaptations.
- ∴ Therefore, sEMG amplitude is predictive of hypertrophic adaptations.

2.1 Premise 1: sEMG Amplitudes Reflect the Neuromuscular Excitation of the Target Muscle(s)

A common assumption among sport and exercise scientists is that sEMG amplitudes are solely a reflection of neuromuscular excitation. However, a growing number of human, animal, and modeling studies have shown that sEMG amplitudes and neuromuscular excitation can be uncoupled. For instance, by simply plantar flexing the ankle, Vieira et al. [3] observed a marked decrease in tibialis anterior sEMG amplitude induced by nerve stimulation (*M*-wave).² This indicates that changing a muscle’s shape or architecture (i.e., length and pennation angle) will drastically affect the sEMG signal, even when neuromuscular excitation is identical. The biophysical origins of this effect are fairly well

² Similar findings have been reported in other studies and muscles [4–11].

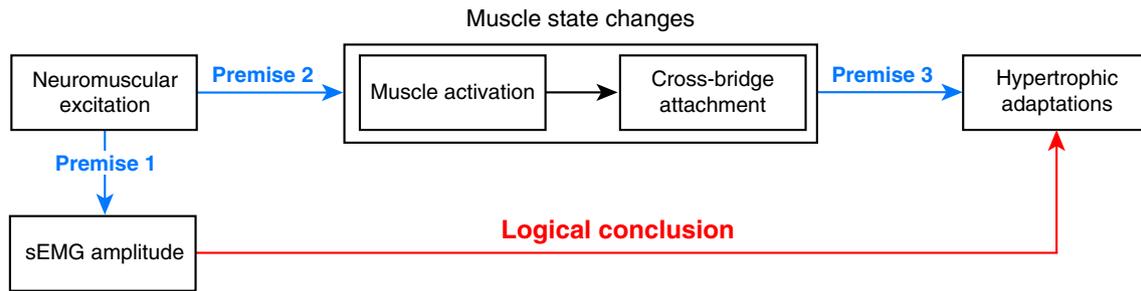


Fig. 1 Mechanistic logic for using surface electromyography (sEMG) as a predictor of hypertrophic adaptations. sEMG serves as a proxy for neuromuscular excitation. Neuromuscular excitation induces muscle state changes, which are said to drive hypertrophic adaptations. This logic implies that acutely measured differences in sEMG ampli-

understood. In particular, the muscle architecture-dependence of the sEMG signal is a consequence of how the action potentials travel through the muscle relative to the positions of the electrodes [12]. Similarly, electrode configurations, subcutaneous thickness, muscle lengths, and contraction modes will bias the surface electromyogram, resulting in an under- or over-representation of the electrical signal from the target muscle(s) that may vary throughout the range of motion [13–19].

These inconsistencies challenge the premise that sEMG amplitudes collected across different exercises genuinely reflect differences in neuromuscular excitation. Although normalizing signals to a single maximal value (e.g., maximum voluntary contraction, *M*-wave) is unlikely to attenuate this effect, the benefits of position-specific normalization remain to be investigated. Indeed, the excitatory origins of sEMG amplitude changes are clearer in well-controlled conditions but can impose an inferential barrier whenever changes in muscle architecture, length, or force are likely to take place [17].

2.2 Premise 2: Neuromuscular Excitation is Directly Related to the State of the Muscle

Neuromuscular excitation is the electrical signal that causes calcium to be released into the sarcoplasm to enable activation. After calcium is released, the state of the muscle is altered by calcium binding to troponin (activation) to enable cross-bridging (attachment), which together determine the muscle’s state [20]. The distinction between neuromuscular excitation and a muscle’s state is subtle but meaningful, as the two can be instantaneously decoupled by exploiting their dynamics.³ This challenges the premise that neuromuscular

³ Changing a muscle’s state over time (e.g., dynamic contractions or changes in force) will complicate the otherwise straightforward neuromuscular excitation-muscle state relationship [2, 21]. Although this process can be modeled, it is typically not in applied sEMG studies in sports science.

tudes can predict hypertrophic adaptations. For this logical conclusion to be true, either (a) all premises must strongly hold; (b) biases or poor relationships in one premise must be “canceled out” by the other premises; or (c) there is some other pathway. *Prima facie*, we consider (b) and (c) to be highly unlikely, so we focus on (a)

excitation provides direct insight into a muscle’s state. However, Premise 2 can hold in study designs that include non-fatiguing, isotonic, isometric contractions, as one can assume a linear and stable relationship between neuromuscular excitation and a muscle’s state.

2.3 Premise 3: Muscle State Changes Drive Hypertrophic Adaptations

The logic that a muscle’s state begets its hypertrophy assumes that (a) changes in muscle state induce muscle protein synthesis (MPS), and (b) MPS triggers a hypertrophic response. It is undoubtedly the case that resistance exercise triggers robust MPS and hypertrophic responses relative to rest [22]. Unfortunately, comparisons to rest tell us little about the muscle state-hypertrophy dose-response relationship.

The relationship between muscle state changes and MPS remains unclear, and the best available evidence suggests it is weak. Morton et al. [23] found that glycogen depletion following exercise, a marker of activation, was only weakly associated with anabolic markers (i.e., mTOR, p70 S6k, etc.; not MPS).⁴ Although the investigators did not study MPS directly, the weak relationship observed between muscle state changes and anabolic markers indicates that the muscle state-MPS relationship may indeed be tenuous.

In contrast to the muscle state–MPS relationship, the link between MPS and hypertrophy is mixed and remains to be fully elucidated. Early work suggests little-to-no correlation between MPS and hypertrophy [25], but more recent work

⁴ Morton et al. [23] also demonstrate that sEMG amplitudes can be decoupled from (a) muscle state changes, as measured via muscle glycogen depletion, which spans Premises 1 and 2, and (b) anabolic signaling, which spans Premises 1–3. However, the authors employed between- rather than within-subject correlations. Here, we think the more relevant question is the within-subject relationship (see [24]), which may be a fruitful avenue for future research.

that accounts for some methodological shortcomings suggests much stronger correlations ($r > 0.9$) [26]. Although debates are ongoing [22], in some contexts, the jump from MPS to hypertrophy may indeed be a reasonable one.⁵

More broadly, the relationship between muscle state changes and growth—spanning both muscle state to MPS and MPS to growth, detailed separately above—is inconsistent across the literature. For example, changes in the muscle state are not necessary for muscle growth, as indicated by studies that have observed growth following stretch protocols in which changes in muscle state did not occur [28]. Evidently, the activation-hypertrophy relationship is not straightforward. The dearth of a clear-cut relationship between muscle state changes and muscle growth undermines Premise 3.

Finally, even if the above premises held, the predictive value of sEMG would still need to be directly validated.

2.4 Logical Conclusion: sEMG Amplitude is Predictive of Hypertrophic Adaptations

The mechanistic rationale for sEMG being a predictor of hypertrophy is attractive. Still, as we briefly discussed in the previous sections, there are theoretical concerns that may yield the relationship to be tenuous. The assertion that sEMG has predictive validity is an example of a slippery slope argument, the strength of which depends on two factors. As eloquently stated by philosopher David Kelley, “The first is the strength of each link in the causal chain; the argument cannot be stronger than its weakest link. The second is the number of links; the more links there are, the more likely it is that other factors could alter the consequences” (p. 123, [29]). Given both the number of links (premises) and their unstable mechanistic foundations, predictive validity arguments for sEMG amplitude are poor. One cannot simply assume that a clear relationship between sEMG amplitude and longitudinal outcomes exists.

Indirect evidence indicates inconsistent relationships between sEMG amplitudes and growth. On one hand, evidence in favor of the sEMG amplitude-hypertrophy relationship is that sEMG amplitudes tend to correspond with growth in the rectus femoris [30–33] and hamstrings [34–37]. For example, multi-joint movements yield low sEMG amplitudes and little growth in the rectus femoris, whereas single-joint movements yield high sEMG amplitudes and marked growth [30–33]. However, one could argue that these results are expected based on biomechanics, meaning there was no clinical equipoise and the sEMG results

did not provide novel insight. On the other hand, sEMG amplitudes were not clearly indicative of growth when comparing different contraction modes [15, 38], fatiguing conditions [23, 39–45], and muscle lengths [4, 46–57]. For example, maximum isometric voluntary contractions across a range of knee joint angles produce discordant quadriceps sEMG amplitudes [4, 46–53]. However, greater hypertrophy is commonly observed when the quadriceps are isometrically trained at longer compared to shorter muscle lengths [54–57]. Perhaps the largest discrepancy between sEMG amplitudes and hypertrophy is in the high- versus low-load resistance training literature. When sets are performed to momentary failure, high- and low-loads produce similar growth [39–41] despite high-loads eliciting greater sEMG amplitudes throughout the entire duration of a set [23, 42–45, 58].

These examples show that indirect evidence is mixed regarding the link between sEMG amplitude and hypertrophic responses. Yet, no study has directly investigated and quantified the association between sEMG amplitude and longitudinal outcomes.⁶ In quantifying this relationship, several ancillary questions will be answered, including the sensitivity of sEMG amplitude as a predictor of hypertrophy. For example, when comparing two exercises, how much greater of a hypertrophic response can we expect from a 10% greater sEMG amplitude? Moderators of this relationship would also be of interest, including the effects of fatigue, contraction mode, and muscle being studied. However, predictive validity does not begin and end with associations.

Establishing predictive validity is a grueling process. To do so, investigators must explicitly demonstrate that any observed relationship is predictive and generalizable to other populations and environments [63]. For sEMG amplitude to be validated and accepted as a predictor of longitudinal adaptations, it would need to be both associated with and predictive of the outcomes of interest (hypertrophy). Although the specification of research designs to address this gap is beyond the scope of this paper, we stress that without such work, it is unacceptable to treat sEMG amplitude as a validated predictor of longitudinal adaptations like hypertrophy.

⁵ For a more complete and nuanced detailing of the MPS-hypertrophy relationship, we direct readers to the recent review by Witard et al. [27].

⁶ We also acknowledge the T_2 -weighted magnetic resonance imaging (MRI) and regional hypertrophy work by Wakahara et al. [59–61] in the triceps brachii and quadriceps and Illera-Dominguez et al. [62] in the hamstrings, quadriceps, and adductors. Despite their mixed findings, there are two caveats to note. Principally, this work used MRI rather than sEMG; to our knowledge, MRI has not been validated against sEMG for intramuscular heterogeneity. Secondly, these studies did not model within-subject relationships directly.

3 Take-Home Points

In light of the aforementioned limitations, the production of comparative studies and reviews on acute sEMG amplitudes, with the implicit or explicit purpose of inferring longitudinal adaptations, should be greatly attenuated. The sheer number of these acute comparative sEMG amplitude studies are increasing at what seems to be an exponential rate, without complementary longitudinal studies. This is problematic: If one exercise leads to greater sEMG amplitude than another, the longitudinal implications of such findings are assumed, not evidenced. In the case of hypertrophy, these assumptions are largely unsupported, and one can apply similar scrutiny to other longitudinal adaptations such as strength. These acute sEMG amplitude studies intended to inform exercise selection without measuring longitudinal adaptations use precious resources, including the time of the researchers, participants, peer-reviewers, editors, and readers, in addition to the costs involved in conducting research.

To be clear,

- At present, acute studies that compare sEMG amplitudes between, for example, two different exercise protocols cannot be used as evidence for longitudinal adaptations (e.g., hypertrophy);
- Acute sEMG amplitude studies may be indicative of longitudinal adaptations, but evidence supporting this assertion is lacking. Arguments that contend otherwise are based on tenuous assumptions and are appeals to ignorance;
- Instead of acute comparative sEMG amplitude studies, investigators should focus on longitudinal outcomes of interest (e.g., measures of hypertrophy);
- Despite these limitations, sEMG is a useful tool for specific research questions, such as those related to coordination (e.g., timing of muscle excitation).

4 Conclusion

We contend that continued submissions and publications of acute comparative sEMG amplitude studies with inferences about longitudinal adaptations are flooding the literature. To help remedy this, we propose that journal editors keep the above points in mind before sending these papers out for review. Reviewers who receive such papers should be mindful of the limitations we detailed. Unless authors communicate the value of their study independently of longitudinal outcomes and beyond that expected from basic biomechanics or functional anatomy, readers should critically appraise their findings and implications.

Declarations

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Consent for publication Not applicable.

Availability of data and material Not applicable.

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Author's contributions ADV and IH devised the paper and wrote the first draft. ADV, IH, GST, and TMV edited the manuscript for content and clarity and approved the final version for publication.

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