Muscle size and strength : debunking the “completely separate phenomena” suggestion

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12-April-2017:

Dear Editors in Chief Westerterp/Westerblad,

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1. Clarifying that we are responding to a letter to the editor (i.e Buckner et al., 2017).
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Regards

Thomas G. Balshaw

TITLE PAGE

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Muscle size and strength: debunking the "completely separate phenomena" suggestion

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Dear Editor,

Buckner et al. (2017) query the conclusion of our recent article ("Changes in agonist neural drive, hypertrophy and pre-training strength all contribute to the individual strength gains after resistance training," Balshaw et al. (2017)), which stated that "muscle hypertrophy in the current study…clearly did contribute to the explained variance in strength and further negates the suggestion that strength and hypertrophy are entirely separate phenomena." and also refuted a hypothesis of theirs that the changes in muscle size and strength after resistance training (RT) are "separate and unrelated adaptations" (Buckner et al., 2016). Here we emphasise that: (a) the aim of our study was to investigate whether there was a relationship between putative predictor variables/adaptations after RT, rather than the nature of the relationship (e.g. causal/coincidental); (b) the existence of the relationship we found undermines the hypothesis of Buckner et al. (2016); and (c) present theory and additional evidence that refutes their hypothesis.

The strength changes after RT observed in our study were correlated with hypertrophy (r=0.461), as well as the changes in agonist neural drive (r=0.576) and pre-training strength (r=-0.429). Subsequently, hypertrophy contributed 19% towards a total of 60% explained variance in strength changes within multiple regression analysis (Balshaw et al. 2017). In addition, our previous upper body study also found hypertrophy to explain 19-23% of the variance in strength gains after RT (Erskine et al. 2014). Thus, hypertrophy and strength gains are not "entirely separate phenomena" or "separate and unrelated adaptations," as we have demonstrated they are consistently and systematically related. Nonetheless, we agree with Buckner et al. (2017) that this evidence alone does not demonstrate causality as these experiments were not designed to address causality.

Experimentally, the first step in understanding the interaction of two variables is to investigate if they are related, and second consider the nature of the relationship (coincidental or causal). Our paper addressed this first step as there are several reasons why this relationship has until recently been somewhat opaque:

(i) It is widely accepted that in humans muscular strength and size are not perfectly coupled (e.g. in cross-sectional MRI studies 38-89% of the variance in isometric strength, across various muscle groups, is explained by muscle size indices), as a number of other factors are considered to also influence strength (neural drive, muscle architecture/composition, moment arm etc.), and this may set an upper limit on the strength of the hypertrophy-strength gain relationship after RT.

(ii) Most RT studies have produced limited hypertrophy (typically <12%) due to their short duration (≤3 months) and lower body RT (less hypertrophy than upper body muscles). Limited hypertrophy has likely restricted the capability of previous research to delineate the hypertrophy-strength gain relationship, and placed a greater emphasis on other adaptations (e.g. neural). One exception was our previous elbow flexor RT study, that found muscle volume to increase by 16% which probably explains why this study found the strongest hypertrophy-strength gain relationship reported to date (r=0.482-0.527; Erskine et al., 2014).

(iii) Measurement issues have clouded the hypertrophy-strength gain relationship after RT:

- Poor muscle size measurements, including low resolution imaging techniques (e.g. ultrasound) and single site/slice measurements, as opposed to multiple slice high-resolution MRI.
- Strength measurements on one occasion, at each time point, that are subject to day-to-day variation and measurement error.
- Additive effect of noise/error when combining measurements at two time points i.e. change data.
- Studies with small participant numbers (<20) for determining relationship.

These experimental issues and the limited evidence for an individual relationship between hypertrophy and strength gains appear to have led Buckner et al. (2016) to the spurious hypothesis that hypertrophy and strength gains could be "separate and unrelated adaptations". In contrast we believe that our recent papers are the first consistent demonstration of a relationship between hypertrophy and strength changes on an individual basis after RT. Moreover, we hypothesise that the strength of this relationship is dependent on the magnitude of hypertrophy and thus training duration, as longer-term RT produces greater hypertrophy (Narici et al. 1996), and a progressively stronger relationship might be expected as RT duration increases, although this has yet to be tested. Finally, given our consistent evidence for a relationship between strength gains and hypertrophy it now appears timely to examine the nature (e.g. causality) of the relationship using a different approach to that taken in our previous experiments.

However, theoretically and empirically we consider Buckner et al.'s hypothesis to be highly implausible. First, in isolated animal muscle physiological cross-sectional area (PCSA) explains >95% of the variance in maximum force (Powell et al. 1984) and thus it has been widely accepted for decades that a muscle's

force generating capacity is proportional to its size, and specifically PCSA which reflects the number of sarcomeres/myosin-actin cross-bridges arranged in parallel and thus able to generate tension between the tendons. Human muscle working in-vivo is clearly a more complex situation (see (i) above), that explains the lower, yet still substantial, size-strength relationship in cross-sectional human studies (R^2=0.38-0.89). This means that in humans muscle size does not perfectly predict strength on an individual basis, rather a given muscle size may define an 'envelope' of strength expression that is regulated by other factors (e.g. neural, biomechanical etc.). Second, whereas the hypertrophy-strength gain relationship has been confounded by the experimental issues (outlined above), concurrent group level changes in hypertrophy and strength have been documented to occur following diverse processes/interventions: growth/maturation, ageing, immobilisation, testosterone administration, as well as RT. These simultaneous increases/decreases in strength and size following diverse stimuli provide strong evidence that hypertrophy and strength are in fact broadly coupled.

In conclusion, we consider the underlying influence of muscle size and thus hypertrophy on strength in healthy muscle to be based on extensive robust evidence and logic, that is most likely underpinned by a causal relationship, although we accept that in the context of RT this has yet to be categorically demonstrated. In contrast, Buckner et al.'s hypothesis appears to deny knowledge/appreciation of theory and evidence and in our view is untenable.

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