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LETTER

The dose–response relationship between resistance training volume and muscle hypertrophy: are there really still any doubts?

We read with interest the letter to the editor entitled “Is it possible to reach reliable conclusions doing meta-analyses with resistance training studies?” (Arruda et al., 2016). We thank the journal for giving us an opportunity to respond to the points raised by the authors.

First, Arruda et al. (2016) stated that meta-analyses “usually combine studies that have important methodological differences” and that “the summary effect can be largely influenced by these differences across studies”. They refer Field (Field, 2015) in this statement. The authors fail to note that, in this context, Field was discussing the use of meta-analysis to aggregate studies with not only different designs but also different questions (such as pooling studies that examine at cognitive behavioural therapy, psychodynamic therapy, etc.). Our analysis was isolated to studies that manipulated the number of sets to examine the impact of set volume on hypertrophy. It should also be noted that Field discussed the use of multilevel models to improve on this limitation of meta-analyses, which is exactly the type of model we used in our analysis. In fact, the model we used is highly conservative, and is ideal for aggregating studies with marked heterogeneity. Our statistical model assumes that studies are heterogeneous and that we cannot account for all possible sources of variance between studies.

Second, Arruda et al. (2016) discussed how “resistance training outcomes are largely influenced by many variables that interact with each other”, and “trying to estimate the impact of one variable when others are not controlled has a high risk of bias”. However, other variables were controlled in each individual study, as we selected studies where the only variable that was manipulated was the number of sets. The Forest plot in Figure 2 of our paper shows the effect size difference between higher and lower volumes in each individual study; each individual point is not impacted by the other studies. If there was no impact of volume, then we would expect a random distribution of these data points around 0. However, this is not what occurred; 13 of the 15 studies are to the right of the centre line favouring higher-volume protocols and only 1 is to the left favouring lower-volume protocols; the probability that this distribution occurred by random chance would be extremely unlikely.

Third, Arruda et al. (2016) stated: “Although the authors made an effort to control for confounding variables by using regression models, these did not include other resistance training variables ...” This is incorrect. The duration of training and the portion of the body trained were included as variables. Other resistance training variables were not included because they were highly homogeneous across studies, and thus would not have any impact on outcomes. For example,

the number of repetitions per set was not included because of the homogeneity and narrow range of repetitions across all studies. Training frequency was not included as it too was highly homogeneous across studies (2 or 3 days per week across nearly all studies). The authors went on to mention potential issues with upper versus lower body resistance training programmes by citing that of the 5 studies that included both upper and lower body training, 4 had different patterns of muscle hypertrophy, with upper body generally responding better to higher volumes than lower body. However, this is incorrect. For example, Radaelli et al. (2014) and Bottaro, Veloso, Wagner, and Gentil (2011), report greater percentage gains in the quadriceps with higher volume, but greater percentage gains in the biceps with lower volume. Ostrowski, Wilson, Weatherby, Murphy, and Little (1997) showed similar volume effects between upper and lower body; the percentage gain in triceps increased from 7 to 14 weekly sets, with no further benefit at 27 weekly sets. Similarly, quadriceps showed increased percentage gains from 6 to 12 weekly sets (volume above 12 weekly sets was not examined). While we agree that our analysis might not have been adequately powered to detect an interaction between volume and the portion of the body trained, it should be noted that 10 studies in our analysis included upper body training and 13 included lower body training. Sub-analysis of the data via meta-regression demonstrated dose–response volume effects both for lower and upper body muscles when considered independently (Schoenfeld, Ogborn, & Krieger, 2016). Thus, there is dose–response effect both for the upper and lower body. However, there is insufficient data to determine whether the effect varies between these conditions.

Fourth, Arruda et al. (2016) stated: “Another possible caveat is analyzing the number of sets per exercise, instead of the number of sets in which the muscle was involved”. Our meta-analysis did in fact consider the muscle groups trained per week, not the sets per exercise. This is clearly stated in the methods section and noted throughout the manuscript. The confusion could lie in the mislabelling of Table 1 as “weekly sets per exercise” when it should have been labelled as “weekly sets per muscle group”. We regret that this error was not detected at the proof-reading stage. Arruda et al. (2016) went on to state that “upper body multi joint exercises should be included when counting the number of sets for arm muscles” and suggest that this was not accounted for in our analysis. This claim is incorrect; we did in fact include the relevant arm muscles involved in all multi-joint upper exercises as part of the meta-analysis.

Fifth, Arruda et al. (2016) make a case that differences in training to failure could have influenced results. They discuss

the method used by of [Radaelli et al. \(2015\)](#), stating it is unclear whether the participants trained to true momentary muscular failure. Often, confusion about methods can be clarified by contacting the authors. We did this, and were told that a load was selected to try to obtain failure in the 8–12 repetition range. Loads were reduced on subsequent sets to try to maintain 8–12 RM. If the chosen load resulted in performance of 13 repetitions or more, the load was increased 5–10% at the next session. Thus, participants did train to momentary muscular failure in all groups, and the concern that all participants did not train with similar effort is without merit.

[Arruda et al. \(2016\)](#) went on to state that it “appears unlikely that participants reached momentary muscular failure in most cases”. There is no evidence that momentary muscular failure was not reached in most studies. The only 2 studies, which the authors note, where muscular failure might not have been reached at times are [Sooneste, Tanimoto, Kakigi, Saga, and Katamoto \(2013\)](#)) and [Cannon and Marino \(2010\)](#). In these studies, we acknowledge that the multiple set groups could have consistently trained closer to failure because of accumulated fatigue. However, it should be noted that these 2 studies had among the smallest weights in our analysis, and removal of these studies in the sensitivity analysis did not impact our results. Also, while we acknowledge this as a potential confounder considering it is impossible to know the true effort expended by participants in a given study, the contention that training to complete failure is necessary for maximising hypertrophic gains is largely unsupported. There is evidence that muscle activation reaches a plateau 3–5 repetitions from the point of failure during a 15 RM set ([Sundstrup et al., 2012](#)), suggesting that additional repetitions in the set do not result in greater muscular adaptations. And while there is evidence that stopping well short of failure leads to suboptimal muscle growth ([Goto, Ishii, Kizuka, & Takamatsu, 2005](#)), recent research shows similar hypertrophic gains when sets are stopped 2 repetitions short of failure compared with an all-out effort ([Sampson & Groeller, 2016](#)). Based on the methods reported for the included studies, it is clear that participants in all of these studies trained with a high level of effort even if “true failure” was not reached in some. Thus, the authors’ claim that “the apparent potential benefits of increasing training volume may have been primarily due to a compensation of a low training intensity of effort” therefore seems misguided. Moreover, the contention that there is a greater need to train to complete failure in low-volume training remains highly speculative, with little supporting evidence.

Sixth, [Arruda et al. \(2016\)](#) caution against inclusion of studies with participants of different training history in the same analysis. It should be noted that the vast majority of studies involved untrained participants; only 2 involved trained participants, and sensitivity analysis indicated that neither of these studies had a marked influence on the results. That we identified a dose–response effect of volume with data mostly consisting of untrained participants only further strengthens the likelihood that volume is an important factor in hypertrophy, as trained participants are less responsive to training as the authors pointed out. [Arruda et al. \(2016\)](#) also stated that other factors, such as the sex of participants and their age, were not

considered and could have influenced our outcomes. This is not true. Sex was included as a covariate in our statistical models, and interactions between volume, sex, and age were explored. Also, as mentioned, our statistical models were multilevel random-effects models, which model both variance between effect sizes in each study, as well as variance among studies. Our analysis assumes that the true effect size of volume varies from 1 study to the next, and that studies in our analysis represent a random sample of effect sizes that could be observed. Our statistical models assume that studies are not functionally equivalent or homogeneous. Thus, the authors’ concern that differences in study characteristics could have contaminated our results is without merit.

In addition, [Arruda et al. \(2016\)](#) questioned our results by stating that in the studies that used more than 2 training groups, “... there is no clear sign of graded results with increasing number of sets”. Given the small sample sizes in resistance training studies, and the heterogeneous responses of individuals to training programmes, it is unlikely that any single study would show a perfect dose–response relationship for every muscle group measured, and it is unreasonable for the authors to expect to identify one; this is precisely the reason that meta-analyses are so important for drawing proper inferences in sports science studies. [Arruda et al. \(2016\)](#) went on to state that the results of [Ostrowski et al. \(Ostrowski et al., 1997\)](#) indicated a possibility of overtraining as the number of sets increased, because of alterations in the testosterone/cortisol ratio. However, overtraining is defined as a long-term decrement in performance capacity in which restoration can take several weeks or months ([Halson & Jeukendrup, 2004](#)). There is no evidence that the participants in the [Ostrowski](#) study were overtrained, especially given that the greatest percentage gains in muscle size occurred in the higher-volume groups. Also, the testosterone/cortisol ratio is not considered a reliable marker of overtraining ([Urhausen & Kindermann, 2002](#)).

Finally, [Arruda et al. \(2016\)](#) stated: “... considering the large number of variables involved in resistance training and the important methodological inconsistencies in the current literature, it seems impossible to make comparisons of different studies or include different studies in the same analysis”. On the contrary, we contend that our rigid inclusion criteria ensured that studies had a high degree of homogeneity. They were all randomised trials that attempted to control for all variables while manipulating only training volume. Regression analysis allowed for additional analysis of potential confounders. And while it must be acknowledged that there are always going to be differences in designs that cannot be completely accounted for between studies, such heterogeneity is not necessarily a detriment to drawing inferences from meta-analyses. In fact, if an effect is seen despite discrepancies, it actually strengthens the case for a cause/effect relationship ([Berman & Parker, 2002](#)). Thus, dismissing our findings on this basis is without merit. [Arruda et al. \(2016\)](#) went on to state that “greater value can be obtained by designing and conducting studies of larger and more homogeneous samples”. Given the practical limitations in carrying out large-scale resistance training studies, such as limitations in funding, obtaining sufficient participants, time constraints, etc., it is highly

unlikely that such studies will be performed. Thus, meta-analyses represent the current best tool to estimate the dose–response effect of training volume on hypertrophy. It should be noted that our results are supported by mechanistic data showing greater protein synthetic responses to higher volumes (Burd et al., 2010; Terzis et al., 2010), and recent evidence has shown that the protein synthetic response correlates with hypertrophy beyond the initial stages of training (Damas et al., 2016) .

In conclusion, we feel that our meta-analysis provided compelling evidence for a dose–response relationship between resistance training volume and muscle hypertrophy, and that our systematic approach was valid in drawing these inferences. Graded effects occurred across all outcomes studied and the effects strengthened when direct measures of muscle growth were used. Sensitivity analysis showed that no single study prejudiced results, and no evidence of publication bias was detected. Given the aforementioned information and based on the preponderance of evidence, we contend that a minimum of 10+ sets per muscle per week is necessary to maximise the hypertrophic response to resistance training. Again, this represents a minimum threshold as there were not enough studies that investigated higher volumes to carry out sub-analysis. What now needs to be determined is where the upper threshold for volume lies to promote the greatest increases in muscular gains.

Disclosure statement

No potential conflict of interest was reported by the authors.

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