Medicina Sportiva
Med Sport 15 (1): 41-43, 2011
DOI: 10.2478/v10036-011-0002-4

RESISTANCE EXERCISE INDUCED ACUTE HORMONAL RESPONSES AND CHRONIC ADAPTATIONS: A NULL FIELD?

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Vingren and colleagues [1] recently wrote a review entitled Testosterone Physiology in Resistance Exercise and Training. In their Abstract they claimed: "*Testosterone is important for the desired adaptations to resistance exercise and training; in fact, testosterone is considered the major promoter of muscle growth and subsequent increase in muscle strength in response to resistance training in men*" (p. 1038). The authors presented very little credible evidence in the review to support that claim. Credible supporting evidence would have been comparative resistance training studies where two groups elicited significantly different exercise induced acute hormonal responses and produced significant differences in chronic adaptations such as muscular hypertrophy or strength gains.

Vingren and colleagues [1] stated: "*In general, circulating total testosterone and free testosterone increase immediately after a bout of heavy resistance exercise in men and return to, or below, baseline within 30 minutes*" (p. 1042). The time (30 minutes) is similar to the time for the acute changes (decrease) in plasma volume to return to baseline. Perhaps most of the acute increases in testosterone could be attributable to the decrease in plasma volume. Even if the brief transient increase in testosterone were real (corrected for changes in plasma volume), Vingren and colleagues did not cite any evidence to suggest that the body will produce significant adaptations (e.g., muscle hypertrophy or strength gains) or any significant so-called *upstream regulatory elements* to the actual stimulus (resistance exercise) within 30 minutes post-exercise.

Referring to a study by Wilkinson and colleagues [2], Vingren and colleagues [1] claimed: "*…the combination of unilateral knee extension and leg press performed with 5-6RM loads does not induce a testosterone response*" (p. 1044). The training protocol was actually 3 sets of 6-10RM (weeks 1-4) and one additional set of 5-10RM during weeks 5-8. The protocol was not 5-6RM, as incorrectly claimed by Vingren and colleagues. Curiously, Vingren and colleagues failed to mention that although the protocol did not elicit a significant pre-exercise to post-exercise increase in testosterone, there was a significant increase in unilateral strength (1RM), muscle cross-sectional area and muscle fiber area. Wilkinson and colleagues concluded: "*All of these changes occurred in the absence of changes in systemic anabolic hormones meaning that the unilateral model is one of hypertrophy that is not augmented by systemic hormones*" (p. 554). Rather than fair and balanced reporting by Vingren and colleagues, it appears to be an intentional biased omission of results; that is, they failed to report the outcomes that were antithetical to the authors' primarily unsupported beliefs.

Vingren and colleagues [1] claimed: "*Large muscle mass exercises are needed to acutely increase circulating testosterone concentrations and as a result when large muscle mass exercises are performed in the beginning of an exercise session, the muscle used during subsequent exercises will be perfused with an elevated testosterone concentration. The importance of elevated anabolic hormones including testosterone was shown by the finding that when the biceps is trained after 4 sets of leg press, it hypertrophied significantly more compared with training of the biceps alone*" (p. 1045). They cited only one study [3] in an attempt to support that claim. In the study by Hansen and colleagues [3], the participants performed 8 sets of leg presses—not 4 sets, as wrongly claimed by Vingren and colleagues. The group that performed the lower body exercises in addition to the biceps exercises performed the lower body exercise *after* the arm exercises—not before, as incorrectly claimed by Vingren and colleagues. Hansen and colleagues specifically noted: "*To stimulate higher plasma hormonal levels after arm training, group LA* [leg plus arm training] *in addition trained the legs immediately after the arm training*" (p. 348). Isometric strength significantly increased in the arm plus lower body exercise group but not in the arm-only group. Hansen and colleagues stated: "*However, due to the*

initial [significant] *difference in isometric strength* [20-25%] *caution must be taken with the interpretation of this finding, which may only indicate a possible link between anabolic hormones and muscle strength with training*" (p. 347). Perhaps the key words are *caution* and *possible.* The 1RM biceps curl, which Hansen and colleagues defined as *functional strength*, significantly increased ~19% in both groups, with no significant difference between groups. Curiously, Vingren and colleagues failed to report this result. Most importantly, and directly related to the erroneous claim by Vingren and colleagues concerning the degree of hypertrophy, Hansen and colleagues did not mention or report any measure of muscular hypertrophy. One has to question if Vingren and colleagues or the reviewers for *Sports Medicine* actually read the study by Hansen and colleagues.

In another review [4] by two of Vingren's [1] coauthors (Kraemer and Ratamess), the only comparative resistance training study cited was the study by Hansen and colleagues [3]—and again it was reported incorrectly. A review by Spiering and colleagues [5], which was coauthored by four of Vingren's colleagues (Kraemer, Anderson, Volek and Maresh), also misreported the study by Hansen and colleagues. Interestingly, all these reviews [1, 4, 5] were published in *Sports Medicine*, which certainly questions how this misinformation survived the scrutiny of a total of 10 authors, three sets of reviewers, and the editor.

In a comparative resistance training study, which was not cited by Vingren and colleagues [1], West and colleagues [6] reported the results of 15 weeks of arm training in 12 recreationally active young males. One arm was trained in a low hormonal environment (arm-only exercise) and the contralateral arm in a high hormonal environment (arm plus lower body exercise) on separate days. There was no significant increase in growth hormone, IGF-I or testosterone pre-exercise to post-exercise in the arm-only exercise condition, but there were highly significant elevations in the aforementioned anabolic hormones in the arm plus lower body exercise condition. However, there was a significant increase in both arms (low and high hormonal conditions, respectively) for isometric strength (20% and 19%), 1RM (23% and 25%), 10RM (46% and 47%), Type I muscle fiber cross-sectional area (9% and 11%), Type II muscle cross-sectional area (21 and 24%), and elbow flexor cross-sectional area (12% and 10%), with no significant difference between contralateral arms for any of these resistance training induced adaptations. West and colleagues concluded: "*Despite vast differences in hormone availability in the immediate postexercise period, we found no differences in the increases in strength or hypertrophy in muscle exercised under low or high hormone conditions after 15 weeks of resistance training*" (p. 64).

Vingren and colleagues [1] devoted whole sections in their review to specific resistance training variables such as *Intensity* (defined by the authors as % 1RM), *Number of Sets* for each exercise, *Volume* (sets x repetitions x resistance), *Choice of Exercise* (free weights or machines, muscle action, and repetition duration), *Order of Exercise* (sequence of exercise within a session), and *Rest Period Duration* (inter-set and inter-exercise rest duration), which may affect acute post-exercise hormonal responses. However, there is very little evidence to suggest that the manipulation of any of the aforementioned variables have any significant effect on specific resistance training adaptations such as muscular strength [e.g., references 7-15].

Vingren and colleagues [1] concluded: "*The increase in testosterone found in men is important for resistance exercise-induced adaptations*" (p. 1049). They failed to present any evidence (comparative resistance training studies) to support that conclusion. In fact, they cited only one study [3] and selectively reported the one questionable (questionable even by Hansen and colleagues) outcome that supported their opinion.

Ioannidis [16] has astutely noted: "*Prestigious investigators may suppress via the peer review process the appearance and dissemination of findings that refute their findings, thus condemning their field to perpetuate false dogma*" (p. 698). He concluded: "*Of course, investigators working in any field are likely to resist accepting that the whole field in which they have spent their careers is a 'null field'*" (p. 700). There is very little credible evidence to show cause and effect between resistance exercise induced acute hormonal responses and chronic adaptations such as muscular hypertrophy or strength gains. Therefore, it appears that this hypothetical unsubstantiated relationship between exercised induced acute hormonal elevations and resistance training adaptations may be a *null field*.

Disclosure

In order to stay within the journal's 1000 word limit, I submitted an abbreviated version of this Brief Commentary as a Letter to the Editor of *Sports Medicine* (Jeremy Shanahan) on 12-05-10. Not surprisingly and without any explanation, Shanahan rejected my letter on 12-06-10. It appears that the Editor is afraid of any challenge to the unsubstantiated opinions and misinformation that are published in *Sports Medicine* or to its defective peer-review process.

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Received: December 09, 2010
Accepted: January 15, 2011
Published: February 07, 2011

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