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EDITORIAL

The long and short of residual force enhancement non-responders

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Residual force enhancement (rFE) refers to the 'extra' force that is achieved in an isometric contraction following active muscle lengthening as compared to a strictly isometric contraction (i.e., fixed end contraction) performed at the same muscle length and level of neuromuscular activation. As an intrinsic property of muscle contractility, rFE is present from the single sarcomere, to whole muscles of humans during electrical stimulation and voluntary activation (Chapman et al. 2018; Seiberl et al. 2015).

In the current issue of EJAP, Bakenecker et al. (2020) investigated the effect of muscle–tendon unit length on transient force enhancement (tFE; force production during an eccentric contraction) and rFE (steady-state isometric force following active lengthening). They found that tFE was indeed present at short and long muscle–tendon unit lengths, while rFE was blunted at shorter lengths. This finding is consistent with the phenomenological mechanisms of rFE, which shows greater magnitudes of rFE on the descending limb of the length–tension relationship as compared with the plateau or ascending limb (Rassier and Herzog 2004). However, it is possible that significant rFE was not observed at short muscle lengths, because 3–4 of the 12 participants displayed no rFE. Conversely, all participants showed rFE at long muscle lengths (see Fig. 3). This offers an important discussion point, from reduced muscle preparations we would expect rFE across all operating muscle lengths, just less at shorter than at longer lengths (Rassier and Herzog 2004). It does not necessarily need to be stated that humans performing voluntary contractions are not isolated muscles in a bath of chemicals, thus a myriad of neuromuscular factors could contribute to blunting rFE. With this, an important consideration in the rFE field is the phenomenon of non-responders.

Non-responders have been identified as human research participants exhibiting negligible or no rFE. Various factors related to neural excitation/inhibition and motor unit recruitment, as well as mechanical factors such as series compliance could all contribute to the variability in reported magnitudes of rFE. Understanding the underlying mechanisms of muscle contractility is crucial, but the implications of an intact human neuromuscular system on modulating rFE cannot be overlooked when investigating this history-dependent property of muscle. Below we discuss some of these considerations.

Level of activation and fibre type: Based on results from the only study directly investigating the EMG–force relationship following active lengthening, relative values (i.e., steady-state force following active lengthening minus isometric force, divided by isometric force) of rFE (~5%) for the human dorsiflexors appear to be similar for tibialis anterior activation levels between 20 and 100% MVC (Paquin and Power 2018). Meanwhile, Oskouei and Herzog (2005) determined that increasing levels of activation (10%, 30%, and 60% MVC) led to greater absolute values of rFE, as well as more responders (i.e., 4/12 subjects exhibiting rFE at 10% MVC vs. 10/12 at 60% MVC), for the human adductor pollicis. Therefore, the level of neuromuscular activation appears to affect absolute, but not relative, values of rFE. One way to get around this apparent activation dependency is with electrically evoked contractions. Lee and Herzog (2002) performed voluntary and electrically-evoked contractions of the adductor pollicis muscle and found no difference in rFE following slow speed lengthening contractions (16% vs. 17%, respectively), however, during the faster speed lengthening contractions, voluntary rFE was less than electrically stimulated (12% vs. 17%, respectively)—thus, it seems with increasing lengthening speeds, there may be factors affecting voluntary performance, such as neural inhibition.

To investigate whether resistance training may alter rFE, Chen and Power (2019) performed a 4-week study

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biased to concentric and eccentric training. They found that concentric training increased rFE while eccentric training decreased rFE for the human ankle dorsiflexors. The rFE increase was attributed to an increase in the number of responders (from 11/15 to 15/15) following training, while the rFE decrease was attributed to increased antagonist muscle (soleus) co-activation. Given that training only lasted 4 weeks—during which adaptations are predominantly neural—these results point to a potential neural contribution to non-responders. More carefully controlled longitudinal studies and day-to-day reliability measures of non-responders are warranted to fully understand the phenomenon of non-responders.

If muscle fibre type contributes to the magnitude of rFE, then recruiting high threshold motor units would presumably increase responders and explain the activation dependency often observed. The first study investigating muscle fibre type differences in rFE was performed by Ramsey et al. (2010), which compared rFE in the soleus (SOL) and extensor digitorum longus (EDL) of rats. Ramsey et al. (2010) reported ~ 55% greater rFE values for the EDL, which is comprised of primarily (95–100%) type II fibres, compared to the SOL, which is primarily (75–80%) type I fibres. The authors speculate that the expression of shorter, stiffer titin isoforms—which contribute greater passive force to total force production—in predominantly fast-twitch muscles may contribute to the fibre type differences in rFE. In contrast to prior studies, Pinnell et al. (2019) found no fibre type differences for rFE in human single fibres of the vastus lateralis (VL), and suggested this may be due to fewer fibre type differences in titin isoforms within a single muscle compared to across different muscles. In humans at least, differences in fibre type across muscles may not be a determining factor in the incidence of non-responders.

Central nervous system excitability: Reduced central drive, increased inhibition, or a combination of both could contribute to the incidence of non-responders. While a reduction in agonist activation is almost certain, it appears to be related, in part, to spinal excitability (Sypkes et al. 2018). Contento et al. (2019) reported an increased tendon-evoked inhibitory reflex during the rFE state compared to strictly isometric contractions. These results likely indicate inhibitory feedback onto the agonist motoneuron pool that is arising from a tension-dependent source within the tendon, most likely the golgi tendon organ, and subsequently reducing spinal excitability (Sypkes et al. 2018). Surprisingly, when vibration was applied to the muscle, vibratory excitation of muscle spindles does not appear to alter rFE (Dalton et al. 2018). Thus, if certain individuals experience more tension-dependent inhibition during and/or following active lengthening, this alteration in descending drive or spinal excitability would likely contribute to the non-responder phenomenon.

Muscle–tendon unit compliance: In some cases, more rFE may just be less residual force depression (rFD). When Raiteri and Hahn (2019) increased the stiffness of the human tibialis anterior muscle–tendon unit (MTU) at the onset of a 'fixed-end' (i.e., isometric) contraction via a small, quick joint rotation, they found that isometric force was increased, owing to reduced internal shortening of fascicles upon activation. In other words, even for isometric contractions where no joint angle changes occur, MTU compliance appears to play a role in rFD, whereby a more compliant tendon permits greater fascicle shortening, and thus, more absolute shortening-induced rFD (and vice versa). Moreover, for 'fixed-end' isometric contractions, where internal shortening of fascicles can occur, decreasing levels of activation result in lower absolute amplitudes of shortening-induced rFD, owing to a smaller magnitude of internal shortening and a potential triggering of rFE-related mechanisms as force drops and fascicles actively relax/lengthen (Raiteri and Hahn 2019). In short, for conventional rFE contractions that compare back to a reference isometric contraction, the level of activation likely influences absolute values of rFE. However, if isometric contractions were adjusted for any potential internal shortening of fascicles, it might reveal underestimations of rFD and possible overestimations of rFE in the literature. What we may see is not really force enhancement but less force depression in the stretched muscle compared to the isometric reference conditions.

How do we deal with non-responders in a data set? To properly answer this question, there needs to be a better understanding of why non-responders exist. Once again, tracking the dynamic behaviour of muscle architecture during contractions might be a good place to start, and this was an elegant aspect of the study by Bakenecker et al. (2020). Depending on the research question, to limit non-responders in a data set, the available literature would point to using large joint excursions biased to long muscle–tendon unit lengths, with high levels of activation. Although, a non-responder in and of themselves may provide critical insight and clues into the everyday relevance of rFE. Therefore, it is important to consider that while these intrinsic contractile properties of muscle always exist during carefully controlled in vitro experiments, the complex human neuromuscular system may or may not always follow suit.

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Compliance with ethical standards

Conflict of interest No conflicts of interest, competing interest of any sort, financial or otherwise, are declared by the authors.

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