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Short communication

Residual force enhancement and force depression in human single muscle fibres

Rhiannan A.M. Pinnell [a], Parastoo Mashouri [a], Nicole Mazara [a], Erin Weersink [b], Stephen H.M. Brown [a], Geoffrey A. Power [a,*]

[a] Department of Human Health and Nutritional Sciences, College of Biological Sciences, University of Guelph, Guelph, Ontario, Canada
[b] Health and Performance Centre, Sports Medicine Clinic, University of Guelph, Guelph, Ontario, Canada

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ABSTRACT

Residual force depression (rFD) and residual force enhancement (rFE) are intrinsic contractile properties of muscle. rFD is characterized as a decrease in steady-state isometric force following active shortening compared with a purely isometric contraction at the same muscle length and level of activation. By contrast, isometric force is increased following active lengthening compared to a reference isometric contraction at the same muscle length and level of activation; this is termed rFE. To date, there have been no investigations of rFD and rFE in human muscle fibres, therefore the purpose of this study was to determine whether rFD and rFE occur at the single muscle fibre level in humans. rFD and rFE were investigated in maximally activated single muscle fibres biopsied from the vastus lateralis of healthy adults. To induce rFD, fibres were activated and shortened from an average sarcomere length (SL) of 3.2–2.6 μm. Reference isometric contractions were performed at an average SL of 2.6 μm. To induce rFE, fibres were actively lengthened from an average SL of 2.6–3.2 μm and a reference isometric contraction was performed at an average SL of 3.2 μm. Isometric steady-state force was lower following active shortening ($p < 0.05$), and higher following active lengthening ($p < 0.05$), as compared to the reference isometric contractions. We demonstrated rFD and rFE in human single fibres which is consistent with previous animal models. The non-responder phenomenon often reported in rFE studies involving voluntary contractions at the whole human level was not observed at the single fibre level.

1. Introduction

Residual force enhancement (rFE) and residual force depression (rFD) are history-dependent properties of muscle investigated extensively over the last six decades (Abbott and Aubert, 1952; Edman et al., 1978; Seiberl et al., 2015; Chen et al., 2019a). rFE and rFD are characterised as an increase or decrease in steady-state isometric force following active lengthening or shortening, respectively, compared to a reference isometric contraction at the same muscle length and level of activation (Abbott and Aubert, 1952). These history-dependent muscle properties have been studied from the level of the sarcomere (Leonard et al., 2010) in rabbit, up to *in vivo* electrically evoked (Lee et al., 2002; Lee et al., 2003), and voluntary contractions (Seiberl et al., 2015; Jones et al., 2016; Paquin and Power, 2018) in humans. While mechanistic insight has been gained from animal models, and phenomenological factors contributing to the history-dependence of force during voluntary contractions have been studied in humans *in vivo*, there have been no investigations of rFE and rFD at the cellular level in humans.

A common observation during *in vivo* human testing of rFE and rFD is the phenomenon of a 'non-responder' (Seiberl et al., 2015; Chapman et al., 2018; Chen et al., 2019a). One aim of the present study was to investigate whether non-responders exist at the single fibre level in human tissue. Approximately 25% of participants do not show the typical rFE and rFD response despite performing the same active muscle lengthening and shortening contractions. This could be due to: the portion of the force-length relationship where the lengthening/shortening is performed, variability in fibre lengthening/shortening during the whole muscle active stretch/shortening (Herzog, 2004), and ambiguity regarding whether rFE and rFD are intrinsic properties of human single muscle fibres. To determine whether rFE and rFD are present at the cellular level,

* Corresponding author at: Neuromechanical Performance Research Laboratory, Department of Human Health and Nutritional Sciences, College of Biological Sciences, University of Guelph, Ontario, Canada.
E-mail address: gapower@uoguelph.ca (G.A. Power).

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we investigated these properties in single human muscle fibres biopsied from the thigh.

2. Methods

2.1. Preparation

Fibres were biopsied from the vastus lateralis (VL), centered between the lateral epicondyle and the greater trochanter of the femur, of young (n = 6, 25.5 ± 1.28yrs, 81.7 ± 5.2 kg, 175.3 ± 2.29 cm) and older (n = 6, 70.7 ± 3.6yrs, 76.9 ± 4.4 kg, 175.3 ± 2.7 cm) males. One to three fibres from each biopsy sample were included in the data set. Samples were placed in a skinning solution for 1 day at 4 °C, then stored in a 1:1 skinning-glycerol solution at −20 °C for 2–4 weeks. Single fibres were dissected in relaxing solution and tied between a length controller (322C, Aurora Scientific, Toronto, ON, Canada) and force transducer (403A, Aurora Scientific). All experiments were performed at 16 °C, this allowed for a balance between force production capacity (Bottinelli et al, 1996), and the number of contractions performed before experiencing force loss. Muscle contraction was initiated by transfer from relaxing solution to a pre-activating solution with reduced Ca^{2+} buffering capacity, followed by an activating solution with high concentrations of Ca^{2+} (pCa = 4.2) and ATP. Please see supplemental material for details on solutions.

2.2. Mechanical testing and force measurements

For all tests, average sarcomere length was determined using a high speed camera (Aurora Scientific Inc., HVSL 901A). Total force was recorded and active force was determined by subtracting passive from total force.

Force depression: Fibres were set to an average SL of 2.6 μm, then passively lengthened to 3.2 μm (working range of the VL *in vivo*). Activation began 10 s after lengthening. After 20 s of contraction, fibres were actively shortened to an average SL of 2.6 μm at $0.23L_0$/s and held for 15 s before deactivation. A 15 s time interval was used to allow the fibres to reach steady state or be as close to steady state as possible without damaging them. For the isometric reference contractions (ISO), fibres were activated at an average SL of 2.6 μm for 35 s (Fig. 1a).

Residual force enhancement: Fibres were set to an average SL of 3.2 μm and passively shortened to 2.6 μm. Activation began 10 s after shortening. After 20 s of activation, fibres were actively lengthened to 3.2 μm at $0.23L_0$/second and held for 15 s before deactivation. For ISO, fibres were set to a SL of 3.2 μm in relaxing solution, then passively shortened to 2.6 μm for 3 s, passively stretched back to 3.2 μm (to account for passive resistance to stretch) and held for 10 s before being activated for 40 s and then deactivated (Fig. 1b).

Instantaneous fibre stiffness was measured in all trials after 35 s of activation by stretching by 0.3% total length at a speed of $3L_0$/s. Following mechanical testing, fibre type was determined via SDS-PAGE (Fig. 2).

2.3. Analysis

Transient aspects of force: Mechanical work of shortening in the rFD trial was calculated as the product of average force and the change in fibre length. Peak eccentric force was the highest value observed during lengthening in the rFE trial.

Stiffness: Instantaneous stiffness was calculated as the difference between peak force during the stretch and the average force over the 500 ms prior to the stretch, divided by the change in fibre length induced by the stretch.

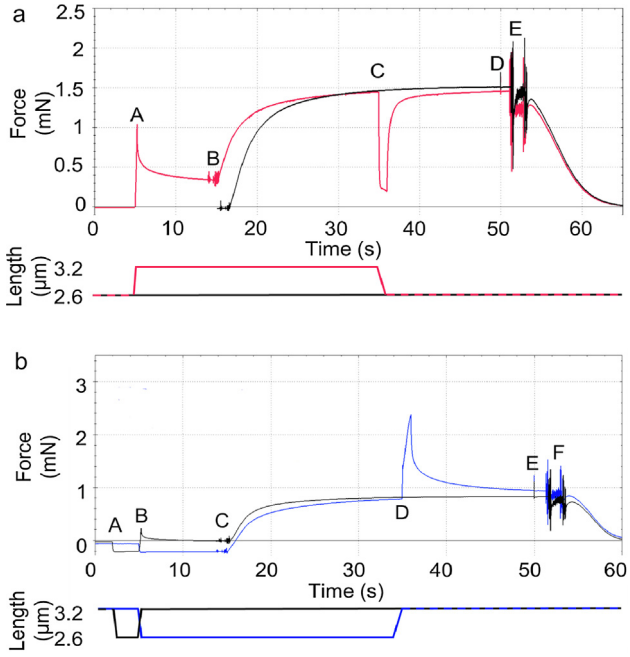

Fig. 1. (a) Representative force tracing for a type II fibre for the force depression (red) and isometric reference (black) conditions. (A) passive stretch of the reference isometric condition from 2.6 μm to 3.2 μm. (B) activation in both conditions. (C) active shortening from 3.2 μm to 2.6 μm in the force depression condition. (D) instantaneous stiffness test in both conditions. (E) deactivation in both conditions. Noise represents changing of baths. (b) Representative force tracing for a type II fibre for the force enhancement (blue) and isometric reference (black) conditions. (A) shortening of the isometric reference from 3.2 μm to 2.6 μm. (B) passive stretch of the isometric reference from 2.6 μm to 3.2 μm and passive shortening of the force enhancement fibre from 3.2 μm to 2.6 μm. (C) activation in both conditions. (D) active stretch in the force enhancement condition stretch from 2.6 μm to 3.2 μm. (E) instantaneous stiffness test in both conditions. (F) deactivation in both conditions. The order of contraction was always performed in the following sequence: rFD, ISO(2.6 μm), ISO(3.2 μm), and rFE. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

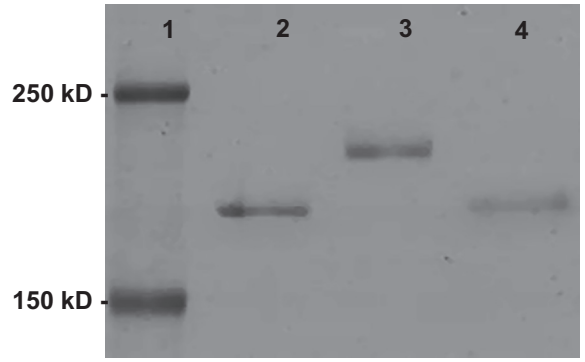

Fig. 2. Representative gel. Fibre type was assessed using SDS-PAGE. Lane 1 is a ladder of molecular weights, lanes 2 and 4 contain fibres expressing myosin heavy chain (MHC) I and lane 3 contains a fibre expressing MHC II.

History-dependent properties of force: For rFD, rFE, and the ISO values, force was reported as an average over the 500 ms before the instantaneous stiffness test at the same time following activation for all conditions. For the rFE and rFD trials, this time point corresponded to the isometric steady-state 15 s following the dissipation of lengthening/shortening force transients. rFD was calculated as the percent difference between the average force in the rFD condition (2.6 μm) compared to the ISO condition. rFE was calculated as the percent difference between the force in the rFE

condition (3.2 μm) compared to the ISO. Following deactivation, passive force was recorded as the average force over the 500 ms prior to the end of data collection. Passive force enhancement (PFE) was calculated as the percent difference between the passive force after deactivation from the rFE and the ISO contraction. All force measures were adjusted for resting passive tension by subtracting the average baseline value for the first 50 ms of the trial. A linear regression was performed between %rFD or %rFE, and the change in instantaneous stiffness between the ISO and the history-dependent conditions.

Statistics: The study was underpowered for age comparisons; thus data was pooled across age (supplemental figure). Paired t-tests were used to compare variables between the ISO and rFD state, as well as between the ISO and rFE state. Unpaired t-tests were used to compare across fibre type. Significance was set at $p < 0.05$.

3. Results

22 single muscle fibres (young, n = 9; old, n = 13) were tested. 15 were type I and 7 were type II. After completing the rFD experiment, 2 fibres were damaged during the high force active lengthening protocol and were not included in the rFE data.

Isometric force: Absolute isometric force was greater for type II compared to type I fibres at 2.6 μm and 3.2 μm average SL, respectively. However, when normalized to CSA to account for myofibular content, there were no differences in specific force across fibre type (Table 1).

Table 1
Single muscle fibre contractile properties across fibre type.

Muscle fibre contractile properties	Type I (n = 15)	Type II (n = 7)	P-value
Iso force - SL 2.6 μm (mN)	0.609 ± 0.076	0.890 ± 0.131	***0.06***†
Normalized Iso Force - SL 2.6 μm (mN/mm^2)	108.5 ± 9.3	139.1 ± 21.2	0.14
Iso force (SL 3.2 μm) (mN)	0.454 ± 0.066	0.735 ± 0.120	***0.04****
Normalized Iso Force - SL 3.2 μm (mN/mm^2)	83.3 ± 6.6	114.7 ± 19.7	0.08
Mechanical Work of Shortening (J)	0.010 ± 0.002	0.042 ± 0.013	***0.04****
Eccentric Force (mN)	1.140 ± 0.137	1.735 ± 0.269	***0.04****
Stiffness (Iso) - SL 2.6 μm (mN/mm)	43.2 ± 4.4	50.3 ± 5.2	0.35
Stiffness (rFD) - SL 2.6 μm (mN/mm)	40.9 ± 4.1	49.4 ± 6.0	0.26
Stiffness (Iso) -SL 3.2 μm (mN/mm)	33.6 ± 4.1	38.3 ± 3.8	0.41
Stiffness (rFE) - SL 3.2 μm (mN/mm)	34.1 ± 4.0	41.8 ± 4.2	0.20

Statistically significant or approaching significance.
* $p < 0.05$.
† Trending towards statistical significance.

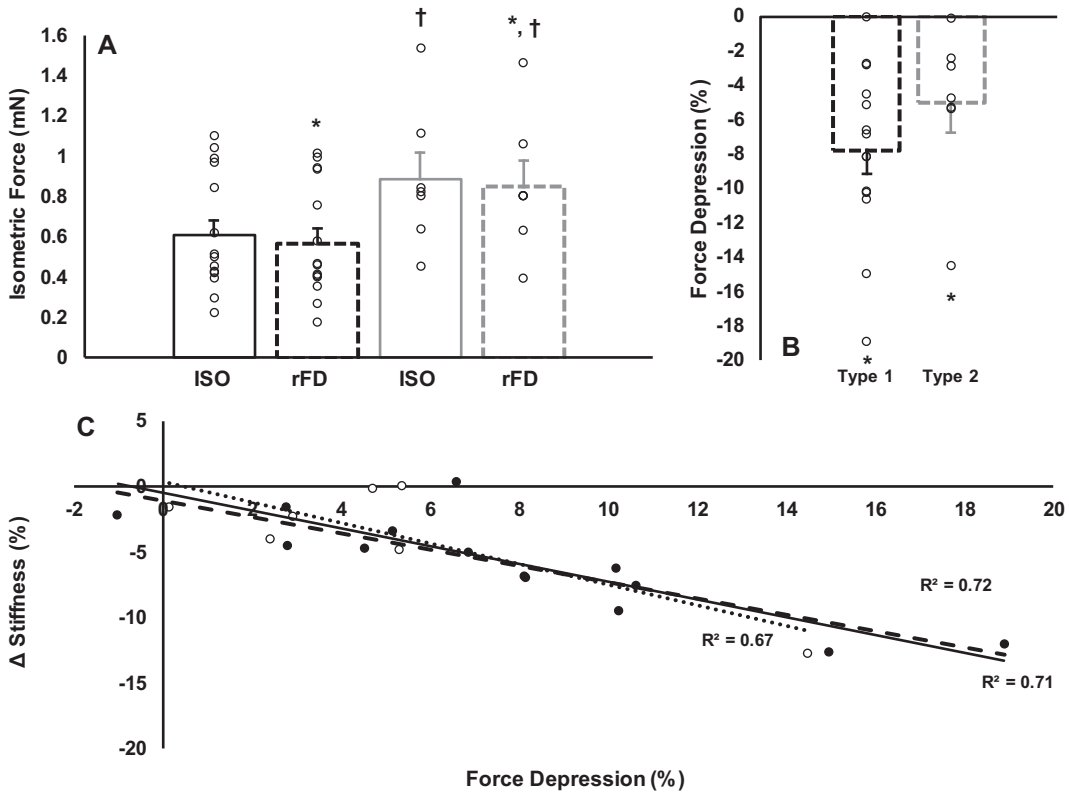

Fig. 3. Shortening induced force depression. (A) Absolute isometric force during the purely isometric contraction (solid bars), and isometric steady-state following active shortening (dotted bars), for type I ($p = 0.004$) (black) and type II fibres ($p = 0.007$) (grey). Type II fibres produced greater force as compared with type I. (B) Force depression was present for both fibre types and did not differ across fibre type ($p = 0.25$). (C) Linear regression of force depression and change in stiffness for type I (n = 13, dashed line, closed circles; two fibres removed for >3SD stiffness values) ($R^2 = 0.72$, $p = 0.004$), type II (n = 7, dotted line, open circles) ($R^2 = 0.67$, $p = 0.006$), and across fibre type (solid line; $R^2 = 0.71$, $p = 0.068$). With increasing levels of force depression there is a greater reduction in stiffness ($p < 0.001$). *Force depression (rFD) different from isometric (ISO), † Effect of fibre type. Data presented as mean ± SEM.

Transient aspects of force: During active shortening, type II fibres performed 76.4% more work as compared with type I (Table 1). During active lengthening, peak force was 34.6% greater for type II fibres as compared with type I (Table 1).

Residual force depression: Force following active shortening was lower as compared with ISO ($p = 0.004$ (TI), p = 0.007 (TII); Fig. 3A) and the magnitude of the decrease did not differ across fibre type (Fig. 3B; $p = 0.25$). rFD for type I fibres was 7.8% (range: 0–19%), and for type II was 5.1% (range: 0–15%). Two fibres demonstrated a rFD of '0'; this may owe to our conservative approach in testing order, wherein we perform rFD first and then ISO, thus fatigue or damage could have lowered the ISO value. Other fibres from the two subjects who had fibres with rFD of "0" exhibited rFD. Instantaneous stiffness decreased 5.2% and 3.6% in the rFD state for type I and II fibres, respectively (Table 1; $p = 0.004$ (TI), $p = 0.006$ (TII)) and did not differ across fibre type ($p = 0.68$). There was a strong negative relationship between %rFD and stiffness (Fig. 3C) for both fibre types, and when pooled across fibre type.

Residual force enhancement: Force following active lengthening was higher as compared with ISO ($p = 0.0001$ (TI), $p = 0.001$ (TII); Fig. 4A), and the magnitude of the decrease did not differ across fibre type (Fig. 4B; $p = 0.64$). rFE for type I fibres was 12.9% (range: 6–25%) and for type II was 11.5% (range: 6–23%). Upon deactivation, passive force was greater in the rFE condition as compared with ISO ($p = 0.02$ (both fibre types); Fig. 5), and was similar across fibre type ($p = 0.79$). Instantaneous stiffness did not differ for type I fibres in the rFE state, however there was a trend for stiffness in type II fibres to increase by 5.1% ($p = 0.08$), and a trend to differ across fibre type ($p = 0.07$). There was a positive relationship between %rFE and stiffness for type I fibres, and when pooled across fibre type (Fig. 4C).

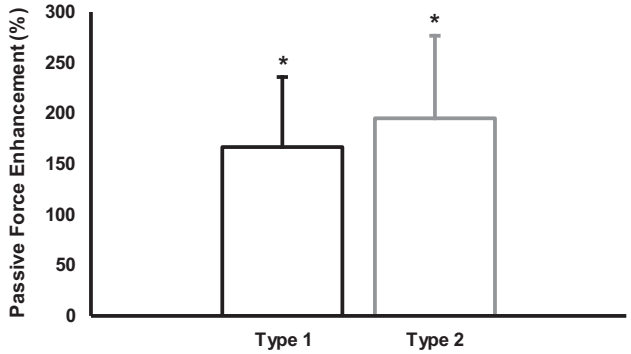

Fig. 5. Passive force enhancement (PFE) was greater following active lengthening as compared with the purely isometric contraction (n = 17). There was no effect of fibre type. *Residual force enhancement (rFE) different from isometric (ISO). Data presented as mean ± SEM.

4. Discussion

The main findings of our study indicate that rFE and rFD are indeed intrinsic contractile properties of human single muscle fibres. The observed values for rFE agree with previously reported values of ~15% in rabbit single fibres (Joumaa and Herzog, 2013), rodent whole muscle (Ramsey et al., 2010), and voluntary contractions in humans (Seiberl et al., 2013, 2015; Chapman

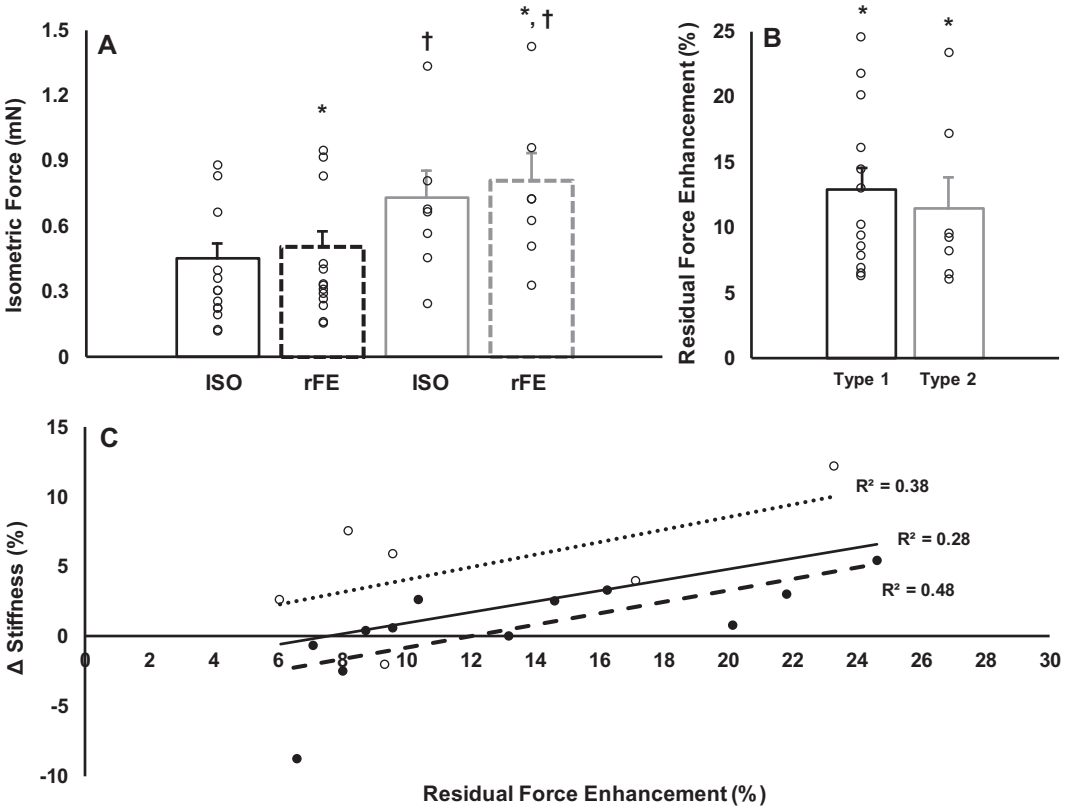

Fig. 4. Lengthening induced residual force enhancement. (A) Absolute isometric force during the purely isometric contraction (solid bars), and isometric steady-state following active lengthening (dotted bars), for type I ($p = 0.0001$) (black) and type II ($p = 0.001$) (grey) fibres. Type II fibres produced greater force as compared with type I. (B) residual force enhancement was present for both fibre types, and there were no differences between fibre types ($p = 0.64$). (C) Linear regression of residual force enhancement and change in stiffness for type I (n = 12, dashed line, closed circles; one fibre removed for >3SD stiffness values) ($R^2 = 0.48$, $p = 0.01$), type II (n = 6, dotted line, open circles; one fibre removed for >3SD stiffness values) ($R^2 = 0.38$, $p = 0.19$), and across fibre type (solid line; $R^2 = 0.28$, $p = 0.02$). With increasing levels of residual force enhancement there is a greater increase in stiffness ($p = 0.024$). *Residual force enhancement (rFE) different from isometric (ISO), † Effect of fibre type. Data presented as mean ± SEM.

et al., 2018). The values for rFD were also consistent with previously reported values in rabbit single muscle fibres (~8%; type I fibres and ~15%; type II fibres; Joumaa et al., 2015), and voluntary contractions in humans (Chen et al., 2019a,b). The 2.6 μm and 3.2 μm average SL used in this study is similar to the physiological SL of the vastus lateralis during knee extension (2.84 μm) and flexion (3.17 μm) (Chen et al., 2016). Our data therefore indicate that rFD and rFE occur in human single muscle fibres across physiologically relevant muscle lengths.

Isometric force, eccentric force, and work of shortening were higher in type II as compared with type I fibres. These results are consistent with previously reported values in human single muscle fibres (Bottinelli et al., 1996). As well, despite type II fibres producing higher eccentric force as compared with type I, this did not translate into higher rFE values, indicating that rFE is independent of peak eccentric force in human fibres, and that the mechanisms of eccentric force are not directly related to the mechanisms of rFE. These findings have previously been reported in animal models (Ramsey et al., 2010; Herzog, 2004).

Force depression likely occurs due to a decrease in the number of attached cross-bridges following active shortening compared to a purely isometric contraction owing to a stress-dependent deformation of actin (Joumaa et al., 2012, 2018; Marechal and Plaghki,1979, Sugi and Tsuchiya,1988) and is directly related to the work of shortening (Herzog and Leonard, 2000). In the present study, rFD was associated with a reduction in muscle fibre stiffness for both fibre types, owing to a decreased proportion of attached force-producing cross-bridges. Joumaa et al. (2015) showed type II fibres (rabbit psoas) exhibited greater rFD than type I when shortened at an absolute speed. However, when they normalized shortening speed to maximal shortening velocity, rFD was no longer dependent on fibre type. Human type I and II muscle fibres are less divergent in contractile velocity than rabbit fibres (Schiaffino and Reggiani, 2011), which may in part explain why we found no fibre-type dependent difference in rFD. Presumably, if the speed of shortening was increased, fibre-type differences would emerge.

The mechanisms responsible for rFE are less clear. One proposed mechanism is an increase in the proportion of strongly bound cross-bridges during active lengthening, which persists during the isometric steady-state (Rassier and Herzog,2005; Linari et al.,2000). Recently, a calcium-dependent increase in the stiffness of the molecular spring titin, (Powers et al., 2017, 2014, 2016; Monroy et al., 2017; Leonard and Herzog,2010) and a reduction in its free-spring length (Herzog,2018), due to actin-titin interaction (Rode et al., 2009; Dutta et al.,2018) has been proposed as the main contributor to stretch-induced rFE. This titin-based mechanism is attractive because it explains the elevated passive force upon muscle deactivation (Herzog, 2018). In the present study we observed significant rFE as well as PFE for both fibre types, and an increased instantaneous stiffness in type I fibres in the rFE state. The change in stiffness may represent an increase in the proportion of attached cross-bridges (Ford et al., 1977). In the present study, the increase in stiffness (i.e., active) and PFE (i.e., passive) support the existence of both previously proposed cross-bridge (active) and non-cross-bridge (passive) based contributions to rFE in human single muscle fibres. In single fibres from the rabbit psoas, no change in stiffness was reported after active stretch (Joumaa and Herzog, 2013), but Rassier and Herzog (2005) reported increased stiffness following active lengthening for amphibian muscle. Therefore, the elevated stiffness values in human type I fibres in the rFE state require further investigation. Ramsey et al. (2010) reported higher rFE values for the fast-twitch rat EDL whole muscle as compared with the slow-twitch soleus muscle; however, in the present study we did not observe an effect of fibre type. There may be fewer fibre-type differences in titin isoforms in a single muscle than across muscles (Fry et al., 1997; Prado et al., 2005). Therefore, we would not expect titin, and its contribution to rFE, to differ between fibres within a single muscle.

We have shown for the first time that rFE and rFD are present in human single muscle fibres following active lengthening and shortening, respectively. Therefore, the 'non-responder' phenomenon must occur upstream from the cellular level. The functional implications of these history-dependent properties on everyday life require further elucidation.

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Declaration of Competing Interest

No conflicts of interest, financial or otherwise, are declared by the authors.

Ethics statement

Participants gave written informed consent prior to testing. All procedures were approved by the human Research Ethics Board of the University of Guelph (16JL006) and, with the exception of registration in a database, conformed to the Declaration of Helsinki.

Data accessibility

Supporting data are available upon request.

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Author contributions

All authors contributed equally.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jbiomech.2019.05.025.

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