Journal of Biomechanics 47 (2014) 3423–3427

Contents lists available at ScienceDirect

Journal of Biomechanics

journal homepage: www.elsevier.com/locate/jbiomech
www.JBiomech.com

Short communication

Decay of force transients following active stretch is slower in older than young men: Support for a structural mechanism contributing to residual force enhancement in old age

Geoffrey A. Power [a,*], Walter Herzog [a], Charles L. Rice [b,c]

[a] Faculty of Kinesiology, Human Performance Laboratory, University of Calgary, Calgary, Alberta, Canada
[b] Canadian Centre for Activity and Aging, School of Kinesiology, Faculty of Health Sciences, The University of Western Ontario, Canada
[c] Department of Anatomy and Cell Biology, The University of Western Ontario, Canada

ARTICLE INFO

Article history:
Accepted 30 August 2014

Keywords:
Aging
Muscle
Eccentric
Residual force enhancement
Stiffness

ABSTRACT

Following active lengthening of muscle, force reaches an isometric steady state above that which would be achieved for a purely isometric contraction at the same muscle length. This fundamental property of muscle, termed "residual force enhancement (RFE)," cannot be predicted by the force-length relationship, and is unexplained by the cross-bridge theory of muscle contraction. Recently, we showed that older adults experience higher RFE than young for the ankle dorsiflexors primarily owing to a greater reliance on passive force enhancement (PFE) and similar RFE for the knee extensors but a greater contribution of PFE to total RFE. Natural adult aging may prove a useful model in exploring mechanisms of RFE which may reside in the dissipation of force transients following stretch. A post-hoc analysis was conducted on previously described RFE experiments in young (~26 years) and old (~77 years) men for the dorsiflexors and knee extensors to fit the force following stretch with a biexponential decay. In both muscle groups the decay half-life of the first exponential was two times slower in the older compared with young men. There were significant associations between PFE and the decay in force, suggesting a greater "non-active" contribution to total RFE across muscles in older compared with young men. The greater "non-active" component of RFE in older adults could be due to structural age-related changes causing increased muscle stiffness during and following stretch.

1. Introduction

Residual force enhancement (RFE) is a fundamental history-dependent property of skeletal muscle, with force values above those predicted by the force-length relationship and unexplained by the current cross-bridge theory of muscle contraction (Abbott and Aubert, 1952; Edman et al., 1982; Herzog et al., 2008; Walcott and Herzog, 2008). Following active lengthening of skeletal muscle, RFE can be observed after the dissipation of force transients upon reaching an isometric steady state relative to a purely isometric contraction performed at the same final muscle length (Edman, 2012). Additionally, when tested on a portion of the force-length relationship coinciding with measureable passive force, the resting force during relaxation following stretch is elevated compared with a purely isometric contraction, and is referred to as passive force enhancement (PFE) (Joumaa et al., 2007). A combination of both active and passive structural properties of muscle force production, related to the force generating and transmitting structures, is thought to contribute to total RFE (Herzog et al., 2012).

Residual force enhancement, upon reaching an isometric steady state, has been well characterized in reduced muscle preparations (Herzog et al., 2006; Hisey et al., 2009; Joumaa et al., 2008; Lee et al., 2007; Mehta and Herzog, 2008; Rassier and Herzog, 2002; Rassier and Pavlov, 2012) and in humans (Edman et al., 1982; Hahn et al., 2010; Lee and Herzog, 2002; Minozzo et al., 2013; Pinniger and Cresswell, 2007; Power et al., 2013b; Power et al., 2012b, c; Seiberl et al., 2010; Shim and Garner, 2012; Tilp et al., 2009). However, few investigations have characterized the force transient phase immediately following stretch to the isometric steady-state force level (Cavagna, 1993; Koppes et al., 2013; Kosterina et al., 2012; Pinniger et al., 2006), and we are not aware of this transient phase reported in humans. Analysis of this transient phase by Koppes et al. (2013) yielded a positive association between the active component of RFE and the rate of decay, such that the greater the presumed "active" contribution to RFE, the faster the decay in force to an isometric steady state. This is a useful analysis to identify potential kinetic mechanisms for the

* Correspondence to: Human Performance Laboratory University of Calgary Faculty of Kinesiology 2500 University Drive NW Calgary, AB, Canada T2N 1N4. Tel.: 403 220 4639.
E-mail address: gapower@ucalgary.ca (G.A. Power).

http://dx.doi.org/10.1016/j.jbiomech.2014.08.026
0021-9290/

recently observed greater PFE contribution to RFE in older compared with young adults (Power et al., 2013b; Power et al., 2012b).

Impaired isometric force generating capacity is a consequence of natural adult aging (Power et al., 2014; Power et al., 2013a). Conversely, older adults benefit greatly during active lengthening (Ochala et al., 2006) whereby force production is maintained similar to that of young, or greater, relative to other contraction modes (i.e. a relative maintenance of eccentric strength) (Power et al., 2012a; Roig et al., 2010). Recently, it was identified that following active lengthening, older adults experience higher RFE than young owing to a greater contribution of PFE for the ankle dorsiflexors. For the knee extensors, the RFE was not different between young and old, but in line with the initial observation for the ankle dorsiflexors there was a greater contribution of PFE to total RFE for the older group (Power et al., 2013b; Power et al., 2012b). In addition, after a conditioning stretch, both older and young adults followed the typical exponential decline in force (Edman, 2012), but the time to reach an isometric steady-state force level was longer in the older men. Accordingly, the greater relative contribution of PFE on RFE and a longer time to isometric steady state following active stretch in older adults support the idea that there might be an increased structural viscoelastic contribution to force production following stretch (Edman and Tsuchiya, 1996; Rassier and Herzog, 2005) in this model of aging. Moreover, the decay of force transients to an isometric steady state have not been systematically investigated which could provide mechanistic insight into the history dependence of force production in older adults.

With natural adult aging, the mechanism of elevated RFE indeed may be biased toward a structural constituent and less so to an active cross-bridge-based component. Assuming that a faster decay in the force transients following stretch to an isometric steady state is associated with a greater active contribution (Koppes et al., 2013), it is reasonable to speculate that older adults who appear to rely more on PFE for total RFE (Power et al., 2013b; Power et al., 2012b) may have less of an active contribution to total RFE compared with young. Therefore, we would expect a slower decay in force transients in older adults following stretch owing to a greater reliance on passive (non-active) contributions to RFE.

2. Materials and methods

A post-hoc analysis to fit and characterize the biexponential decay in force transients following stretch was conducted on previously described RFE experiments in old ($n=17$, 77.1 ± 5.8 year, 80.3 ± 8.1 kg, 174.3 ± 6.0 cm) and young men ($n=18$, 25.7 ± 2.6 year, 78.6 ± 11.5 kg, 177.0 ± 5.5 cm) for the ankle dorsiflexors (Power et al., 2012b), and knee extensors (Power et al., 2013b). Only those individuals who participated in both studies were included in the post-hoc analysis.

2.1. Experimental arrangement

As described in detail previously (Power et al., 2013b; Power et al., 2012b), all testing was conducted on a HUMAC NORM dynamometer (CSMi Medical Solutions, Stoughton, MA). For the ankle dorsiflexors, all voluntary and evoked dorsiflexion isometric reference contractions were performed at an ankle joint angle of 40°of plantar flexion (PF) (0° PF referring to 90° between the shank and sole of the foot). Lengthening contractions were performed from 10° to 40° of PF at 15°/s. For the knee extensors (straight knee=180°), voluntary and evoked (i.e., single twitch for voluntary activation determination) knee extension isometric reference contractions were performed at a knee joint angle of 80° of knee extension (KE). Lengthening contractions were performed from 140° to 80° KE at 30°/s.

2.2. Experimental procedures

For the dorsiflexors and knee extensors, a 3–5 s duration isometric baseline MVC was performed. Voluntary activation was assessed using the interpolated twitch technique as described in detail previously (Power et al., 2013b; Power et al., 2012b):

$$\textit{voluntary activation}\ (\%) = \left[1 - \frac{\textit{interpolated } P_t}{\textit{resting } P_t}\right] \times 100\%$$

2.3. Force enhancement

The protocol used to determine RFE involved first a 10 s isometric reference MVC at 40° of PF (dorsiflexors) or 80° of KE (knee extension) followed by 3 min of rest; and secondly an experimental contraction, in which the muscle group was voluntarily activated maximally for 10 s, consisting of a 1 s isometric contraction at the shortened muscle length (10° PF or 140° KE), followed by a 2 s lengthening and ending with a 7 s isometric MVC at the same joint angle as the reference MVC. Passive force enhancement (PFE) was calculated as the difference between resting torques after the experimental stretch and the isometric reference contractions (Fig. 1A).

2.4. Data analysis and statistics

In the previously described studies (Power et al., 2013b; Power et al., 2012b), RFE was calculated as the difference between the mean torque value over 1 s during the reference MVC and the mean torque value for 1 s during the steady state of the MVC following the end of stretch corresponding to the same time interval and joint angle as the reference MVC (Fig. 1A). This difference was expressed as a percentage relative to the isometric reference MVC torque. Off-line, steady-state isometric torque was determined over 6 s following the active stretch; achieving a steady state when there was no statistical difference between mean torque values across adjacent pairs of 1 s torque epochs.

2.5. Force transient analysis

To quantify the force decay following lengthening, the torque data for the first 4 s following the end of lengthening were fit using a biexponential function (Fig. 1B, C).

$$\boldsymbol{f(x) = a_0 e^{-\frac{x}{a1}} + a_2 e^{-\frac{x}{a3}} + a_4}$$

The decay half-life of the first and second exponential rate constant was determined using a decay function:

$$\boldsymbol{N(t) = N_o e^{-t/\tau}}$$

SPSS software (version 21, SPSS Inc. Chicago, IL) was used to perform a one-way analysis of variance (ANOVA) to assess baseline neuromuscular function of the older and young adults. A two-way ANOVA (Age x Muscle) was performed to assess the exponential decay half-life for young and older adults across both muscle groups. The level of significance was set at $\alpha=0.05$. A linear regression analysis was performed to evaluate the relationship and shared variance between the half-life of the exponential decay, RFE and PFE.

3. Results

Participant contractile characteristics are shown in Table 1.

3.1. Decay in force transients to isometric steady state

The goodness of fit for the biexponential curve characterizing the decay in force to the isometric steady state was $R^2 \geq 0.93$ (Table 1). The half-life of the first exponential was two times slower in old than young for both the dorsiflexors and knee extensors ($p < 0.05$; Fig. 2A). The half-life of the second exponential was slower in old than in young for the dorsiflexors ($p < 0.05$), but was not different for the knee extensors ($p > 0.05$; Fig. 2B).

3.2. Regression analysis

The major age-related difference in the biexponential decay in force following active stretch was identified in the first exponential time constant, therefore the half-life of the first exponential was compared with PFE and RFE for the dorsiflexors and knee extensors (Fig. 3). Dorsiflexion RFE showed a significant association with the decay half-life (Fig. 3A; $R^2=0.23$; $p < 0.05$). Knee extension RFE did not share a significant association with half-life (Fig. 3B; $R^2=0.11$; $p > 0.05$). When both the dorsiflexors