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Short report

Shortening-induced torque depression in old men: Implications for age-related power loss

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ABSTRACT

Following active muscle shortening, the steady-state isometric torque at the final muscle length is lower than the steady-state torque obtained for a purely isometric contraction at that same final muscle length. This well-documented property of skeletal muscle is termed shortening-induced torque depression (TD). Despite many investigations into the mechanisms of weakness and power loss in old age, the influence of muscle shortening on the history dependence of isometric torque production remains to be elucidated. Thus, it is unclear whether older adults are disadvantaged for torque and power production following a dynamic shortening contraction. The purpose of this study was to evaluate shortening-induced TD in older adults, and to determine whether shortening-induced TD is related to power loss. Maximal voluntary isometric dorsiflexion contractions (MVC; 10 s) in 8 young (25.5 ± 3.7 years) and 9 old (76.1 ± 5.4 years) men were performed on a HUMAC NORM dynamometer as a reference, and then again following an active shortening of 40° joint excursion (40°PF–0°PF) at angular velocities of 15°/s and 120°/s. Work and instantaneous power were derived during shortening. Shortening-induced TD was calculated and expressed as a percentage by determining the mean torque value over 1 s during the isometric steady state of the MVC following shortening, divided by the mean torque value for the same 1 s time period during the isometric reference MVC. To assess muscle activation, electromyography (root mean square; EMG_{RMS}) of the tibialis anterior (TA) and soleus (SOL) was calculated at identical time points used in assessing shortening-induced TD, and voluntary activation (VA) was assessed using the interpolated twitch technique. Old were 18% weaker than young for MVC, and ~40% less powerful for 15°/s and 120°/s of shortening. Old produced 37% and 21% less work for 15°/s and 120°/s than young, respectively. Furthermore, old experienced 60% and 70% greater shortening-induced TD than young for 15°/s and 120°/s, respectively with similar EMG_{RMS} and VA across all conditions. A significant relationship between shortening-induced TD and instantaneous power was found only at the fast angular velocity for both the old ($R^2 = 0.32$) and young ($R^2 = 0.45$) men. The older men experienced greater shortening-induced TD than young while maintaining similar levels of voluntary activation. This previously unaccounted for history-dependent property of muscle may provide insight into power loss in old age.

1. Introduction

Natural adult aging is associated with a loss of neuromuscular function. One aspect contributing to impaired function in old age is muscle weakness owing to reductions in muscle quality and quantity (Power et al., in press). Factors responsible for weakness include, but are not limited to, decreased voluntary activation, decreased agonist 'central drive', loss of motor units, muscle fibre atrophy, loss of myosin content, and alterations to muscle architecture [for reviews, see (Narici et al., 2008; Power et al., 2013a; Prochniewicz et al., 2007)]. Despite many investigations into the mechanisms of muscle weakness in old age, the influence of history-dependent muscle shortening on isometric torque production has yet to be elucidated. Shortening-induced torque depression (TD) has direct and functional implications on the force-velocity characteristics of muscles (McDaniel et al., 2010; McGowan et al., 2013) and therefore *in situ* power production. Thus, it is unclear whether reduced power and torque production (i.e., weakness) in older adults is compromised during and following a dynamic shortening contraction.

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During a shortening contraction, while contracting actively throughout a functional range of motion and maintaining torque output at the final muscle length, torque production is lower than a purely isometric contraction at that same final muscle length (Abbott and Aubert, 1952). This well-documented property of skeletal muscle is termed shortening-induced TD and is observed at the whole muscle (De Ruiter et al., 1998; Tilp et al., 2009) and reduced muscle preparation level (Abbott and Aubert, 1952; Joumaa et al., 2012) in both animals and humans. The widely accepted mechanism for a torque-depressed state following muscle shortening is a stress-dependent inhibition of cross-bridge attachments in the newly formed actin-myosin overlap zone owing to actin filament angular deformation (Daniel et al., 1998; Marechal and Plaghki, 1979). Also, a stress-induced inhibition of cross-bridges in the previous actin-myosin overlap zone and reduced average force per bridge contributes to total force depression (Joumaa et al., 2012). In line with the above proposed mechanisms, stiffness of the muscle decreases in direct proportion with the amount of shortening-induced TD, indicating a decreased proportion of attached cross-bridges in the TD steady-state phase following shortening (Lee and Herzog, 2003; Sugi and Tsuchiya, 1988). Shortening-induced TD increases with the magnitude of muscle shortening (Edman, 1975) and decreases with increasing shortening velocity (Marechal and Plaghki, 1979; Meijer et al., 1998). Ultimately, shortening-induced TD appears to be proportional to the mechanical work performed during shortening (Herzog et al., 2000; Josephson and Stokes, 1999). Results from a recent study in young adults (Dargeviciute et al., 2013) show greater shortening-induced TD following muscle damage (i.e., muscle weakness) compared to an undamaged muscle. As a consequence, for the same shortening muscle action, in a weakened system, there was greater relative shortening-induced TD despite performing less mechanical work. Thus, older adults who have a weakened neuromuscular system may incur greater shortening-induced TD than young.

Maximal power production is dependent upon an optimal trade-off of velocity and torque, a reduction in either or both will lead to a disproportionately greater loss of power than each variable alone (Power et al., 2013a). Power production in older adults is diminished, especially during fast dynamic contractions (Dalton et al., in press). Impairments in contractile speed appear to be the key contributing factor in explaining compromised power loss with adult aging. Therefore, in order to compensate for slower contracting muscles, older adults must rely more on torque to generate maximal power than do young adults (Dalton et al., in press). If older adults have a greater history-dependent influence on torque production, this could be a potential unexplored mechanism for the disproportionate loss of power in old age.

Therefore, the purpose of this study was to evaluate shortening-induced TD in older and young adults and to determine whether shortening-induced TD is related to power. We hypothesised that the slower and weaker older adults will experience greater shortening-induced TD than the young.

2. Materials and methods

2.1. Participants

All young ($n = 8$, 25.5 ± 3.7 years, 179.8 ± 7.5 cm, 80.1 ± 9.8 kg) and old men ($n = 9$, 76.1 ± 5.4 years, 176.1 ± 6.5 cm, 85.7 ± 10.7 kg) were asked to refrain from unaccustomed and strenuous exercise the day before testing and not to consume caffeine within 2 h prior to testing. All participants were recreationally active with no known neurological or musculoskeletal conditions. The young adults were recruited from the university population and the older adults were recruited from a local senior's group which includes walking, and light calisthenics twice weekly. Participants had been involved in previous experiments in our laboratory and were well familiarized with the procedures and neuromuscular techniques used. This study was approved by the local University Research Ethics Board for Research Involving Human Subjects and conformed to the Declaration of Helsinki. Informed written consent was obtained prior to testing.

2.2. Experimental arrangement

All testing was conducted on a HUMAC NORM dynamometer (CSMi Medical Solutions, Stoughton, MA) as described previously (Power et al., 2012c). The non-dominant foot was fastened tightly to the ankle attachment footplate with inelastic straps, aligning the lateral malleolus with the rotational axis of the dynamometer. Extraneous movements were minimized using non-elastic shoulder, waist and thigh straps. Participants sat in a slightly reclined position with the hip, and knee angles of the experimental leg set at 110°, and 140° (180°; straight), respectively. All voluntary and evoked isometric dorsiflexion contractions were performed at an ankle joint angle of 0° of plantar flexion (PF). Shortening contractions began at 40° PF until 0° of PF, through a 40° excursion.

2.3. Electromyography (EMG)

Electromyography signals were collected using self-adhering Ag-AgCl surface electrodes (1.5 × 1 cm; Kendall, Mansfield, MA) in a monopolar configuration to optimize M-wave recordings. The active electrode was positioned over the proximal portion of the tibialis anterior (TA), and the reference electrode was placed over the distal tendinous portion of the TA at the level of the malleoli. The active electrode for the soleus was positioned 2 cm distal to the lower border of the medial head of the gastrocnemius and the corresponding reference electrode was placed over the calcaneal tendon. The ground was placed over the patella.

2.4. Experimental procedures

Contractions of the dorsiflexors were evoked electrically with a standard clinical bar electrode (Empi, St. Paul, Minnesota, USA), coated in conductive gel positioned to maximize the compound muscle action potential (M-wave) for the purpose of normalizing voluntary EMG. The anode was positioned anterior and the cathode posterior to the fibular head over the deep branch of the common fibular nerve. A computer-triggered stimulator (model DS7AH, Digitimer, Welwyn Garden City, Hertfordshire, UK) set at 400 V provided the electrical stimulation using a pulse width of 100 μs. Peak twitch torque (P_t:1 Hz) was determined by increasing the current until a plateau in dorsiflexor P_t and tibialis anterior M-wave peak to peak amplitude were reached, and then the current was further increased by at least 15% to ensure activation of all motor axons via supramaximal stimulation. This stimulation intensity was used to assess voluntary activation. Additionally, a maximal M-wave, elicited by supramaximal stimulation of the tibial nerve using procedures described above, was evoked from the soleus muscle to normalize antagonist EMG. A commercially available clinical stimulating bar electrode (Chalgren Enterprises Inc., Gilroy, California, United States) was held firmly in the distal portion of the popliteal fossa between the origins of the heads of the gastrocnemeii to electrically activate the tibial nerve where it is readily accessible.

Following maximal twitch determination, participants were familiarized with the dynamic 15°/s (slow), and 120°/s (fast) contractions. To begin, subjects performed 5 contractions at one speed, each contraction separated by 10–15 s, and then at the other speed. This was followed by 5 attempts of two uninterrupted contractions and was determined to be maximal when the torque values for the two subsequent contractions were similar. This was repeated for both velocities, and 3-min rest was given between velocities. After a 5-min rest, three MVCs were performed, each for 3–5 s and separated by 3 min rest. Voluntary activation was assessed using the twitch interpolation technique during the MVCs during which participants were provided visual feedback of the torque tracing on a computer monitor and were exhorted

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