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Review

The influence of strength training on muscle activation in elderly persons: A systematic review and meta-analysis

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ABSTRACT

Age-related muscle weakness is only partially related to muscle atrophy, due to neuromuscular changes including reduced voluntary muscle activation and antagonist muscle co-activation. The respective contribution of these mechanisms in exercise-induced strength gains at higher age is unclear. Here the literature was systematically reviewed for studies reporting exercise-induced effects on voluntary muscle activation and antagonist muscle co-activation in elderly persons. Seventeen relevant studies were identified, 4 investigated voluntary muscle activation, 8 antagonist muscle co-activation and 5 studies investigated both. Meta-analysis showed an exercise-induced improvement in voluntary activation in plantar flexors (weighted mean difference (WMD) +8.8%, $p < 0.001$), and knee extensors (WMD +1.8%, $p < 0.001$), with greater gains in activation capacity obtained in subjects with lower voluntary activation level prior to the onset of training. We found no significant overall effect of strength training on antagonist co-activation during ankle plantar flexion (WMD +0.6%, $p = 0.686$) or knee extension (WMD −1.1%, $p = 0.699$ for the RCT's and −1.8%, $p = 0.516$ for the non-controlled trials). Based on our results we can conclude that there is evidence for exercise-induced increase in voluntary activation related to strength gains in the lower extremities in elderly persons. The results for exercise-induced effects on antagonist co-activation are inconsistent and more research is necessary to determine its contribution to strength gains following resistance training in elderly persons.

1. Introduction

Sarcopenia, defined as the age-related loss of skeletal muscle mass and resulting in an important change in body-composition and function in elderly persons (Rosenberg, 1997), has been extensively described in the literature (Bautmans et al., 2009; Cruz-Jentoft et al., 2010; Fielding et al., 2011; Rosenberg, 2011). Due to differences in approaching sarcopenia in epidemiological studies, the reported prevalence rates vary widely (Bautmans et al., 2009). Recently, clinical criteria for diagnosing sarcopenia-related disability have been proposed (Cruz-Jentoft et al., 2010; Fielding et al., 2011), based on a combination of muscle atrophy, muscle weakness and reduced physical function. Intriguingly, age-related loss of muscle strength is only weakly associated with the reduction in muscle mass (Clark and Manini, 2008; Delmonico et al., 2009; Mitchell et al., 2012). In fact, the decrease in muscle strength is much more rapid compared to loss of muscle mass (Delmonico et al., 2009). The mechanisms explaining this age-related muscle weakness are not yet fully understood. Since sarcopenia originally refers to the age-related muscle atrophy, the term dynapenia has been proposed to describe the age-related loss of muscle strength and power (Clark and Manini, 2008, 2012). Neuromuscular mechanisms that are supposed to be involved in dynapenia are a deficit in maximal voluntary muscle activation and increased antagonist muscle co-contraction. Muscle weakness due to age-related changes in myocyte properties (e.g. muscle fibre atrophy, and Ca^{2+} dysregulation) is beyond the scope of this review and is extensively described in the recent work of Russ et al. (2012).

Age-related changes at the level of the motor cortex and the spinal cord can influence the voluntary muscle activation (Manini and Clark, 2012; Russ et al., 2012). Conflicting evidence exists on alterations in the central nervous system affecting the capacity to fully activate the muscle during maximal voluntary contraction (MVC) at older age (Klass et al., 2007; Manini and Clark, 2012). The twitch interpolation technique is commonly used to assess deficits in the ability to completely activate the skeletal muscle (Merton, 1954). During (usually an isometric) MVC, electrical stimuli are superimposed at the level of the peripheral nerve, thus stimulating the motor axons of the contracting muscle. When the force output is increased by these superimposed

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electrical stimuli, the subject's voluntary activation (the *"completeness"* of skeletal muscle activation during voluntary contraction) is considered to be sub-maximal (Shield and Zhou, 2004). In general, two methods are used to quantify voluntary activation (Shield and Zhou, 2004). One method is to compute the interpolated twitch (IT)-ratio as:

$$IT(\%) = [1-(\text{superimposed twitch}/\text{control twitch})] \times 100 \quad (1)$$

where "superimposed twitch" is the force increment noted during a maximal contraction at the time of stimulation and "control twitch" the force evoked by the same electrical stimulation in the relaxed muscle.

An alternative approach is to express MVC force as a percentage of the total force produced during superimposed stimulation (Kent-Braun and Le Blanc, 1996). This index is known as the central activation ratio (CAR), calculated as:

$$CAR = MVC/(MVC + \text{stimulated force}) \quad (2)$$

where "stimulated force" is the total force generated during the superimposed stimulation.

Another mechanism that may contribute to dynapenia in elderly persons is the co-activation of antagonist muscle(s) during agonist muscle contraction. Co-activation of the antagonist muscle might be useful for joint stabilization, but disproportional co-activation can lower the net force exerted by the agonist muscle, as well as inhibit the voluntary activation of the agonist muscle. The primary spinal coordinator of agonist–antagonist muscle activity is the disynaptic reciprocal inhibition through the Ia inhibitory interneuron (Hortobagyi and Devita, 2006). When agonist Ia afferents are activated, inhibition of the antagonist motor neurons occurs by Ia inhibitory interneurons, causing a smooth movement. A decline in reciprocal inhibition with advancing age is associated with increased antagonistic muscle activity during voluntary movement (Hortobagyi and Devita, 2006). The magnitude of co-activation of the antagonist muscle is most often approached by expressing the EMG activity of the antagonist muscle during agonist contraction, as a percentage of the maximal antagonist muscle EMG activity (i.e. during a maximal contraction) (Kellis, 1998). Studies that measured the influence of ageing on antagonist muscle co-activation during isometric and dynamic contractions have been reviewed previously (Klass et al., 2007). Co-activation appears to be higher in elderly adults during isometric contractions. During MVC's of knee extensors a difference of ~5% between old and young subjects was reported in co-activation of the m. biceps femoris (Izquierdo et al., 1999). A difference of ~20% between older and young women in co-activation of the biceps femoris during knee extension also has been reported (Macaluso and De Vito, 2004). During (isometric) MVC's of the elbow flexors and extensors, a difference in co-activation of the antagonist muscles of respectively ~5 and 8% ($p < 0.01$) was reported (Bautmans et al., 2011; Klein et al., 2001).

Strong evidence shows that resistance training is the most effective strategy to counter and prevent age-related muscle weakness (Liu and Latham, 2009; Macaluso and De Vito, 2004; Peterson et al., 2010). Important strength gains (up to >50%) have been reported, already after a relatively short period (i.e. 6–9 weeks) of strengthening exercise, even in very old persons. Given the rapid strength gains, it is widely accepted that neural adaptations are involved. To date, the respective contribution of changes in voluntary muscle activation and antagonist muscle co-activation in exercise-induced strength gains at higher age remains unclear. The purpose of this study was to review systematically the literature for studies regarding the influence of resistance training on voluntary muscle activation and antagonist muscle co-activation in elderly persons.

2. Material and methods

2.1. Literature search

Pubmed and Web of Knowledge were screened (last search on November 20, 2013) using the following keywords: *(aged [Mesh] OR aged, 80 and over[Mesh] OR frail elderly[Mesh]) AND (resistance training[Mesh] OR exercise[Mesh] OR strength training) AND (muscle, skeletal[Mesh]) AND (voluntary activation OR twitch interpolation OR co-activation OR co-contraction OR antagonist)* for PubMed and *Topic = (elderly) AND (Topic = (resistance training) OR Topic = (exercise) OR Topic = (strength training)) AND (Topic = (muscle) OR Topic = (skeletal muscle)) AND (Topic = (voluntary activation) OR Topic = (twitch interpolation) OR Topic = (co-activation) OR Topic = (co-contraction) OR Topic = (antagonist))* for Web of Knowledge. This action resulted respectively in 84 and 69 articles (Fig. 1). For these articles, titles, keywords and abstracts were screened for relevance. Studies were included if they met the following criteria: written in English, reporting training interventions in healthy subjects aged >60 years (mean age), and outcomes for muscle activation (measured with the twitch interpolation technique or electromyography) and/or calculation of antagonist muscle co-activation. Training intervention was defined as a strength training regimen using external resistance. Papers were excluded if the training intervention did not meet these criteria and if the study population had a specific impairment or medical condition. Randomised as well as semi-randomised controlled trials and non-controlled experimental studies were included. Inclusion and exclusion criteria were applied independently by two reviewers. Disagreement was resolved by discussion and consensus method. This procedure resulted in 17 relevant articles (see Fig. 1).

2.2. Quality assessment

Randomised controlled trials (RCT) investigating the effect of training interventions were assessed using the methodology checklist for RCT's from the National Institute for Health and Clinical Excellence (NICE, Appendix D, 2009). This checklist is designed to assess the internal validity of the study and contains four sections (A–D), each of which addresses a potential source of bias. It concerns selection bias (A), performance bias (B), attrition bias (C) and detection bias (D). All assessments were performed by two independent reviewers. Scores were attributed here as "yes", "no", "unclear" or "not applicable" (Table 1). Papers for which quality assessment resulted in disagreement between raters were reassessed and a consensus based final score was attributed.

2.3. Data extraction

Study populations' characteristics (gender, mean age and range or standard deviation) were identified. Although in some studies young participants were compared to old, the data concerning the older (mean age >60 years) subjects were the main focus in this review. The following data were extracted: training programme (type of exercise, duration, number and frequency of training sessions and repetitions), assessment method of outcome measure, muscle group investigated, exercise-induced changes in the level of voluntary activation and/or antagonist muscle co-activation.

2.4. Data analysis

For data of voluntary activation and antagonist co-activation obtained in (randomised) controlled studies, weighted mean differences between groups (WMD, with 95% confidence interval [95% CI]) were calculated. For data of voluntary activation and antagonist co-activation obtained in non-controlled trials, treatment mean differences (TxMD, change score pre- and post-training with 95% CI) were calculated. Analyses were conducted in OpenMeta[Analyst] software for