Is co-contraction responsible for the decline in maximal knee joint torque in older males?

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Abstract While it is often reported that muscular coactivation increases with age, the mechanical impact of antagonist muscles, i.e., the antagonist torque, remains to be assessed. The aim of this study was to determine if the mechanical impact of the antagonist muscles may contribute to the age-related decline in the resultant torque during maximal voluntary contraction in knee flexion (KF) and knee extension (KE). Eight young (19–28 years old) and eight older (62–81 years old) healthy males participated in neuromuscular testing. Maximal resultant torque was simultaneously recorded with the electromyographic activity of quadriceps and hamstring muscles. The torque recorded in the antagonist muscles was estimated using a biofeedback technique. Resultant torques significantly

decreased with age in both KF (–41 %, $p<0.005$) and KE (–35 %, $p<0.01$). Agonist and antagonist torques were significantly reduced in KF (–44 %, $p<0.05$; –57 %, $p<0.05$) and in KE (–37 %, $p<0.01$; –50 %, $p<0.05$). The torque elicited by double twitch stimulation (–37 %, $p<0.01$) and the activation level (–12 %, $p<0.05$) of quadriceps was significantly lower in older men compared to young men. This study showed that antagonist torques were not responsible for age-related declines in KF and KE resultant torques. Therefore, decreased resultant torques with age, in particular in KE, can primarily be explained by impairments of the peripheral factors (excitation–contraction coupling) as well as by decreased neural agonist activation.

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Introduction

Aging induces several modifications of human motor control. A primary alteration is a decrease in force production capability, notably at the knee joint (Vandervoort and McComas 1986). This production of force is generally quantified by the resultant torque recorded via an ergometer. Simultaneous to the recording of the resultant torque, it has been demonstrated that both agonist and antagonist muscles were activated (Kellis 1998). Consequently, the resultant torque at the knee joint is equal to the difference of torques developed by the agonist (e.g., quadriceps during extension) and the opposite antagonist muscles (e.g., hamstrings). Although the co-contraction or coactivation phenomenon (agonist and antagonist activations) has been mainly described as a protective mechanism at a joint (Baratta et al. 1988; Solomonow et al. 1988), the amount of antagonist co-contraction could also significantly influence the resultant torque.

The coactivation phenomenon is usually examined by assessing the electromyographic (EMG) coactivation level (Kellis 1998). The magnitude of coactivation during maximal voluntary contraction (MVC) is typically assessed by expressing EMG activity in the antagonist muscle as a percentage of its activity when acting as an agonist during a maximal effort. This coactivation level appeared to depend on the training level, the action mode (i.e., eccentric, isometric, concentric), the angular joint, the angular velocity, the muscles studied, the force level, and the age of the participants (Klass et al. 2007; Remaud et al. 2007). As age increases, it has been reported that the coactivation level could be higher than (Izquierdo et al. 1999; Macaluso et al. 2002) or similar to young adults (Morse et al. 2004; Simoneau et al. 2005). While no consensus has been observed in the literature, some recent studies have demonstrated that the coactivation level alone is not adequate to determine the mechanical impact of the antagonist muscles, particularly at the ankle joint (Simoneau et al. 2009; Billot et al. 2010, 2011). This discrepancy can be explained by the linear or curvilinear relationship between the EMG activity and the exerted torque. These studies demonstrated that the coactivation level overestimates the antagonist torque during plantar flexion and

underestimates the antagonist torque during dorsiflexion. Furthermore, as for the coactivation level, previous studies have reported that antagonist torque depends on angular position (Krishnan and Williams 2010; Billot et al. 2011), the muscles involved (Simoneau et al. 2009; Billot et al. 2010, 2011; Krishnan and Williams 2010), and age (Simoneau et al. 2009). As age increases, while Simoneau et al. (2009) indicated that the maximal resultant torque in dorsiflexion was not significantly altered, they showed that the agonist torque significantly declined. A fivefold higher antagonist torque in young participants compared to their older counterparts explained this result. This finding highlights the relevance of investigating antagonist torque in clinical and biomechanics research in order to investigate maximal torques at a joint.

Although some studies have reported possible modifications of the EMG coactivation level at the knee joint with aging (Izquierdo et al. 1999; Macaluso et al. 2002), no indication of change has been reported on the antagonist mechanical impact at this joint. It is not obvious to conclude that an increased EMG signal would induce a significant modification of the antagonist torque as age increases. Therefore, for a similar torque produced by the agonist muscular group, a higher antagonist torque would induce a lower resultant torque. Even if the mechanical impact of the antagonist muscles could be responsible for the decline in maximal torque production with age, this parameter has not been properly quantified yet. As stated in two recent reviews (Clark and Taylor 2011; Clark and Fielding 2012), there is a lack of evidence that proves that antagonist torque contributes to weakness in older adults. It would then be relevant to highlight the proportion of age-related torque decline that could be attributed to alterations in the antagonist mechanical output in knee flexion (KF) and knee extension (KE). In order to determine other factors also responsible for torque decline with aging, the peripheral contractile component of the agonist muscles and the agonist voluntary activation were investigated. Therefore, the main purpose of this study was to identify the possible role of antagonist muscle in torque decline at the knee joint with age. Due to the coactivation phenomenon, it was expected that the decline in maximal torque production with age could partly be due to higher antagonist mechanical contributions during KE and KF MVC in older men.

Materials and method

Participants

Eight young men (age 24.8 ± 3.0 years; height 1.77 ± 0.05 m; body mass 67.6 ± 8.4 kg) and eight older males (age 68.1 ± 7.3 years; height 1.69 ± 0.05 m; body mass 69.5 ± 8.4 kg) volunteered to participate in this study. All older participants were community dwellers and lived independently. Participants had no history of knee surgery or other orthopedic or neurological abnormalities of the lower limb during the 2 years preceding the study. Participants performed recreational physical activities, but none were engaged in regular strength training or competitive sports. All participants met the inclusion criteria purposed for exercise studies on older people (Greig et al. 1994). Written informed consent was obtained, and all experimental procedures conformed to the standards set by the Declaration of Helsinki and were approved by the local Committee on Human Research.

Mechanical and EMG recordings

Torque measurements were recorded using an isokinetic dynamometer (Shirley, NY Biodex 3) during isometric MVC and submaximal contraction in KF and KE. The axis of the dynamometer was aligned with the knee extension axis, and the lever arm was attached to the shank with a strap. Extraneous movement of the upper body was limited by two crossover shoulder harnesses and a belt across the abdomen. To allow the recording of the hamstrings' EMG activity, a board was placed underneath the participant with a hole where the electrodes were positioned so as to avoid any compression of the surface electrodes and wires. Experiments were performed on the right leg with a knee joint angle of 90° of flexion (180° —knee fully extended) and a hip angle of 110° (180° correspond to the anatomical position, fully extended). Surface EMG activities of the vastus lateralis, vastus medialis, rectus femoris for the quadriceps femoris muscles, and the biceps femoris and semi-tendinous for the hamstring muscles were recorded with pairs of silver chloride circular (recording diameter of 10 mm) surface electrodes (Swaromed, Nessler Medizintechnik GmbH, Innsbruck, Austria) positioned lengthwise over the middle of the muscle belly with an interelectrode (center-to-center) distance of 20 mm. Low impedance (<5 k Ω) at the skin–electrode interface

was obtained by shaving and cleaning the skin with alcohol. The ground electrode was attached to the patella of the left knee. Torques and EMG signals were acquired with a sampling frequency of 2 kHz and processed with a multi-channel analog-digital converter (Model MP 150, Biopac Systems Inc., Santa Barbara, CA, USA). The EMG signal was band pass filtered in a range of 10–5,000 Hz and amplified ($\times 1,000$).

To estimate antagonist torque, a validated EMG bio-feedback method was used (Billog et al. 2010). Participants were asked to maintain a level of EMG activity according to the visual biofeedback displayed on a screen: The current agonist EMG activity of the muscle had to correspond with its previously recorded antagonist EMG activity during the MVC. For the EMG bio-feedback task, the root means square (RMS) value of the EMG signal was provided by an integrated circuit, true RMS-to-DC converter (model AD536A, Analog Devices, USA; characteristics: maximal error for true RMS-to-DC conversion=0.5 %, bandwidth>450 kHz). This circuit instantaneously computed the true RMS level of the amplified EMG signal for three channels separately with an integration time of 375 ms. In the present study, the EMG RMS values of vastus lateralis, vastus medialis, and rectus femoris muscles for quadriceps femoris and biceps femoris and semi-tendinous for hamstring muscles were calculated, and the sum of the EMG RMS was done by the circuit for both muscular groups. The circuit displayed the summed EMG RMS value of quadriceps femoris or hamstring muscles on a screen depending on the task the participant had to perform. The EMG RMS value obtained was displayed on a monitor that was located 2 m in front of the participant.

Electrical stimulation

Transcutaneous electrically evoked contractions were induced by using a high-voltage (maximal voltage 400 V) constant-current stimulator (model DS7, Digitimer, Hertfordshire, UK). The femoral nerve was stimulated using a monopolar cathode ball electrode (0.5 cm diameter) pressed into the femoral triangle by the experimenter. The anode was a 50-cm² (10 $\times$ 5 cm) rectangular electrode (Compex SA, Ecublens, Switzerland) located in the gluteal fold opposite the cathode. The optimal intensity of stimulation (i.e., that recruited all knee extensors motor units) was considered to be reached when an increase in the stimulation

intensity did not induce a further increase in the amplitude of the twitch force and the peak-to-peak amplitude of knee extensors compound muscle action potentials (M-wave). The stimulus duration was 1 ms, and the interval of the stimuli in the doublet was 10 ms. Once the optimal intensity was found, it was kept constant throughout the session for each participant.

Experimental procedure

All measurements were carried out in one experimental session for each participant, and the experimental setup is presented in Fig. 1. Prior to performing MVCs in KF and KE, a warm-up for the neuromuscular testing was performed, consisting of two sets of ten KF and KE sub-maximal efforts. After the warm-up, the participants were asked to perform two 5-s isometric MVC in KE and KF. If the variability of the torque exerted was higher than 5 %, a third MVC was performed. During the KE MVC, the twitch interpolation technique was used to determine the activation level (Allen et al. 1995; Behm and Sale 1996). Paired stimuli were delivered 7 s before the KE MVC (control doublet), over the isometric plateau (superimposed doublet) and about 7 s after the isometric KE MVC (potentiated doublet).

Antagonist torque was estimated in four steps: (a) During KE MVC, the EMG activity of the hamstring muscles was recorded, (b) this EMG activity of hamstring was displayed to the participant as a target line

and the real time EMG activity of the hamstring muscle was displayed on the same screen via an oscilloscope, (c) the participant was asked to perform a KF in order to match the real-time EMG activity of the hamstring muscles on the target line, and then (d) KF torque exerted during the maintained EMG activity target was recorded as the antagonist torque of the hamstring muscles during KE MVC. The same procedure was applied during KF MVC to estimate the antagonist torque exerted by the quadriceps femoris (for details, please refer to Billot et al. 2010).

Data analysis

For all participants, MVC was considered as the mean torque attained during the contraction over a 0.5-s period before the doublet stimulation. The maximal voluntary activation level was estimated according to the following formula, voluntary activation level = $[1 - (\text{superimposed doublet amplitude} / \text{potentiated doublet amplitude})] \times 100$ (Behm et al. 1996). After the estimation of the antagonist torque via the EMG biofeedback method, the agonist torque was deduced by the addition of the estimated antagonist torque to the recorded resultant torque. The amplitude of the control doublet and the potentiated doublet was analyzed for both age groups. In the current study, maximal evoked torque elicited by double electrical stimulation was applied to assess muscular capacities of the knee extensors.

Fig. 1 Front view and back view of the experimental setup. The participant was seated with the right leg strapped to the dynamometer (1). The EMG activities of the thigh muscles were recorded with Biopac system (2), and the stimulations were electrically evoked by Digitimer stimulator (3) via a monopolar cathode ball

electrode (4). For the bio-feedback task, the EMG activities were integrated with a true RMS-to-DC converter (5) and displayed to a screen in front of the participant via an oscilloscope (6). The participant had to produce an effort to match the current EMG activity (*solid line*) on the target line (*dashed line*) (7)

In order to calculate the value of the coactivation level, maximal EMG RMS of the antagonist muscles was quantified during maximal agonist muscles over a 0.5-s period after the torque had reached a plateau. In order to compare the EMG coactivation level with mechanical data, we calculated the mechanical ratio corresponding to the antagonist torque normalized to the maximal agonist torque of the same muscle (Billot et al. 2010).

Statistical analysis

All statistical tests were performed with Statistica software (version 6.1, StatSoft, Tulsa, OK, USA). Descriptive statistical methods, including means and their standard deviations (SDs), were calculated for each parameter. The data are presented as means $\pm$ SD in the text, the figures, and the table. Normality of the data was checked using the Kolmogorov–Smirnov test, and equality of variances was verified by the Levene test. Three separate two-way ANOVAs with repeated measures examined the effect of the method (resultant vs. agonist MVC torques), the ratio (coactivation level vs. mechanical ratio), the agonist/antagonist balance (agonist vs. antagonist MVC torques), and their respective interactions with age in KE and in KF. A Student *T* test was performed to analyze the age effect on the antagonist torque, the antagonist/agonist ratio, and the potentiated doublet. A non-parametric Mann–Whitney *U* test was used to examine the age effect on the activation level. The relationships between different parameters were statistically analyzed by a coefficient of correlation defined from the Pearson statistic table. For all analyses, the level of significance was set at $p<0.05$. When a significant main effect was found, a least significant differences Tukey post hoc test was used to identify the significant differences among the selected means.

Results

Resultant, agonist, and antagonist torques

Regardless of the age group, resultant torque was significantly lower than agonist torque for both KF and KE ($p<0.001$). Older men had lower resultant (44.1 ± 9.1 vs. 75.3 ± 26.8 N m, $p=0.040$) and agonist (50.3 ± 11.5 vs.

89.7 ± 25.0 N m, $p=0.013$) KF torques than young men (Fig. 2a). Likewise older men had lower resultant (150.1 ± 34.1 vs. 232.6 ± 58.0 N m, $p=0.026$) and agonist (158.7 ± 37.9 vs. 249.8 ± 53.9 N m, $p=0.008$) KE torques

Fig. 2 Resultant, agonist (*plus sign*), and antagonist (*minus sign*) torques during maximal voluntary contraction (MVC) in **a** knee flexion (KF) and **b** knee extension (KE) for young (*in white*) and older (*in grey*) adults. * $p<0.05$ and *** $p<0.001$ significant difference between young and old adults, respectively. \$ $p<0.001$ significant difference between resultant and agonist torques

compared to young men (Fig. 2b). The antagonist torque appeared to significantly decrease with age for the quadriceps femoris during KF MVC (14.4 ± 6.9 vs. 6.2 ± 4.1 N m, $p=0.012$) (Fig. 2a) and for the hamstrings during KE MVC (17.3 ± 9.9 vs. 8.6 ± 5.0 N m, $p=0.043$) (Fig. 2b).

For KF MVC, a positive linear correlation was found between the agonist and antagonist torques for older adults ($r=0.69$, $p<0.05$), but not for young adults. However, if the two young outlier participants were not considered (black circles on Fig. 3a), a positive linear relationship was found between KF agonist and antagonist torques regardless of the age group ($r=0.76$, $p<0.001$) (Fig. 3a). Similarly, for KE MVC, a positive linear correlation was found between agonist and antagonist torques for older adults ($r=0.79$, $p<0.01$), but not for young adults when two other outliers were considered. If these two young participants were not considered (black circles on Fig. 3b), a positive linear relationship was found between agonist and antagonist torques regardless of the age group ($r=0.84$, $p<0.001$) (Fig. 3b). Considering all participants, the ratio between antagonist and agonist torques was not significantly different between young and older adults in KF (17.3 ± 10.6 vs. 11.8 ± 6.5 %, $p=0.23$) and KE (7.3 ± 4.1 vs. 5.2 ± 2.2 , $p=0.21$).

Coactivation level and mechanical ratio

During KF, although EMG coactivation level and mechanical ratio of quadriceps muscles were not significantly different between young and older adults, the interaction showed that the EMG coactivation level was significantly lower than the mechanical ratio for young adults (4.5 ± 2.7 vs. 6.3 ± 4.3 %, $p=0.040$) and higher than the mechanical ratio for older adults (6.5 ± 2.2 vs. 3.8 ± 2.3 %, $p=0.006$) (Fig. 4).

During KE, no significant difference was obtained for the comparison of EMG coactivation level vs. mechanical ratio of hamstring muscles for both age groups ($p=0.46$), and no significant age effect was found ($p=0.72$). Then, there was no significant interaction between ratio and age ($p=0.34$).

Activation level and potentiated doublet

Results of activation levels and potentiated doublets are presented in Table 1. The activation level of the quadriceps muscles was lower for older than young adults (-10.5 %, $p=0.046$). Muscular parameters indicated

that the potentiated doublet decreased significantly with age (-35.9 %, $p=0.004$).

Discussion

The aim of this study was to investigate the impact of antagonist torque during maximal isometric contractions in KE and KF in older males. The main results showed that antagonist torques did not contribute to weakness with aging for both KF and KE. Regarding the agonist muscles, the peripheral characteristics (assessed via double stimulation) and the central component (assessed via the activation level) appeared to significantly participate in the decrease in agonist KE torque in older men. First, methodological considerations will be debated regarding the quantification of maximal capacity of torque production at the knee joint: Which parameter should preferentially be investigated between resultant and agonist torques? Second, the different components that could be responsible for torque decline observed with age will be discussed: peripheral, agonist activation, and antagonist mechanical impact. Then, specific cases reported in the results will be discussed.

In order to evaluate strength capacity, resultant torque and the estimated agonist torque were assessed with an EMG bio-feedback method. Regardless of the age group, the results indicated that resultant torques underestimated the capacity of torque production, i.e., agonist torque, by ~ 6 % for knee extensors and ~ 14 % for knee flexors. Similar results were found in young males: 5 – 7 % in KE and 16 % in KF (Kellis and Katis 2008; Krishnan and Williams 2010). These findings indicated that the antagonist mechanical impact of quadriceps muscles on KF resultant torque was higher than the impact of hamstrings on KE resultant torque. At the ankle joint, while it has been found that antagonist plantar flexors had an important impact on dorsiflexion resultant torque with a difference of ~ 70 % between resultant and agonist torques, dorsiflexors had negligible impact on plantar flexion (~ 3 %) (Simoneau et al. 2009; Billot et al. 2010). Together, these findings suggest that the gap between agonist and resultant torques could depend on the muscles involved at a joint. Then, depending on the muscular group under investigation, care must be taken when using resultant torque in order to express the maximal strength capacity of an individual.

Fig. 3 **a** Relationship between agonist and antagonist torques for young (*empty squares and black circles*) and older adults (*grey squares*) for **a** knee flexion (KF) MVC and **b** knee extension (KE) MVC. A *full line* represents linear regression for all the participants except the two outliers (*black circles*)

Regarding maximal torques produced at the knee joint, both resultant and agonist torques were significantly lower with aging in KE (−35 and −37 %, $p < 0.01$) and KF (−41 and −44 %, $p < 0.05$). These findings support common results on strength decrease with age at this joint (Aniansson et al. 1986, 1992; Vandervoort

and McComas 1986; Frontera et al. 1991, 2000; Lindle et al. 1997; Hunter et al. 2000; Vandervoort 2002). This strength decline can be explained at different levels: peripheral and/or central.

With regards to peripheral level, it has been reported that the sarcopenia phenomenon has a great impact on

Fig. 4 EMG coactivation level (square) vs. mechanical ratio (circle) for young and old adults. Full square and circle represented the mean of the EMG coactivation level and mechanical ratio,

respectively. * $p < 0.05$ and ** $p < 0.01$ significant difference between EMG coactivation level and mechanical ratio, respectively

force production (Lexell et al. 1988; Frontera et al. 1991; Doherty 2003; Narici and Maffulli 2010). Although the present study did not investigate muscular volume, a significant deterioration in the excitation–contraction coupling due to aging has been found (Vandervoort and McComas 1986; Scaglioni et al. 2003). This observed reduction in the peak of the electrically induced mechanical twitch could result from a slower electrical propagation related to impaired muscle membrane excitability and to a desynchronization of motoneurons. Therefore, as described in the literature,

peripheral components are in part responsible for a decrease in muscular torque with aging (Clark and Fielding 2012). Since voluntary contraction is mediated by neural activation, torque production may not be attributed to peripheral components only.

With regards to the central level, the inability to fully activate muscles in older adults could induce a decrease in torque production. The activation level of the agonist quadriceps muscles significantly decreased with age (−12 %). This result was in agreement with part of the literature (Stackhouse et al. 2001; Stevens et al. 2001) and in disagreement with another part (Roos et al. 1999). In fact, it has been reported that voluntary activation in older adults could depend on the testing methods (De Serres and Enoka 1998) and/or the muscular group tested (De Serres and Enoka 1998; Kent-Braun and Ng 1999; Roos et al. 1999; Bilodeau et al. 2001; Stackhouse et al. 2001; Jakobi and Rice 2002; Morse et al. 2004). As previously reported, the present

Table 1 Activation level and potentiated doublet for young and older adults

	Young	Older	<i>p</i> value
Activation level (%)	94.1±1.9	83.6±11.2	0.046
Potentiated doublet (N m)	100.4±19.6	64.4±22.1	0.004

study showed that the decrease in activation level with age can be one of the parameters of the strength decrease in older men (Stackhouse et al. 2001; Stevens et al. 2001; Klass et al. 2007). Therefore, reduction in KE maximal agonist torque in older adults can be due to both contractile material alterations and neural impairments.

While some reviews reported clear peripheral and neural modifications of agonist muscles with age (Klass et al. 2007; Narici and Maffulli 2010; Clark and Taylor 2011; Clark and Fielding 2012), there is a lack of knowledge about the possible alteration of coactivation phenomenon and its mechanical impact on maximal torque production. The present study showed that the coactivation level was not significantly different with age during KE and KF MVC. Similarly, Macaluso et al. (2002) and Izquierdo et al. (1999), with populations comparable to the present study regarding mean age, activity level, and health status, did not find any age effect on coactivation level for quadriceps muscles during KE MVC. However, they reported that hamstring coactivation level was higher for older adults. This discrepancy with the present data could be explained by the fact that Macaluso et al. (2002) investigated coactivation level in females. Indeed, it has been previously shown sex differences in coactivation levels, with greater values for females than males (Solomonow et al. 1988; Krishnan and Williams 2009). The presence of greater adipose tissue in older women, compared to older men, may have increased the danger of crosstalk and therefore led to inaccurate assessment of coactivation (Solomonow et al. 1994; Lowery et al. 2003). Although age-related modifications in coactivation level is still controversial (Klass et al. 2007), Hortobagyi and Devita (2006) suggested that the mechanisms involved in the EMG coactivation level increase with age at the wrist joint results from a shift in the horizontal inhibitory connection between cortical representation areas of agonist–antagonist muscles and could be a part of the age-related reorganization of cortical structures in response to neurodegenerative processes. These authors explained the age-related augmentation of EMG coactivation level by a significant reduction in reciprocal inhibition. Although EMG coactivation level was largely reported in the literature, it has also been previously reported that EMG activity could not properly estimate antagonist torque at the ankle joint (Billot et al. 2010). To our knowledge, the present study is the first to investigate the mechanical

role of the hamstring and quadriceps muscles between age groups in regard to the EMG coactivation level. In KE MVC, EMG coactivation level and mechanical ratio were not altered with age and were not significantly different. This means that EMG coactivation level can be used to determine the mechanical impact of hamstrings' co-contraction. In KF MVC, while no significant difference was observed in EMG coactivation level with age, it was found that while this parameter was lower than the mechanical ratio for young males, it was higher than the mechanical ratio for older adults. Then, EMG coactivation level should not be used to determine the mechanical impact of quadriceps' co-contraction. Indeed, the use of EMG coactivation to assess antagonist torque during KF would lead to an underestimation in young adults and to an overestimation in older adults. While it has been shown that the antagonist/agonist ratio significantly decreased in dorsiflexion with age (Simoneau et al. 2009), as for plantar flexion, no difference in this ratio has been found with age in both KE and KF. Therefore, the antagonist torque exerted during maximal effort at the knee joint cannot explain, nor contribute, to the age-related decreases in resultant torques. These findings suggested that the agonist–antagonist balance control at this joint was not altered with age. Furthermore, a positive correlation was found between agonist and antagonist torques, suggesting that antagonist torque regulation depends on the amount of agonist torque, regardless of the age group. Moreover, this result reinforced the hypothesis of a common descending drive control for agonist–antagonist muscles, as postulated by De Luca and Mambrito (1987) and Crone and Nielsen (1994).

It seems, therefore, that the higher the agonist torque, the higher the antagonist torque. However, our data did report four specific cases (two in KF and two others in KE MVCs, please see Fig. 3a, b). In KF MVC, while these two particular young persons exerted the lowest agonist torques of their group, they also exerted the greatest antagonist torques. In KE MVC, inversely, while the two other particular cases exerted the greatest agonist torques, they also exerted the lowest antagonist torques of the young group. A hypothesis could be proposed for these particular cases. Although none of the participants were involved in a training program, a possible long-term training impact on the mechanical contribution of the antagonist torque might be evoked. This hypothesis is very speculative and needs to be verified. Currently, the effects of training on

coactivation are controversially discussed. Indeed, it has been reported that EMG coactivation level increased (de Boer et al. 2007), decreased (Carolan and Cafarelli 1992; Hakkinen et al. 1998; Macaluso et al. 2002; de Boer et al. 2007; Tillin et al. 2011), or did not change (Colson et al. 1999; Hortobagyi et al. 2001; Reeves et al. 2003; Morse et al. 2005; de Boer et al. 2007) following a training period. Among these studies, de Boer et al. (2007) showed that EMG coactivation increased for the tibialis anterior muscles, did not change for the gastrocnemius medialis, and decreased for the gastrocnemius lateralis in older women, reinforcing the confusion. These results suggest that training could have different effects on coactivation phenomenon. However, as previously discussed, all these studies investigating EMG coactivation level did not report the mechanical modifications induced by training. Nevertheless, long-term strength training of ankle joint muscles in old age showed that the level of coactivation in plantar flexion first increased from pre- to post-6 months and then decreased from post-6 to post-12 months (Simoneau et al. 2007). This result would be congruent with the fact that the antagonist torque would first increase with the increase in agonist torque (as shown by the agonist–antagonist torques relationship) and then one solution to be even stronger would be to decrease the antagonist torque (over a force threshold). This theory could explain the two particular cases observed in KE MVC. Further investigations are needed in order to examine the effects of training on antagonist torques in both young and older adults. Specifically, it could be assumed that strength training in older adults would induce a shift in their performance toward the performance of the young people. In fact, the value of the agonist torque of the stronger older adult (231 N m) was close to that of the mean of the young group (~250 N m) during KE MVC. It should be noted that this individual was one of the youngest of the older group. However, since there was no significant relationship between age (for older participants) and MVCs (agonist and antagonist torques), the rather wide age range of the older group (62–81 years old) may have had no impact on the present data.

The parameters examined in the present study could be influenced by knee joint angle. Notably, in a previous study, it has been reported that joint position could influence the mechanical impact of the antagonist muscles (Billot et al. 2011). This study showed that the antagonist torque exerted by the dorsiflexors remained

constant regardless of the ankle joint angle, while it was not the case for the torque exerted by the plantar flexors. Since joint angle can have a significant effect on antagonist torque, further investigations are needed to determine this effect at the knee joint. Likewise, the present study should be reproduced with isokinetic contractions because coactivation level is higher in dynamic than in static conditions (Izquierdo et al. 1999). As aforementioned, this investigation has been realized in males only especially because it has already been shown that there is a sex effect on EMG coactivation level (Krishnan and Williams 2009; Solomonow et al. 1988). Further investigations are also needed to determine the sex effect on mechanical contribution of antagonist muscles.

From a clinical point of view, the present study provides reference data for clinicians and/or researchers who are interested in assessing the proper capacity of torque production of muscles around the knee joint. It is worth noting that an identical resultant MVC torque does not imply that the agonist/antagonist balance is the same. Caution must be exercised when considering the conclusions drawn from the common analysis of the sole resultant MVC torque. As a consequence, both agonist and antagonist torques should be taken into consideration, especially because it has been shown that these torques are not negligible. Since antagonist muscles co-activation is one of the adaptation used to compensate for anterior cruciate ligament deficit (Aalbersberg et al. 2005), the assessment of agonist–antagonist torques can be useful in the follow-up, as well as in the design of individualized retraining for these people. As well, it seems that post-stroke patients could exhibit a distortion in the balance between opposing muscles (Klein et al. 2010). It could be then relevant to assess agonist/antagonist torques in healthy population and for those with conditions affecting the knee joint.

In conclusion, the current study suggested that age-related decline in maximal torque production in KE could be attributed to impairment in both contractile properties and activation level of the quadriceps muscles. Contrary to what was expected, antagonist torques had no significant deleterious effect on the capacity of torque production with age and could thus not explain the observed torque declines at the knee joint. In addition, antagonist torque, which can be directly assessed by means of the level of EMG coactivation during KE but not during KF, seemed to be positively correlated to agonist torque. However, it appeared that this was not

always true for every participant. Further investigations are needed to explain these specific behaviors.

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