

The role of age on neuromuscular performance decay induced by a maximal intensity sprint session in a group of competitive endurance athletes

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Abstract

Age-related changes in the neuromuscular system functions may affect profoundly high-level athletes' performance across their careers. The present study aimed to analyse the fatiguing effect of a maximal intensity sprint session (MISS) on competitive athletes of different ages. Thirty-one competitive endurance athletes completed a knee extensors and flexors' maximal-voluntary-isometric-contraction (MVC) test before and after a maximal-intensity-sprint-session (MISS) consisting of 4x15s Wingate-tests. The data have been stratified considering three age categories (18-28, n=11, 29-38; n=10; 39-43, n=10). Overall, both quadriceps and hamstring muscles early and late rate of torque development (RTD) dropped significantly more than the maximal voluntary torque (MVT) ($p<.05$). Age had a significant effect on early RTD, with older athletes exhibiting greater RTD ($p<.05$). A significant effect of age also emerged for the changes in surface sEMG variables, in which the frequency spectrum variables dropped significantly more than the sEMG amplitude (RMS) ($p<.05$). The dynamics of changes in neuromuscular performance markers after a MISS suggested that getting older competitive athletes may potentially experience a greater loss in early explosive strength compared to maximal or late explosive strength.

Key Words: Physical performance; maximal strength; explosive strength; fatigue.

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Advances in human performance analysis and understanding biomechanical and physiological mechanisms and training adaptations brought a trend in career lengthening of competitive level athletes.¹⁻⁴ However, although the career length increases observed in the last two decades, maintaining high physical performance levels in later career phases remains a critical and ambitious goal for the athlete himself and his staff.⁵⁻⁷ In this sense, many factors can be ascribed as possible determinants of the drop in performance across competitive athletes' careers.^{2,5,8}

Among them, age-related changes in the neuromuscular system functions may affect profoundly high-level exercise capacity across the competitive career and potentially determine its length and sustainability.^{8,9} Indeed, ageing is accompanied by a consistent, progressive loss of motor units, changes to the

morphology and properties of existing ones, and altered inputs from the peripheral, spinal, and supraspinal centre.⁹ Such changes may consequently lead to alterations and, in particular, reductions in motor performances and abilities (e.g., drop-in strength and power, increased fatigability).^{9,10} Neuromuscular fatigue, or fatigability,¹¹ has been defined as the exercise-induced decline in the maximal isometric contraction (MVC) force or torque (MVT) of a muscle or muscle group,¹² and both, drops in peak torque during MVCs, and drops in the rate of torque development (RTD) after exercise have been associated with fatigability.¹²

In this regard, age-related changes in neural activation, maximal and explosive strength, as well as a reduction in force or power of a muscle or muscle group in response to exercise (i.e., fatigability) have been described.⁹ In

particular, the RTD seems to be more susceptible to exercise-induced neuromuscular fatigue than maximal voluntary torque (MVT),¹² and more sensitive to detect fatigue response changes induced by age.¹³ Additionally, early RTD (≤ 100 ms from the onset of contraction), largely dependent on motor unit recruitment speed and maximal discharge rate, showed a greater sensibility to exercise-induced fatiguability compared to late RTD (> 100 ms from the onset of contraction), more dependent on muscle-tendon units structural variables.¹² Significant decreases in RTD have been observed after endurance exercise (e.g., running),^{14–16} and after high-intensity intermittent exercise.^{17–20}

In both cases, the drop in RTD, particularly early RTD, exceeded that of MVF.¹² Thus, from one side, drop-in neuromuscular function markers (i.e., MVF and RTD) are reliable indicators of exercise-induced fatiguability, and on the other, to be a valid tool to evaluate the impact of age on the neuromuscular system functions in response to exercise-induced fatigue.^{9,12} Traditionally, coupled to maximal and explosive strength assessment, electromyographic signals are acquired and analysed to evaluate exercise-induced fatiguability.²¹ During maximal all-out exercise, most motor units should be recruited with a characteristic muscle activation pattern in which fast-twitch fibres would be expected to generate a greater proportion of the total work.²² After that, such fibres would become progressively fatigued at a rapid rate, potentially leading to an escalating greater reliance on slow-twitch fibres.²³ Thus, a drop in maximal and explosive strength, changes in muscles recruitment patterns and alteration of firing rate after maximal all-out exercise result in specific fatigue accumulation dynamics, characterised by changes in EMG signals and neuromuscular functions profile.^{12,23} Mean frequency (MNF) and median frequency (MDF) are two useful frequency-domain parameters of EMG analysis, representing the shift of the EMG signal's frequency spectrum, which have been consistently used to detect fatiguability of target muscles/muscle groups.²¹

Taken together, it is conceivable that athletes may experience a different response to exercise-induced fatiguability along with their competitive career, with potential drops in explosive strength and alterations in muscle recruiting patterns and firing rate dynamics. However, although different studies evaluated the impact of exercise-induced fatigue through different exercise protocols, involving athletic or non-athletic populations or investigating the impact of age on exercise-induced fatiguability, less is known about the impact of age on competitive athletes' fatiguability in response to exercise. Therefore, in the present study, we aimed to analyse the impact of a maximal intensity sprint session (MISS) on fatiguability patterns in competitive athletes of different ages, and accordingly, to characterise the possible performance domains subjected to fatiguability in the different age phases of an athlete's competitive career.

Materials and Methods

Study design and participants

In this observational, cross-sectional study, 31 male endurance (i.e., cyclists, runners, and triathletes) competitive athletes (32.8 ± 7.2 years, 78.5 ± 8.7 kg, 181.1 ± 5.1 cm), with a minimum of 5 years of experience in competitive endurance disciplines, fulfilling at least two of the training and race status descriptions of "well-trained athletes" of Jeukendrup et al. (2000)²⁴ and without any past or recent injury involving the lower limbs, voluntarily participated in the study after signing an informed consent form. This research was designed following the Declaration of Helsinki (2008) and the Fortaleza update,²⁵ and was approved by the local institutional review board (Lithuanian Sports University, NR. MNL-SVA(M)-2020-323).

Testing protocol

Participants were familiarised with the testing equipment and measurements during a separate visit to the laboratory 3–4 days before the testing day. The same investigator performed the measurements for all participants. The testing room was kept at 22 °C. Before the warm-up procedures, body composition was analysed using a Tanita-305 body-fat analyser (Tanita Corp., Tokyo, Japan). Self-adhesive preamplified electrodes (PGC10C; Fiab, Vicchio, Florence, Italy) with a 25-mm interelectrode distance were attached to the skin area that had been shaved lightly abraded and cleaned with a sterilised alcohol wipe skin. The electrodes were placed over the dominant leg vastus lateralis, and bicep femoris, and a ground electrode was fixed over the patella, following the SENIAM recommendations.²⁶ Before the start of each testing session, the athlete, performed a warm-up comprising cycling at 100 W for 3 min and 150 W for a further 3 min, followed by 3–5 submaximal isometric contractions before the maximal voluntary isometric contraction (MVIC) test. After this warm-up, the participant completed 3x5 s of knee extensors and flexors MVICs with 60 s recovery between each contraction (Figure 1). MVIC was measured using a Biodex System 3 isokinetic dynamometer (Biodex Medical Systems, Inc., Shiple, NY, USA). The recordings MVIC test were synchronised with the recording of surface electromyographic (sEMG) signals sampled at 1000 Hz through a Biopac 12-bit analogue-to-digital converter system (EL254S; Biopac Systems, Santa Barbara, CA, USA) and AcqKnowledge software (version 4.1, Biopac Systems). After the MVIC test, the subjects completed a maximal-intensity sprint session (MISS) comprised of four maximal Wingate test sprints of 15 s duration interspersed by 2 min of passive recovery. Each sprint was performed on a Wingate cycle ergometer (Monark 894E, Stockholm, Sweden) with a load corresponding to 7.5% of the subject's body mass. The athletes were carefully instructed to sprint as fast and forceful as possible on a given signal from the test

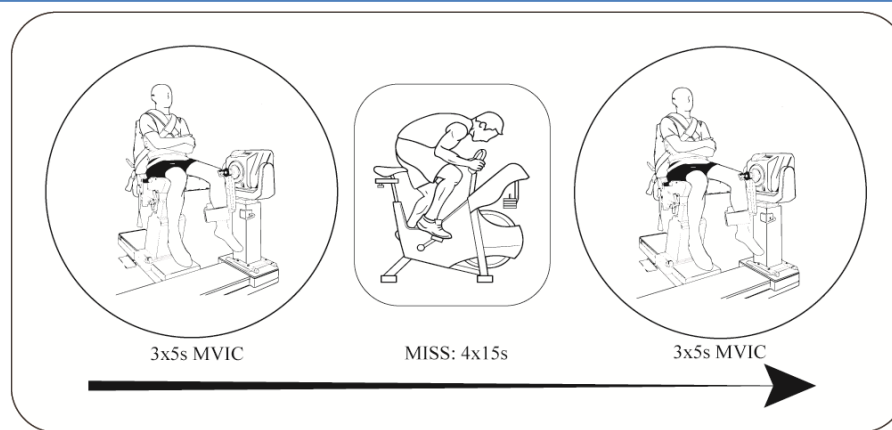


Fig 1. Descriptive representation of the study protocol. The subjects performed in sequence a maximal voluntary isometric contraction (MVIC) test for knee extensors and flexors (3x5s contractions), followed by a maximal intensity sprint session (MISS) on a Wingate cycloergometer and by a second MVIC test for knee extensors and flexors (3x5s contractions).

supervisor and were given strong verbal encouragement by the supervisor during each sprint. After completing the MISS, the subjects repeated the MVIC test following the previously described modality. All the athletes were asked to refrain from any form of physical training in the 48 hours preceding the testing procedures.

Maximal voluntary isometric contraction test

For the MVIC test, each athlete was strapped firmly to a Biodex System 3 dynamometer with two transversal shoulder-to-hip belts fixing the trunk, one hip belt, and one belt at the distal thigh. The athlete was in a seated position with 90° of flexion at the hip and the knee joint at 90° for isometric extensions, and 60° of knee flexion (0° represent full extension) for isometric flexions. The femoral lateral epicondyle was aligned to the dynamometer axis of rotation, and the lower leg was fixed to the lever arm of the dynamometer just above the medial malleolus (Figure 1). The setting was adjusted further to ensure minimal hip and knee joint movement and to minimise vertical displacement between the lower back and the backrest during muscular force exertion. The athlete was carefully instructed to contract as fast and forcefully as possible on a given signal from the test supervisor; the supervisor provided strong verbal encouragement during the test.²⁷ The MVIC was defined as the peak isometric torque (Nm) exerted within the entire contraction phase. Following the procedures described by Maffiuletti et al. (2016),²⁷ the contractile RTD was calculated as the average slope of the torque–time curve (Nm s⁻¹) in the early contraction phase time intervals (0–50 ms and 0–100 ms) and late contraction phase time intervals (0–200 ms and 0–300 ms). The onset of contraction was defined as the time when the knee extensor torque exceeded 2.5% of the difference in the baseline relative to the peak isometric torque.²⁷ MVIC and RTD0–50, RTD0–100, RTD0–200, and RTD0–300

were averaged from the three contractions, and the average value was used for the analyses. All the RTD calculations were performed using a customised Excel spreadsheet using the raw data exported from the AcqKnowledge software.

Surface electromyography signal processing

As previously described, to assess the sEMG activity during the MVIC, the recordings of the MVIC test were synchronised with the recording of sEMG signals sampled at 1000 Hz through a Biopac 12-bit analogue-to-digital converter system (EL254S; Biopac Systems, Santa Barbara, CA, USA) and processed using the AcqKnowledge software (version 4.1, Biopac Systems). sEMG signals were processed via a fast Fourier transformation algorithm, using the EMG power and frequency analysis of the AcqKnowledge software, and with an epoch of 5s covering the time of each isometric contraction. sEMG signals were filtered with a bandpass of 10–500 Hz using a fourth-order Butterworth filter and the EMG amplitude was transformed into a root mean squared (RMS) value by integrating the moving average with 0.03-s width for each isometric contraction.

Statistical analysis

All data were analysed using IBM SPSS Statistics (version 21.0; IBM Corp., Armonk, NY, USA), and GraphPad Prism (version 7.0; GraphPad Software, San Diego, CA, USA) used to produce the graphs. Descriptive statistics (mean ± SD) were calculated for each variable, and data were stratified according to three age categories (AG1: 18–28 years, n=11, AG2: 29–38 years; n=10; AG3: 39–43 years, n=10). The Shapiro–Wilk test was used to assess the normality of the distribution of the samples, revealing normally distributed values. Additionally, Levene's test was adopted to assess the homogeneity of variance for the studied variables indicating a $p > 0.05$. Therefore, a one-

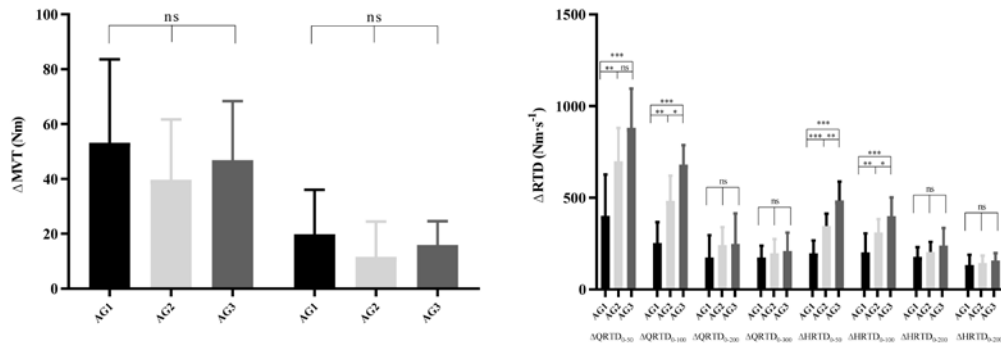


Fig 2. Differences in $\Delta QMVT$ and $\Delta QRTD$, and ΔHMT and $\Delta HRTD$, between the three age groups. Notes: *: $p < 0.05$; **: $p < 0.01$; ***: $p < 0.001$.

way ANOVA was performed to assess the differences in MVT and RTD across the different age groups at baseline and to analyse the differences from pre to post MISS, in MDF, MNF, and RMS values (Δ), of the VL and BF muscles between age groups (ΔMDF , ΔMNF , ΔRMS). A repeated measures ANOVA with ΔMVT vs. ΔRTD as the within subjects' factor and the age category as the between subjects' factor was performed to analyse whether the mean change in the outcome from pre to post MISS differed between ΔMVT and ΔRTD , and to assess the differences between the three age groups. In case of statistical differences, Tukey posthoc analysis was applied. Mean differences across pairwise comparison with 95% confidence intervals (95%CI) were also calculated. Additionally, partial eta squared (η_p^2) was used as ANOVA effect size and interpreted as: (<0.039 – no effect; 0.04 to 0.24 – minimum; 0.25 to 0.63 – moderate; >0.64 – strong (Ferguson, 2009)).²⁸ Pearson correlational analysis with linear regression was used to identify relationships between the criterion variable (age) and the neuromuscular performance decay variables (ΔMVT and ΔRTD). The following criteria were adopted to interpret the magnitude of correlations between measured variables: <0.09 , trivial; 0.10 to 0.29, small; 0.30 to 0.49, moderate; 0.50 to 0.69, large; 0.70 to 0.89, very large; and >0.90 , nearly perfect.²⁸ An alpha level of $p \leq 0.05$ was considered to be significant.

Results

Non-significant differences emerged for the MVT and RTD variables between the three age groups at baseline ($p > 0.05$), while significant differences emerged between $\Delta QMVT$ (46.8 ± 24.9 Nm) and $\Delta QRTD$ [$\Delta QRTD_{0-50}$ (652.6 ± 285.7 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.91$ – strong; $\Delta QRTD_{0-100}$ (465.45 ± 212.9 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.93$ – strong; $\Delta QRTD_{0-200}$ (220.12 ± 131.3 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.70$ – strong; $\Delta QRTD_{0-300}$ (192.9 ± 80.1 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.78$ – strong], and between ΔHMT (15.93 ± 13.0 Nm) and $\Delta HRTD$ [$\Delta HRTD_{0-50}$ (338.21 ± 285.7 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.94$ – strong; $\Delta HRTD_{0-100}$

(300.6 ± 122.9 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.92$ – strong; $\Delta HRTD_{0-200}$ (206.3 ± 71.6 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.90$ – strong; $\Delta HRTD_{0-300}$ (144.8 ± 45.7 Nm·s⁻¹): $p < 0.001$, $\eta_p^2 = 0.89$ – strong]. A significant effect of age on the comparison between ΔMVT and ΔRTD emerged. In particular, $\Delta QRTD_{0-50}$ resulted significantly greater than $\Delta QMVT$ [$p < 0.01$, $\eta_p^2 = 0.54$ – moderate; AG1 vs. AG2: $p = 0.007$, mean difference = -141.6 Nm·s⁻¹, 95%CI (-240.4 to 42.8); AG1 vs. AG3: $p < 0.001$, mean difference = -236.7 Nm·s⁻¹, 95%CI (-335.5 to 138.0); AG2 vs. AG3: $p = 0.64$, mean difference = -95.1 Nm·s⁻¹, 95%CI (-196.2 to 5.9)], $\Delta QRTD_{0-100}$ resulted significantly greater than $\Delta QMVT$ [$p < 0.01$, $\eta_p^2 = 0.71$ – strong; AG1 vs. AG2: $p = 0.01$, mean difference = -108.1 Nm·s⁻¹, 95%CI (-163.9 to 52.3); AG1 vs. AG3: $p < 0.001$, mean difference = -210.1 Nm·s⁻¹, 95%CI (-265.9 to 154.3); AG2 vs. AG3: $p = 0.01$, mean difference = -102.0 95%CI (-159.1 to -44.8)], $\Delta HRTD_{0-50}$ resulted significantly greater than ΔHMT [$p < 0.01$, $\eta_p^2 = 0.70$ – strong; AG1 vs. AG2: $p = 0.001$, mean difference = -70.0 Nm·s⁻¹, 95%CI (-107.1 to 33.0); AG1 vs. AG3: $p < 0.001$, mean difference = -142.3 Nm·s⁻¹, 95%CI (-179.4 to -105.2); AG2 vs. AG3: $p = 0.001$, mean difference = -72.2 Nm·s⁻¹, 95%CI (-110.1 to -34.3)], and $\Delta HRTD_{0-100}$ resulted significantly greater than $\Delta QMVT$ [$p < 0.01$, $\eta_p^2 = 0.48$ – moderate; AG1 vs. AG2: $p = 0.027$, mean difference = -50.6 Nm·s⁻¹, 95%CI (-95.1 to 6.1); AG1 vs. AG3: $p < 0.001$, mean difference = -96.7 Nm·s⁻¹, 95%CI (-141.2 to -52.2); AG2 vs. AG3: $p = 0.048$, mean difference = -46.0 Nm·s⁻¹, 95%CI (-91.6 to 0.49)] (Figure 2). By contrast, no differences between the three age groups emerged between ΔMVT , ΔRTD_{0-200} and RTD_{0-300} ($p > 0.05$).

A significant effect of age on $\Delta QMDF$ ($F_{(2,28)} = 8.825$, $p < 0.001$, $\eta_p^2 = 0.38$ – moderate), $\Delta QMNF$ ($F_{(2,28)} = 5.979$, $p = 0.007$, $\eta_p^2 = 0.30$ – moderate), $\Delta HMDF$ ($F_{(2,28)} = 5.192$, $p = 0.013$, $\eta_p^2 = 0.27$ – moderate), and $\Delta HMNF$ ($F_{(2,28)} = 4.954$, $p = 0.025$, $\eta_p^2 = 0.26$ – moderate) emerged. In particular, the $\Delta QMDF$ was significantly greater for AG2 (5.9 ± 1.4 Hz; $p = 0.006$), and AG3 (6.3 ± 1.7 Hz; $p = 0.002$), compared to AG1 (3.8 ± 1.2 Hz).

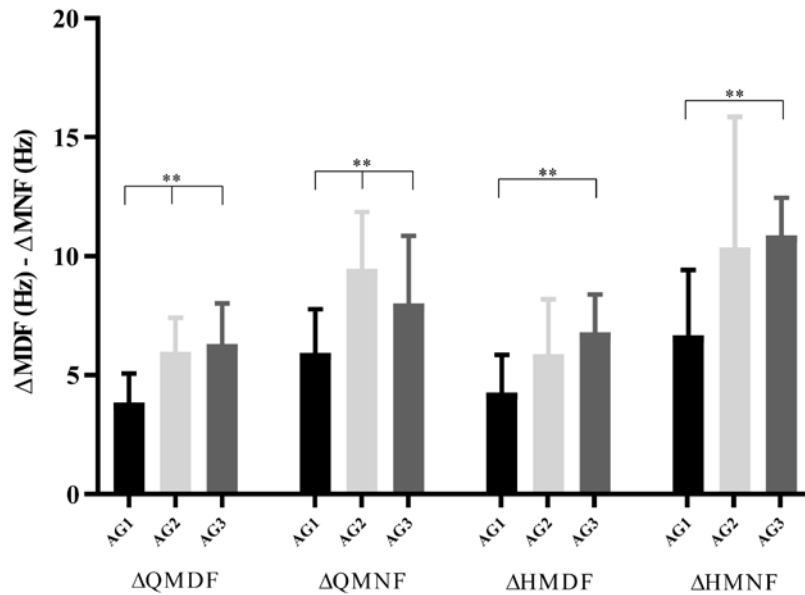


Fig 3. Differences between $\Delta QMDF$ and $\Delta QMNF$, and between $\Delta HMDF$ and $\Delta HMNF$. Notes: **: $p < 0.01$.

The $\Delta QMNF$ was significantly greater for AG2 (9.4 ± 2.3 ; $p = 0.005$), and AG3 (8.3 ± 2.6 Hz; $p = 0.025$), compared to AG1 (5.9 ± 1.8 Hz) (Figure 3). The $\Delta HMDF$ was significantly greater for AG3 (6.8 ± 1.6 Hz; $p = 0.011$), compared to AG1 (4.2 ± 1.5) (Figure 3). The $\Delta HMNF$ was significantly greater for AG3 (10.8 ± 1.6 Hz; $p = 0.034$), compared to AG1 (6.6 ± 2.7 Hz) (Figure 3). By contrasts, no significant differences emerged for $\Delta QMDF$ and $\Delta QMNF$ between AG2 and AG3 ($p > 0.05$), for $\Delta HMDF$ and $\Delta HMNF$ between AG1 and AG2 and between AG2 and AG3 ($p > 0.05$), and no differences between the three age groups emerged for the knee extensors and flexors RMS values ($p > 0.05$).

Moderate positive relationships emerged between age, $\Delta QRTD_{0-50}$ [$p < 0.01$; $r = 0.60$ (95%CI: 0.31 to 0.78); $R^2 = 0.36$], and $\Delta HRTD_{0-100}$ [$p < 0.01$; $r = 0.63$ (95% CI: 0.35 to 0.80); $R^2 = 0.40$]. Large positive relationships emerged between age, $\Delta QRTD_{0-100}$ [$p < 0.01$; $r = 0.79$ (95%CI: 0.60 to 0.89); $R^2 = 0.62$], and $\Delta HRTD_{0-50}$ [$p < 0.01$; $r = 0.82$ (95% CI: 0.66 to 0.91); $R^2 = 0.68$] (Figure 4). By contrast, trivial to moderate correlations emerged between age and ΔMVT values.

Discussion

In the present study, we aimed to analyse the impact of a maximal intensity sprint session (MISS) on fatiguability patterns of competitive endurance athletes categorised in three different age groups and characterise the possible performance domains more subjected fatiguability according to the different career age-phases of competitive athletes. The main findings from the present

investigation are that endurance athletes in later phases of their career may experience a greater drop in explosive strength than maximal strength in response to maximal intensity exercise. In addition, in older athletes we observed greater decreases in EMG frequency values (MDF and MNF) compared to youth counterparts, while no significant differences emerged for the EMG amplitude values (RMS). The present results are in line with previous studies that reported either a significant reduction in explosive power,¹² or a shift towards lower values of MDF and MNF in response to maximal intensity sprint exercises.^{23,29,30} Indeed, the RTD, and in particular early phases RTD, largely dependent on motor unit recruitment speed and maximal discharge rate, seems to be more susceptible to exercise-induced neuromuscular fatigue than MVT,¹² as well as more sensitive to detect fatigue response changes induced by age.¹³ Significant decreases in RTD have also been consistently reported after high-intensity intermittent exercise¹⁷⁻²⁰, with the drop in RTD, particularly early RTD, exceeding that of MVT.¹² The reduction in EMG frequency variables may be linked to increases in intramuscular acidosis and a concomitant slowing muscle fibres conduction velocity.³⁰ However, interpretation of the EMG frequency spectrum variations should be made with caution, as they represent indirect measures of physiological variables.³¹ Taken together, both the significant drops in early RTD and the variations in the EMG frequency spectrum observed due to high-intensity sprints exercise-induced fatiguability are in line with previous findings. However, the present results

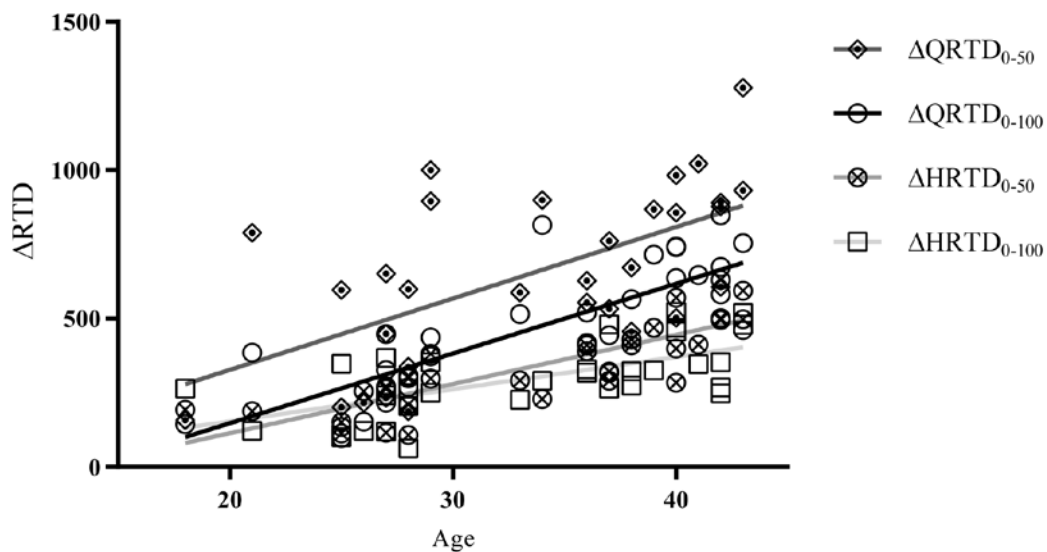


Fig 4. Scatter plot showing the linear regression between age and early Δ RTD.

additionally suggest how older athletes may be more susceptible than their younger counterparts in experiencing drops in early phase RTD and reductions in EMG frequency spectrum variables. Although it has been suggested that consistent motor units remodelling accompanies ageing, and altered inputs from peripheral, spinal, and supraspinal centres, less have been reported regarding the effect of the ageing process on the performance domains of competitive athletes across their career.⁹ Changes in neuromuscular functions may potentially lead to alterations and, in particular, reductions in motor performance (e.g., drop-in strength and power, increased fatigability),⁹ and the results of the present investigation seem to suggest how older athletes may experience increased fatigability of the explosive neuromuscular components and variations in the frequency spectrum of the EMG. Muscle fibres of old adults typically exhibit reduced contractile speed compared to those of young adults,⁹ nevertheless, our results suggest that older athletes may display similar explosive and maximal strength at baseline. However, maximal intensity exercise can impact to a greater extent contractile velocity. Age-related slowing of whole muscle seems to be accompanied by reductions of myosin heavy chain II fibres (MHC II) and a lower maximal shortening velocity of single muscle fibres even in myosin heavy chain I fibres (MHC I) in very old and inactive old subjects.^{9,32,33} Competitive athletes may in part overcome the age-related decline in neuromuscular performance by keeping a constant stimulus to the neuromuscular system through training.^{34,35} However, a progressive physiological age-related decline in performance is expectable also in high-level athletes.^{6,36,37} Indeed, we observed similar baseline

values in neuromuscular performance markers among the three age groups, while a significantly greater drop in explosive strength for older athletes emerged only after the MISS. Exercise-induced fatigability has been related to a decrease in neural activation of the muscle (i.e., central fatigue) and biochemical changes at or distal to the neuromuscular junction, causing attenuated contractile responses to neural input (i.e., peripheral fatigue), and expressed as a reduction in force of power of a muscle/muscle group in response to exercise.⁹ In particular, age difference in fatigability seems to depend on changes in neuromuscular functions as an altered contraction velocity.⁹ The mechanism for the velocity-dependent age difference in fatigability onset look to involve processes that are associated with contractile speed and inadequate muscle activation during fast contractions, contributing to increased variability of motor output across fatiguing tasks as well as potential drops in explosive strength.⁹ Typical neuromuscular responses during short duration (e.g., < 1 min) all-out exercises as cycling have been consistently documented, with fatigue determined by a decline in muscle pH and inhibition of anaerobic metabolism.²³ Anaerobic metabolism is well developed and sustained by MHC II fibres. The reduction in power output during a maximal intensity sprint or a MISS and the consequent drop in muscle explosive and maximal strength is predominantly due to declining contribution from MHC II fibres.^{9,23,32,33} The reduction in fibre type contribution also suggests altered motor unit recruitment and, therefore, the possible consequences on neuromuscular activity during maximal intensity exercise. The high pedalling rate sustained during maximal intensity cycling sprints further support the hypothesis by which the primary contribution during

a cycling MISS may come from MHC II fibres and only later on sustained by MHC I.²³ This, coupled to the observation suggesting an age-related decline MHC II fibres and slower maximal shortening velocity also in MHC I fibres, may explain the greater performance drop, after the MISS, observed for the explosive strength in older athletes. In addition, since the early RTD components are largely dependent on motor unit recruitment,¹² our results may suggest that muscle fibres slower shortening velocity and changes in motor unit recruitment and discharge rate in older athletes behind the greater drops in explosive strength we observed. Cifrek and colleagues (2009)³⁸ outlined how decreased muscle force coincides with decreased EMG amplitude and a leftward shift in the EMG spectrum. Accordingly, we observed how older athletes presented significantly greater leftward shifts in EMG frequency, but non-significant trends in RMS, suggesting a significant greater impact of MISS on fatiguability. Overall, the present investigation emerged that older competitive athletes might maintain a high level of maximal force but suffer from a greater drop in explosive power and fatiguability. The present results may be of practical relevance across their career. Athletes may consider polarising or including training to stimulate explosive strength adaptations and monitor the fatiguing response to different efforts. The main limitation of the present study resides in its cross-sectional design, by the impossibility to recruit older athletes than 45 years old and by the lack of deeper direct analysis of aspects such as muscle fibre conduction velocity or measurement of physiological variables linked with muscle damage and fatiguability. In this regard, future studies may aim to involve larger populations with wider age spectrums and involve training interventions targeting explosive strength adaptations and evaluating the possible impact on neuromuscular functions and fatiguability in senior/older competitive athletes.

In conclusion, older athletes experienced greater explosive drops than maximal strength in response to maximal intensity exercise bouts among competitive endurance athletes. In addition, older athletes displayed a more remarkable leftward shift in the EMG frequency spectrum, suggesting greater force losses and fatigue accumulation than younger counterparts. Endurance athletes may thus consider deeper monitoring and training strategies to sustain explosive strength across their career to overcome the ageing effects on neuromuscular functions and maintain a high-performance level.

List of acronyms

MDF - Median frequency
MISS - Maximal intensity sprint session
MNF - Mean frequency
MVC - Maximal voluntary contraction
MVF - Maximal voluntary force
MVIC - Maximal voluntary isometric contraction

MVT - Maximal voluntary torque
RMS - Root mean squared
RTD - Rate of torque development
MHC I – Myosin heavy chain I
MHC II – Myosin heavy chain II

Contributions of Authors

Conceptualisation LC, DS; methodology, LC, DS, NE; software, LC, DS; validation, LC, DS, GC, AI; formal analysis, LC, DS; data curation, LC, DS, NE, AI, GM. Writing—original draft preparation, LC; writing—review and editing LC, DS, NE, AI, GM; visualisation, LC, DS; supervision, DS. All authors have read and agreed to the published version of the manuscript.

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Conflict of Interest

The authors declare no conflict of interest.

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Ethical Publication Statement

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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