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# Reduced knee extensor torque production at low to moderate velocities in postmenopausal women with knee osteoarthritis

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This study aimed to determine deficits in knee extensor muscle function through the torque-time and torque-velocity relationships and whether these deficits are associated with reduced functional performance in postmenopausal women with knee osteoarthritis (KOA). A clinical sample of postmenopausal women with established KOA ($n = 18$, ≥55 years) was compared to an age-matched healthy control sample (CON) ($n = 26$). The deficits in different parameters of the knee extensor torque-time (maximal isometric torque and rate of torque development) and torque-velocity relationship (maximum muscle power, maximal velocity and torque at 0–500°·s$^{-1}$) were assessed through a protocol consisting of isometric, isotonic and isokinetic tests. Functional performance was evaluated with sit-to-stand and stair-climbing tasks using a sensor-based technology (ie, time- and power-based outcomes). Postmenopausal women with KOA showed reduced maximal isometric torque (Hedge's $g$ effect size ($g$) = 1.05, $p = 0.001$) and rate of torque development ($g = 0.77$–$1.17$, all $p \leq 0.02$), combined with impaired torque production at slow to moderate velocities ($g = 0.92$–$1.70$, $p \leq 0.004$), but not at high or maximal velocities ($g = 0.16$, $p > 0.05$). KOA were slower ($g = 0.81$–$0.92$, $p \leq 0.011$) and less powerful ($g = 1.11$–$1.29$, $p \leq 0.001$) during functional tasks. Additionally, knee extensor deficits were moderately associated with power deficits in stair climbing ($r = 0.492$–$0.659$). To conclude, knee extensor muscle weakness was presented in postmenopausal women with KOA, not only as limited maximal and rapid torque development during isometric contractions, but also dynamically at low to moderate velocities. These deficits were related to impaired functional performance. The assessment of knee extensor muscle weakness through the torque-time and torque-velocity relationships might enable individual targets for tailored exercise interventions in KOA.

**KEYWORDS**

aging, force–velocity relationship, muscle power, muscle weakness, physical disability, rate of force development

## 1 | INTRODUCTION

Knee osteoarthritis (KOA), the most prevalent form of chronic arthritis, is characterized by an abnormal remodeling of joint tissues, involving inflammatory processes and leading to pain and functional disability.[1] Knee extensor muscle weakness in particular seems to be an important determinant of symptomatic and functional decline in people with KOA[2] and gains in knee extensor strength partially mediate pain-relief and subjective physical function.[3] Major changes have been observed regarding muscle morphology (eg, reduced muscle mass, atrophy of type II muscle fibers, increase of intramuscular fat and connective tissue)[4–7] and function (eg, declined maximal isometric, concentric and eccentric force) in people with KOA compared to healthy controls.[8] Changes in muscle function might be especially relevant in postmenopausal women since age and female sex have been proposed as independent factor for clinical KOA progression.[9]

Recently, due to its great relevance in daily tasks (eg, stair climbing), the ability to rapidly exert force during isometric (ie, rate of force development, Δforce/Δtime) and dynamic actions (ie, muscle power, the product of force and velocity) have gained research attention.[10–12] However, most of these studies only addressed a single point of the corresponding force-time and force-velocity curve (eg, maximal isometric force or maximum muscle power). This approach does not allow examining whether knee extensor weakness in KOA is influenced by movement velocity or contraction duration and how this affects physical function. Exploring the full force-time and force-velocity relationship in KOA would enhance our understanding on knee extensor muscle weakness. Both a deficit in force and in velocity will have an impact on muscle power production,[13] but a detailed evaluation of both components would allow for more tailored intervention strategies.

Furthermore, physical function outcomes have primarily been obtained from either self-reported questionnaires[11,12] or from manually recorded time required to perform the task.[10,11] Instrumented measurements of functional movements (eg, sit-to-stand from a chair), in which body-fixed sensors are used to automatically detect sub-durations, allow for a more detailed quantification of the movement strategy[14] and appear to have greater clinical relevance than manual recordings.[15] In addition, sensors can use trunk kinematics to accurately measure power production during sit-to-stand transitions and stair climbing,[16] which appears more clinically relevant than time-based assessments to evaluate functional trajectories among older adults or mobility-limited populations.[17,18] Studies in KOA should therefore include detailed assessments of functional movements, both in terms of movement strategies and sub-durations as well as power production, to better comprehend potential deficits caused by KOA and to set targets for physical therapy to improve functional performance. For example, people with advanced KOA seem to adapt their movement strategy during a sit-to-stand transition to reduce knee moment arm in favor of using the hip extensor muscles.[19] This might point out a particular limitation in the knee extensor muscles, demanding more focus on these muscles in physical therapy.

The current study compares postmenopausal women with KOA and age-matched controls on (I) knee extensor muscle function through the evaluation of both the force-time and force-velocity relationships and (II) instrumented measurements of (sub)-durations and power during functional performance tasks. In addition, the relationship between hypothesized knee extensor muscle weakness and deficits during functional tasks will be explored. To the best of our knowledge, this is the first study investigating the full force-time and force-velocity relationship of the knee extensor muscles in KOA. This approach offers a broader view on potential deficits, allowing for more tailored exercise interventions (eg, selecting the most optimal training load and velocity). Sit-to-stand and stair-climbing tasks were employed as functional performance tasks, in line with the recommendations of the Osteoarthritis Research Society International (OARSI).[20]

## 2 | METHODS

### 2.1 | Experimental approach and study participants

This cross-sectional study was conducted at KU Leuven, in collaboration with University Hospitals Leuven (UZ Leuven), in September 2018. The study was approved by the ethical committee research UZ/KU Leuven in accordance with the Declaration of Helsinki. Women with diagnosed KOA, as defined by the American College of Rheumatology criteria including a Kellgren-Lawrence (K-L) classification grade of ≥2 (on a scale of 0, normal to 4, severe),[21] were recruited at UZ Leuven (KOA). As a control group (CON), age-matched healthy women (ie, asymptomatic and no history of knee pain) were recruited through a previous cohort study[16,22] and using advertisements in the local community. The following exclusion criteria were set: <55 years old, systematic participation in strength and/or endurance training in the past 6 months, knee or hip prosthesis, unstable cardiovascular disease, known neurological condition, acute back or knee problems (the latter only for CON), and any physical or cognitive impediment that makes understanding or performance of a test impossible. An a priori sample size calculation was conducted using GPower (version 3.1.9.2) based on the following parameters: effect size = 0.93 (based on a previous study about the reduced rate of torque development of the knee extensors in older adults with KOA),[23] $\alpha = 0.05$, power $(1-\beta) = 0.8$, allocation ratio = 1. This resulted in a required sample size of 20 participants per group (actual power = 0.82). Initially, 18 eligible women with KOA and fifteen healthy controls of a previous cohort study[16,22] accepted to participate. After noticing a significant age difference between groups, ten postmenopausal women were additionally recruited for CON. All participants provided written informed consent. Participants' flow chart is presented in Figure 1.

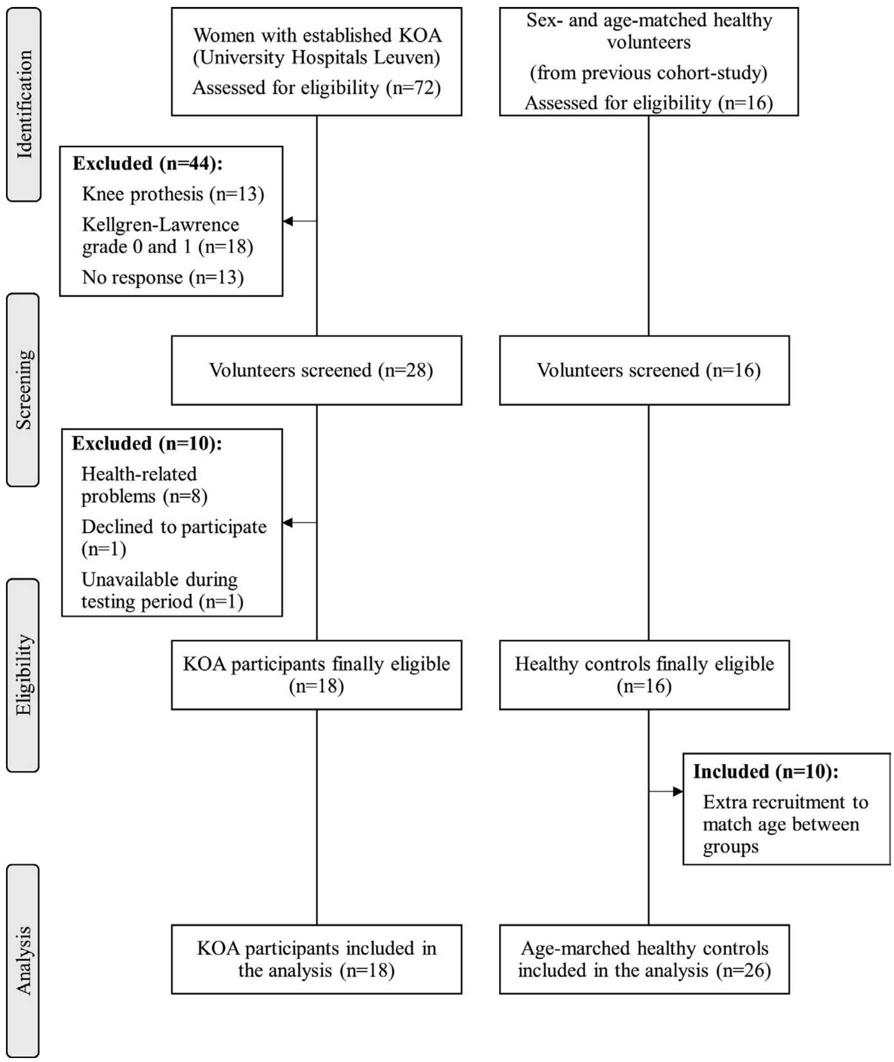


**FIGURE 1** Flow diagram of participants' recruitment, screening, eligibility and final sample size for statistical analysis

## 2.2 | Outcomes

### 2.2.1 | Anthropometry

Body height and weight were determined using a stadiometer (Holtain, Crymych, UK) and a digital scale (Seca, GmbH & Co. KG, Germany), respectively. All participants wore minimal clothing and were barefoot. Body mass index (BMI) was calculated as body mass (kg) divided by squared body height (m).

### 2.2.2 | Functional performance

To evaluate the functional performance of participants, the short physical performance battery (SPPB) was conducted.[24] Additionally, an instrumented 6-step stair climb test (SCT) and five repetition sit-to-stand test (STS) were conducted as described elsewhere.[16] The participants were asked to execute both tests as fast as possible without using the arms/handrail. Two trials were registered for both tests. During these tests, data were recorded by means of a body-fixed sensor (DynaPort MoveTest, McRoberts BV, The Hague, NL). This portable inertial sensor, fixed by an elastic band around the waist and adjacent to the lumbar spine, combines a tri-axial gyroscope and accelerometer. Previous studies have demonstrated the sensor's accuracy and discriminative validity in repeated STS tests.[25,26]

*Signal and data processing*

The sampling rate was set at 100 Hz and commercially available software (DynaPort MoveTest, McRoberts, The Hague, NL) was used to analyze the data. Methods have been described previously.[15,16] For the SCT, the rise (vertical velocity >0.1 m $s^{-1}$) and stance (vertical velocity <0.1 m $s^{-1}$) phase of each step was determined. Total ascent duration, total ascent rise duration and total ascent stance duration (s) were calculated by summation of the six rise and/or five stance durations (no stance duration in final step). Mean power (watt) was assessed for each single rise phase. The highest mean power output (for the power variable) and the trial with the lowest total ascent duration (for the duration parameters) were used in the analyses. Excellent reliability for this method was reported, with ICCs ranging from 0.93 to 0.94 and SEM (%) from 4.0 to 6.3.[16] For the STS, the total duration (s) was determined from movement initiation until the fifth standing position. In addition, the duration of the subphases (ie, sit-to-stand transition, stand, stand-to-sit transition, sit) was defined.[26] The mean power and the trunk flexion range of movement during the STS transition of each repetition were calculated. A greater trunk flexion reduces the demand on the knee extensor muscles[27] and this movement strategy has been observed in weaker compared to stronger older adults[14] and in people with advanced KOA.[19] The highest mean power output (watt), the overall mean trunk flexion range (°) and the trial with the lowest total STS duration (for duration parameters) were used in the analyses.

### 2.2.3 | Knee extensors torque-time and torque-velocity relationship tests

All the knee extensors muscle function tests were carried out on a Biodex Medical System 3® dynamometer (Biodex Medical Systems, Shirley, NY), as described elsewhere.[28] Torque was used as the rotational equivalent of linear force. Briefly, the protocol consisted of four

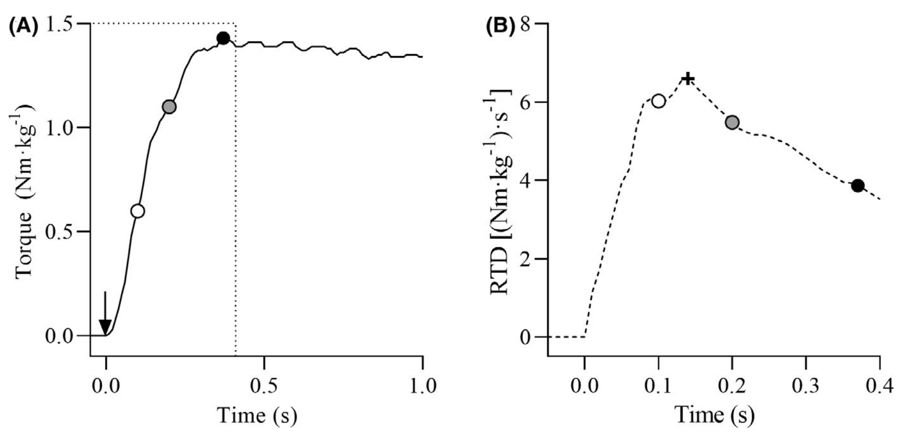


**FIGURE 2** Torque-time curve (solid line) from a representative individual of KOA obtained from an explosive isometric contraction (A). The rate of torque development at 100 ms ($RTD_{100}$, white circle), 200ms ($RTD_{200}$, gray circle) and the corresponding peak value ($RTD_{peak}$, plus sign) were extracted from the first derivative of the torque-time curve (dashed line, panel B). The black arrow indicates the onset of torque production and the black circle correspond to the maximal isometric torque

standardized tests: an isometric, an isotonic, an isokinetic and an isometric explosive test. For the dynamic tests, the preset range of motion was 90° to 160° knee-joint angle (180° full extension). All participants were able to move in this range of movement. Firstly, participants executed four 5-s maximal voluntary isometric ramp contractions at a knee-joint angle of 90°, separated by a 20-s rest interval. For the isotonic test, participants performed four ballistic contractions against four different loads in the following fixed order: 40%, 20%, 0% and 60% of the maximal isometric torque ($T_{max}$, detailed below). The loading conditions were separated by a 20-s rest interval. For the isokinetic test, three maximal consecutive extension-flexion movements were performed at 60°·s$^{-1}$. Finally, eight explosive isometric contractions, separated by a 15-s rest interval, were performed in a knee-joint angle of 90°. Parallelly, participants were asked to report whether they were hindered by pain to perform maximally. Trials that were influenced by pain were deleted from analyses. The most affected leg (based on clinical symptoms) was evaluated in KOA, whereas the right leg was tested in CON.

### *Signal and data processing*

Torque and angular velocity signals were sampled at 100 Hz and processed offline through a commercial software package (Matlab R2015b, The MathWorks Inc., Natick, Massachusetts, USA). From the maximal voluntary isometric contractions, $T_{max}$ (Nm) was defined as the highest value of the torque-time curve (Figure 2A). From the explosive isometric contractions, the rate of torque development (RTD) was calculated as the linear slope of the torque-time curve from the onset of torque production (set at 7.5 Nm) in different time intervals of 100 ms ($RTD_{0–100}$, $RTD_{0–200}$ and $RTD_{100–200}$). Additionally, the highest RTD value within the torque-time curve was determined ($RTD_{max}$) (Figure 2B). The trial with the highest $RTD_{100}$ value was considered for the analyses. From the isotonic and isokinetic contractions, instantaneous knee extensor power (W) was calculated as the product of torque (Nm) and angular velocity (rad·s$^{-1}$) signals. Peak power, and the corresponding torque and velocity, were determined for each repetition. For all parameters, the best performance out of all trials was used for the analyses.

To note, for each participant, the abovementioned number of valid trials were registered for each of the standardized tests (ie, four isometric, two isotonic at each load, three isokinetic, eight explosive isometric), except for the explosive isometric tests where three participants in KOA reported limiting pain. Consequently, all data of the explosive isometric tests were excluded from the analyses for these three participants.

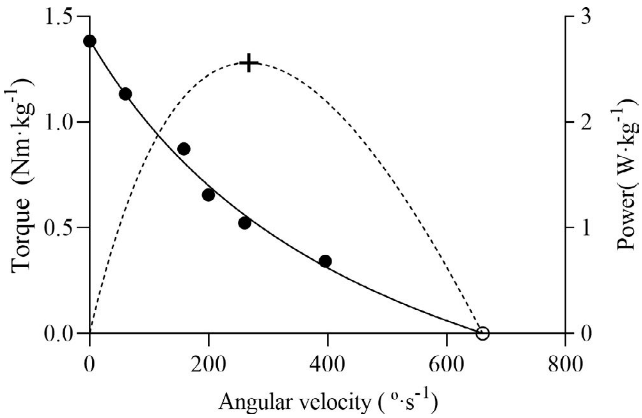


**FIGURE 3** Torque-velocity relationship (solid line) obtained from measured data (black circles) in a representative individual with KOA. The *x*- and *y* axis intercepts correspond to the theorical maximal isometric torque ($T_0$) and maximal shortening velocity ($V_0$, white circle), respectively. The power-velocity relationship (dashed line) derived from the product of torque and velocity is also presented, of which the peak corresponds to the maximum muscle power (plus sign)

The torque-velocity relationship was estimated, based on isometric $T_{max}$, isokinetic and isotonic torque and velocity in all conditions, through Hill's hyperbolic equation[29] (non-linear least square method):

$$(T + a)(V + b) = (T_0 + a)b$$

where $a$ and $b$ are constants, $T_0$ corresponds to $T_{max}$ and $T$ and $V$ correspond to torque and velocity in the isokinetic and isotonic conditions, respectively. From this equation, the ratio $a/T_0$ was calculated. A lower ratio corresponds to a higher curvature of the relationship. Moreover, the velocity at zero torque ($V_0$), determined by the intercept of the velocity axes, the maximum muscle power ($P_{max}$) as well as the corresponding velocity ($V_{opt}$) were calculated from the resulting equation (Figure 3). In addition, torque at different velocities (100, 200, 300, 400 and 500° s$^{-1}$) was estimated from the resulting equation as representative performance along the velocity spectrum. Finally, the coefficient of determination ($R^2$) and standard error of the estimates (SEE) revealed the goodness of fitting of the Hill's model.[30] The combination of isometric, isokinetic and isotonic contractions allowed evaluating the highest range of torque and velocity values, an approach that is strongly recommended to assess the torque-velocity relationship[31] and previously employed.[32] To ensure that the torque-velocity relationship was obtained from trials performed maximally, those trials indicated to be limited by pain and/or deviating from the estimated relationship were excluded. Then, the parameters derived from the torque-velocity relationship were recalculated. This protocol has shown high test-retest reliability (ICC > 0.91) in similar older populations.[28,33]

## 2.3 | Statistical analyses

Data were presented as mean ± standard deviation (SD) unless otherwise stated. To minimize body size cofounding factors, torque and power values were normalized to body mass. Normality distribution was analyzed using a Shapiro-Wilk test. Differences among groups were examined using an unpaired *t*-test (or Mann-Whitney U test in case of non-normal distribution). Differences among KOA with respect to CON were expressed using *t*-scores calculated as:

$$t\,\text{score} = \frac{(x_i - \overline{Y})}{\text{SD}_Y}$$

where $x_i$ is the KOA individual value, whereas $\overline{Y}$ and $\text{SD}_Y$ correspond respectively to the mean and SD of CON. T-scores were employed into a repeated-measures ANOVA to analyze whether knee extension torque deficits in KOA were similar across angular velocities. Huynh-Feldt corrections were applied when the sphericity assumption was violated and pairwise comparisons were carried out applying Bonferroni corrections. Furthermore, Pearson correlation coefficients were calculated using *t*-scores to determine the relationship between knee extensor torque-velocity deficits and functional performance deficits. The effect size statistics were calculated applying the corrected effect size of Hedges's g.[34] An effect size of ≤0.20 was considered negligible; 0.20 to 0.50 small; 0.50 to 0.80 medium and ≤0.80 large.[34] Statistical analyses were performed using SPSS v24 (SPSS Inc., USA) and the level of significance was set at $\alpha = 0.05$.

# 3 | RESULTS

Participants' characteristics are described in Table 1. Ten participants of KOA demonstrated a K-L score of 2, four of 3 and four of 4. The tibiofemoral joint was involved in all KOA participants, concretely, the medial ($n = 8$), lateral ($n = 4$) or both ($n = 6$) compartments. All but one suffered from bilateral KOA.

Mean age was not different between groups ($p = 0.134$). However, KOA showed a trend toward higher body mass ($p = 0.066$) and had significantly higher BMI ($p = 0.022$) compared to CON. Moreover, KOA obtained lower SPPB total score ($p < 0.001$), motivated by a diminished STS performance ($p < 0.001$). No differences were found on balance and habitual gait speed tests of SPPB (both, $p > 0.05$).

Instrumented SCT and STS test data are showed in Table 1. Briefly, KOA completed the SCT with a longer total ascent and ascent rise duration than CON ($p = 0.005$ and $p = 0.003$; respectively), with no differences in total stance duration ($p = 0.650$). In addition, power during SCT was lower in KOA compared to CON ($p < 0.001$). For the STS, KOA registered longer total duration ($p = 0.011$), longer sit-to-stand transition duration ($p = 0.017$) and a trend toward longer stand-to-sit transition duration ($p = 0.061$) than CON. No differences were found in the sit or stand sub-phase duration ($p \geq 0.261$). Moreover, lower mean power values were found in KOA compared to CON ($p = 0.001$). Regarding movement strategy, a trend to increased trunk flexion range was observed in KOA during the sit-to-stand transition ($p = 0.143$).

Torque-time and torque-velocity relationships derived parameters of the knee extensors are shown in Table 1. During the isometric test, KOA participants showed lower $T_{max}$ ($p = 0.001$) values than CON. In addition, moderate to large differences in RTD were demonstrated in KOA versus CON along the torque-time curve ($p < 0.05$). However, these differences disappeared after normalizing RTD values to $T_{max}$ ($g = 0.53$–$0.21$, $p \geq 0.133$) (data not shown).

The Hill's model goodness of fit was excellent in both groups (KOA: $R^2 = 0.995 \pm 0.007$, SEE = 8.65 ± 6.50 Nm; CON: $R^2 = 0.996 \pm 0.005$ and SEE = 9.0 ± 5.8 Nm). Apart from a lower $T_0$ ($p < 0.001$), a lower $P_{max}$ ($p < 0.001$) was found in KOA compared to CON. However, no differences were found in $V_{opt}$ ($p = 0.886$), nor in $V_0$ ($p = 0.599$). When comparing torque values at different velocities between KOA and CON, lower torque values were found in KOA at velocities from $0°\cdot s^{-1}$ to $400°\cdot s^{-1}$ (all $p < 0.05$), while no differences were shown at $500°\ s^{-1}$ ($p = 0.336$). Furthermore, repeated-measures ANOVA comparing these differences using calculated *t*-scores showed a main effect of velocity ($F = 19.299$, $p < 0.001$), indicating a larger difference as the velocity decreased (except for $0°\cdot s^{-1}$) (Figure 4). Attending to post hoc pairwise comparisons, the largest differences were shown at 100 to $200°\cdot s^{-1}$, then at $300°\cdot s^{-1}$ and finally at $400°\cdot s^{-1}$ compared to those observed at $500°\cdot s^{-1}$.

Correlation coefficients between deficits (*t*-scores) on knee extensor muscle function and STS and SCT parameters (total duration, sub-phase duration and power) are summarized in Table 2. Briefly, stronger correlations with knee extensor deficits were found by using power parameters rather than duration parameters for both functional tasks. In particular, the deficit in STS power was more related to maximal torque capabilities (ie, $T_{max}$ and $T_0$) and late $\text{RTD}_{100-200}$. The deficit in SCT power was strongly correlated to most parameters of the torque-time curve (except for later phase $\text{RTD}_{100-200}$) and to torque production from low to high velocities ($0$–$400°\cdot s^{-1}$).

**TABLE 1** Participants' characteristics, functional performance, and knee extensor muscle function.

| | KOA ($n$ = 18) | | CON ($n$ = 26) | | | |
|---|---|---|---|---|---|---|
| | **Mean** | **SD** | **Mean** | **SD** | ***p*** | ***g*** |
| Age (years) | 69.6 | 7.3 | 66.1 | 7.7 | 0.134 | 0.46 |
| Body height (cm) | 159 | 6.5 | 162 | 5.7 | 0.266 | 0.33 |
| Body mass (kg) | 69.9 | 14.0 | 64.5 | 8.9 | 0.066 | 0.57 |
| BMI (kg·m$^{-2}$) | 27.4 | 4.8 | 24.5 | 3.8 | **0.022** | 0.72 |
| SPPB total (points) | 11.0 | 1.5 | 11.9 | 0.3 | **<0.001** | 1.23 |
| Balance | 3.9 | 0.2 | 4.0 | 0.0 | 0.229 | 0.36 |
| Gait | 3.9 | 0.5 | 4.0 | 0.0 | 0.229 | 0.36 |
| STS | 3.2 | 1.0 | 3.9 | 0.3 | **<0.001** | 1.23 |
| K-L score (grade) | 2.9 | 0.7 | | | | |
| 2 | $n$ = 10 | | | | | |
| 3 | $n$ = 4 | | | | | |
| 4 | $n$ = 4 | | | | | |
| Functional performance | | | | | | |
| *SCT* | | | | | | |
| Total ascent duration (s) | 2.49 | 0.72 | 1.93 | 0.43 | **0.005** | **0.92** |
| Rise duration (s) | 2.18 | 0.49 | 1.72 | 0.39 | **0.003** | **0.99** |
| Stance duration (s) | 0.31 | 0.36 | 0.21 | 0.14 | 0.650 | 0.13 |
| Power (W·kg$^{-1}$) | 4.93 | 1.20 | 6.99 | 1.77 | **<0.001** | **1.29** |
| *STS* | | | | | | |
| Total duration (s) | 10.59 | 3.20 | 8.45 | 1.15 | **0.011** | 0.81 |
| Sit duration (s) | 0.32 | 0.35 | 0.16 | 0.07 | 0.615 | 0.15 |
| Sit-to-stand transition duration (s) | 5.49 | 1.15 | 4.70 | 0.89 | **0.017** | 0.76 |
| Stand duration (s) | 0.45 | 0.69 | 0.22 | 0.07 | 0.261 | 0.34 |
| Stand-to-sit transition duration (s) | 5.48 | 1.56 | 4.63 | 0.90 | 0.061 | 0.58 |
| Power (W·kg$^{-1}$) | 2.98 | 0.6 | 3.67 | 0.6 | **0.001** | 1.11 |
| Trunk flexion range (°) | 51.5 | 9.6 | 47.3 | 9.2 | 0.143 | 0.45 |
| Knee extensors muscle function | | | | | | |
| *MVIC* | | | | | | |
| $T_{max}$ (Nm·kg$^{-1}$) | 1.35 | 0.55 | 1.87 | 0.45 | **0.001** | 1.05 |
| *Torque-time relationship* | $n$ = 15 | | $n$ = 26 | | | |
| $RTD_{max}$ [(Nm·kg$^{-1}$)·s$^{-1}$] | 8.94 | 3.35 | 13.96 | 5.32 | **0.002** | 1.04 |
| $RTD_{0–100}$ [(Nm·kg$^{-1}$)·s$^{-1}$] | 6.76 | 2.43 | 9.79 | 2.60 | **0.001** | 1.17 |
| $RTD_{0–200}$ [(Nm·kg$^{-1}$)·s$^{-1}$] | 5.08 | 1.82 | 7.22 | 1.94 | **0.001** | 1.11 |
| $RTD_{100–200}$ [(Nm·kg$^{-1}$)·s$^{-1}$] | 3.39 | 1.68 | 4.66 | 1.57 | **0.020** | 0.77 |
| *Torque-velocity relationship* | | | | | | |
| $T_0$ (Nm·kg$^{-1}$) | 1.18 | 0.45 | 1.90 | 0.40 | **<0.001** | 1.73 |
| $V_0$ (°·s$^{-1}$) | 692 | 145 | 676 | 170 | 0.599 | 0.16 |
| $a$ | 372 | 295 | 457 | 468 | 0.905 | 0.04 |
| $b$ | 3654 | 2914 | 2159 | 1947 | 0.066 | 0.57 |
| $a/T_0$ | 5.6 | 4.2 | 3.9 | 3.9 | 0.168 | 0.42 |
| $P_{max}$ (W·kg$^{-1}$) | 2.74 | 0.85 | 4.09 | 0.90 | **<0.001** | 1.65 |
| $V_{opt}$ (°·s$^{-1}$) | 382 | 104 | 388 | 129 | 0.886 | 0.04 |
| $R^2$ | 0.995 | 0.007 | 0.996 | 0.005 | 0.830 | 0.06 |
| SEE (Nm) | 8.65 | 6.5 | 9.00 | 5.8 | 0.673 | 0.13 |

Abbreviations: $a$ and $b$, Hill's constants; $a/T0$, curvature of the torque-velocity relationship; CON, age-matched control group; K-L score, Kellgren-Lawrence classification; KOA, knee osteoarthritis group; MVIC, maximal voluntary isometric contraction; *P*max, maximum muscle power; $R^2$, coefficient of determination; RTD, rate of torque development; SCT, stair climb test (6-step staircase); SEE, standard error of the estimate; SPPB, Short Physical Performance Battery; STS, 5-repetition sit-to-stand test; $T_0$, torque intercept; $T_{max}$, maximal isometric torque; $V_0$, velocity intercept; $V_{opt}$, velocity at maximum muscle power.

p<0.05 is highlighted in Bold.

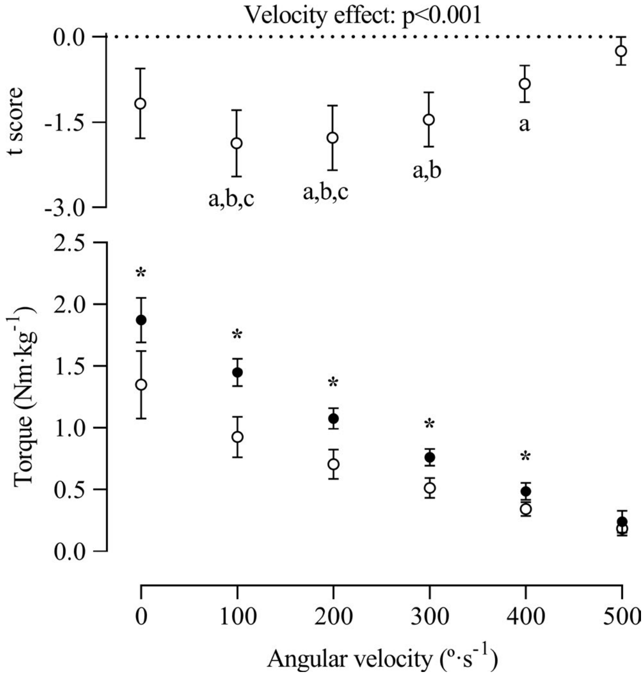


**FIGURE 4** Mean and 95% confidence intervals for torque produced at different velocities for KOA (white) and CON (black) are shown in the bottom panel. Standardized torque differences between KOA compared to CON (*t*-scores) at different velocities are shown in the top panel. *Statistically different from CON ($p < 0.05$). Velocity effect denotes differences across velocities. a,b,c denote statistical significance for pairwise comparison with respect to 500, 400 and 300°·s$^{-1}$, respectively

## 4 | DISCUSSION

The main findings of the present study were that, compared to age-matched controls, postmenopausal women with KOA showed (I) reduced $T_{max}$ and RTD during isometric contractions (ie, downward shift in the torque-time curve), (II) limited torque production capacity during dynamic contractions at slow to moderate velocities, and thus lower power production (ie, downward shift in torque-velocity relationship), and (III) declined functional performance that was closely related to deficits in $T_{max}$ and power production, in particular during stair climbing.

To the best of our knowledge, this is the first study to evaluate the main parameters of both the torque-time curve and torque-velocity relationships in postmenopausal women with KOA compared to healthy age-matched controls. Using this methodology, we obtained a broader point of view of knee extensor muscle functioning in KOA, facilitating the comparison between studies employing a wide variety of isotonic loads or isokinetic velocities in their (single-point) assessments.

In the current study, a 28% deficit in $T_{max}$ was found in KOA compared to CON (Table 1), which is in line with previous comparable studies reporting deficits of 20% to 40%.[8,35] Accordingly, a similar 27%–31% deficit was present in RTD along the entire torque-time curve, confirming previous studies showing that postmenopausal women

**TABLE 2** Pearson correlation coefficients (*p* value) between deficits represented as *t*-scores on different parameters obtained during sit-to-stand (STS) and stair-climbing (SCT) tasks and knee extensor muscle function in the knee osteoarthritis group

| | STS | | | SCT | | |
|---|---|---|---|---|---|---|
| | Total duration | Sit-to-stand transition duration | Power | Total duration | Rise duration | Power |
| $T_{max}$ | −0.164 (0.515) | −0.276 (0.267) | **0.587 (0.010)** | −0.368 (0.133) | **−0.496 (0.036)** | **0.567 (0.014)** |
| $RTD_{100}$ | −0.104 (0.711) | −0.109 (0.698) | 0.218 (0.434) | **−0.564 (0.028)** | −0.488 (0.065) | **0.659 (0.008)** |
| $RTD_{100\text{-}200}$ | −0.342 (0.213) | −0.458 (0.086) | **0.599 (0.018)** | −0.405 (0.134) | −0.409 (0.130) | 0.395 (0.145) |
| $RTD_{200}$ | −0.227 (0.415) | −0.284 (0.305) | 0.423 (0.116) | **−0.564 (0.029)** | −0.514 (0.050) | **0.622 (0.013)** |
| $RTD_{max}$ | −0.018 (0.950) | 0.035 (0.900) | 0.055 (0.845) | −0.459 (0.085) | −0.384 (0.157) | **0.593 (0.020)** |
| $T_0$ | −0.423 (0.080) | −0.437 (0.070) | **0.472 (0.048)** | **−0.521 (0.027)** | **−0.496 (0.036)** | **0.592 (0.010)** |
| $V_0$ | 0.198 (0.430) | 0.209 (0.405) | −0.284 (0.254) | 0.149 (0.554) | 0.074 (0.771) | −0.147 (0.561) |
| $P_{max}$ | −0.180 (0.475) | −0.157 (0.533) | 0.229 (0.361) | −0.384 (0.116) | −0.400 (0.100) | **0.568 (0.014)** |
| $T$ at 100°·s$^{-1}$ | −0.354 (0.149) | −0.356 (0.147) | **0.416 (0.086)** | **−0.485 (0.041)** | −0.464 (0.052) | **0.587 (0.010)** |
| $T$ at 200°·s$^{-1}$ | −0.283 (0.255) | −0.274 (0.271) | 0.338 (0.170) | −0.443 (0.066) | −0.435 (0.071) | **0.580 (0.012)** |
| $T$ at 300°·s$^{-1}$ | −0.196 (0.435) | −0.178 (0.481) | 0.217 (0.386) | −0.388 (0.112) | −0.411 (0.090) | **0.571 (0.013)** |
| $T$ at 400°·s$^{-1}$ | −0.051 (0.841) | −0.020 (0.937) | −0.016 (0.951) | −0.262 (0.293) | −0.343 (0.163) | **0.492 (0.038)** |
| $T$ at 500°·s$^{-1}$ | 0.132 (0.601) | 0.164 (0.516) | −0.307 (0.216) | −0.017 (0.946) | −0.151 (0.550) | 0.212 (0.399) |

Abbreviations: $P_{max}$, maximum muscle power; RTD, rate of torque development; $T$, torque; $T_0$, torque intercept; $T_{max}$, maximal isometric torque; $V_0$, velocity intercept.

The background grayscale indicates the strength of correlation (i.e., darker background represents greater association).

with severe KOA needed twice as much time to achieve their $T_{max}$ compared to controls.[23,36] However, Callahan et al.[23] pointed out that this difference in RTD disappeared after normalizing to $T_{max}$, which is in line with our findings after applying the same normalization approach. This suggests that people with advanced KOA might already experience such a major deficit in $T_{max}$ that RTD is automatically limited. The ability to rapidly produce force is essential in reactive functional task, such as fall prevention following sudden perturbations,[37] and has been associated to physical function after total knee arthroplasty[38] and to maximal gait speed in people with KOA.[36]

As in the torque-time curve, muscle weakness of the knee extensors was also noted throughout the torque-velocity relationship. The reduced $T_0$ observed in KOA highlighted their impaired maximal torque capabilities compared to CON. This torque deficit was also observed at low to moderate contraction velocities (up to 400°·s$^{-1}$, approximately 60% $V_0$), but not at the low-torque, high-velocity portion of the torque-velocity relationship. Accordingly, $P_{max}$ was approximately 33% lower in KOA compared to CON (Table 1), whereas the greatest torque and power deficits were found at angular velocities of 100 to 200°·s$^{-1}$ (Figure 4). Therefore, knee extensor muscle weakness in people with KOA might not only be influenced by torque demands, but also by contraction velocity, at least during concentric actions. This reinforces the greater relevance of muscle power compared to $T_{max}$ in physical functioning of people with KOA,[10–12] as already confirmed in older people.[13,28]

The physiological determinants of knee extensor muscle weakness in postmenopausal women with KOA might be numerous and complex, since different factors such as the aging process, disuse, and KOA itself may play a determinant role. Briefly, the loss of muscle mass, and especially the atrophy of type II fibers, seem to be major contributors to the loss of maximal torque capabilities.[4,5,39] In addition, other factors related to muscle quality, such as muscle architecture (ie, reduced physiological cross-sectional area),[7] increased profibrotic environment and limited muscle fiber remodeling capacity[6] and decreased single fiber contractile function seem to be involved as well. Indeed, single muscle fibers studied in vitro show reductions in force, power and contractile velocity in older women with KOA compared to controls.[40] In contrast, the current study did not find differences in maximal shortening velocity (ie, $V_0$) assessed in vivo. These differential findings of in vitro and in vivo studies might be partly related to the major contribution of the fastest muscle fibers (ie, type IIx) to the whole muscle-tendon unit force production capacity at high contraction velocity.[31] Of course, we cannot ignore the role of neural and arthrogenic muscle inhibition factors, such as the reduced firing rate and recruitment of larger motor units found in the vastus lateralis muscle of people with KOA,[41] and the role of the muscle-tendon unit behavior as force transducer.[42]

Regarding functional performance observed during daily tasks (ie, STS and SCT), KOA presented clinically meaningful limitations compared to CON (Table 1). In particular, KOA needed more time to complete the STS task (+25%, $p = 0.011$), mainly attributed to a slower sit-to-stand transition phase (ie, concentric phase) (+17%, $p = 0.017$) in addition to a slower stand-to-sit transition phase (ie, eccentric phase) (+18%, $p = 0.061$). This limitation during the STS became more evident by the reduced power production in KOA (−28%, $g = 1.11$, $p = 0.001$) compared to CON. Furthermore, KOA adopted a greater trunk flexion than CON (+8.9%), although this trend was not statistically significant ($p = 0.143$). Our results are consistent with those recently summarized in a meta-analysis reviewing the altered kinematics and kinetics during the STS in people with KOA.[43] In line with the results on STS, KOA needed more time to complete the SCT (+25%, $p = 0.005$), because of a slower ascent rise phase (+28%, $p = 0.003$). Power production during this rise phase showed the largest differences between groups (+28%, $g = 1.29$, $p < 0.001$). To summarize, power production during the concentric phase of both functional tasks (STS and SCT) appear to be crucial components in daily life functioning of women with KOA.

As previously outlined by Sonoo et al,[43] the longer STS time in KOA appeared to be related to changes in movement strategy, to pain and to knee muscle weakness. In the current study, we investigated whether the deficits (represented by *t*-scores) in knee extensor muscle weakness and functional performance were related. We clearly observed that sensor-based power production measured during functional tasks was more closely related to knee extensor muscle weakness than time-based outcomes, even when sub-phase duration was analyzed (Table 2). This emphasizes the value of objective power measurements in addition to time-based outcomes. More in detail, we found that the deficits in maximal torque capabilities (ie, $T_{max}$ and $T_0$) were moderately related to muscle power deficits during STS and SCT tests ($r = 0.472–0.587$ and $r = 0.567–0.592$, respectively) (Table 2). While power deficits during SCT also appeared to be closely related to explosive RTD (except for $RTD_{100–200}$) or torque deficits at low to moderate velocities ($r = 0.593–0.659$ and $r = 0.492–0.587$, respectively) (Table 2), this was not the case for STS. STS is a bilateral task and therefore less challenging than SCT, which is unilateral. Unilateral tasks require more balance and joint proprioception and involve additional hip musculature shown to be weakened in this population.[44] Therefore, this unilateral and complex functional task might be preferential to evaluate physical function decline in postmenopausal women with KOA. Due to the limited sample size, we could not perform a multiple regression analysis to address whether different components of knee extensor muscle weakness could explain more of the variance in physical function compared to one specific component, but as we have pointed above, muscle power seems to be a determinant factor.[10–12]

Some methodological considerations should be kept in mind when interpreting our findings. Firstly, $T_{max}$ and $T_0$ should not be considered interchangeable, at least in KOA. Significant differences between these variables were found in KOA, with $T_0$ being lower than $T_{max}$ ($p = 0.016$, $g = 0.32$, data not shown). The torque deficits observed at low and moderate velocities provoked a flattening of the high-torque portion of the torque-velocity relationship, resulting in a lower $T_0$. This highlights the additional limitations in KOA in dynamic movements over isometric contractions in one knee-joint angle (90°). Secondly, the order of the tests (isometric–isotonic–isokinetic–isometric explosive) and loads during isotonic tests (40%–20%–0%–60% $T_{max}$) was fixed. Potential influence of fatigue and/or a learning effect on subsequent performance cannot be fully discarded. However, this protocol has shown high test-retest reliability (ICC's > 0.91) in similar older populations.[28,33] In addition, we are convinced that the non-randomization did not have an influence on the results. The largest difference between controls and people with KOA was situated at velocities of 100–200 °/s, this is somewhere in between 40% and 60% of $T_{max}$ (ie, the load performed first and the load performed last). If learning effect would be more of an issue in KOA, the difference would turn out to be smaller around 60% load. If fatigue would be more of an issue in KOA compared to control, the difference would be greater around 60°/s (as this isokinetic test was performed after the isotonic tests). Even more, the benefit of plotting the full torque-velocity profile instead of using all points as "single measurement points" allows excluding trials that deviate from the torque-velocity relationship (ie, those trials where participants failed to reach their maximal performance), further improving the reliability of the method.[45] Thirdly, the severity of KOA might influence the magnitude of muscle weakness. Due to limited sample size, we were not able to cluster participants by structural KOA and/or pain severity, which should be considered in further studies. Finally, we used the t-score to calculate the individual difference compared to a reference control group, but the latter might not correspond to the normative values for postmenopausal women.

In conclusion, postmenopausal women with KOA demonstrated knee extensor muscle weakness compared to age-matched controls. This weakness was largely due to limited high force production capabilities and was more evident in explosive conditions and at low to moderate contraction velocities (ie, at high force demands). On the contrary, postmenopausal women with KOA do not seem to have a deficit in maximal velocity nor in force production at very high velocities compared to age-matched controls. In addition, knee extensor muscle weakness is closely related to impaired physical function. More specifically, deficits in STS performance are mainly linked to deficits in maximal force, while deficits in SCT performance are related to deficits in early onset of force production (0–100 ms) and in the ability to develop force at moderate velocities.

## 5 | PERSPECTIVE

On a general basis, maximal force capacity (eg, MVIC or one-repetition maximum) has been extensively evaluated to assess muscle weakness in people with KOA. In recent years, maximal muscle power has gained attention as one of the most valuable biomarkers of muscle function in this population. These outcomes refer to single points of the force-time and force-velocity relationship, while a broader view of knee extensor muscle functioning can be obtained when investigating the full force-velocity and force-time relationship. Evaluating force deficits among different contraction velocities allows determining the targets for personalized physical therapy, considering that resistance training performed at distinct velocities elicits velocity-specific adaptations.[46] Explosive resistance training (ie, to maximize power output) is safe, feasible and provides additional benefits on muscle function in people with KOA compared to traditional resistance training.[47,48] Therefore, explosive resistance training should be included as part of a strength training regimen in people with KOA. Our findings suggest that people with KOA might benefit from explosive resistance training protocols performed across the force-velocity spectrum, especially within the moderate to high force portion (ie, 40% to 80%–100% of their maximum isometric force). We should however acknowledge that our findings are exploratory and high-quality randomized controlled trials are needed to support this claim.

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## CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in Center for Open Science at https://osf.io/z72xj/.

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