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The role of physical activity in mitigating age-related changes in the neuromuscular control of gait

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Exercise induces neural and muscular adaptations, improving muscle mass and function in older adults. We investigated its impact on gait neuromuscular control in young and older adults, classified as more active (young: $n = 15$, 5185 ± 1471 MET-min/week; old: $n = 14$, 6481 ± 4846 MET-min/week) or less active (young: $n = 14$, 1265 ± 965 MET-min/week; old: $n = 14$, 1473 ± 859 MET-min/week). Isometric maximal voluntary torques were assessed for proximal (knee) and distal (ankle) extensors, and muscle mechanical properties of these muscles were assessed using Myoton. Gait was analysed using ground reaction forces, motion capture, and electromyography. Less active older adults exhibited shorter steps, higher mechanical cost, and greater collision at heel strike. These differences were linked to altered neuromuscular control, wider activation of lumbar and sacral motor pools, different activation timing, and reduced muscle-tendon stiffness. Our findings highlight that physical activity preserves neuromuscular control, muscle mechanical properties, and gait efficiency, mitigating age-related decline.

With extended life expectancy, the loss of mobility of older adults has dramatic individual and societal impacts. Among the many factors contributing to frailty (e.g., metabolic, hormonal, and immunological changes), the loss of muscle mass and strength[1,2], alterations in connective tissue properties[3], and neurodegenerative changes[4] play a key role in the decline of mobility and functional capacity in older adults. These neuromuscular alterations result in an increased metabolic rate of locomotion in older adults, associated with a loss of independence and an increased risk of morbidity and mortality[5–8].

Several age-related modifications of the muscle and tendon tissues have been described, such as loss of muscle fibres[9–11], a shift in muscle fibre type distribution[12], modification of the tendon and muscles cross-sectional area[13,14] or changes in the stiffness and viscoelastic properties of tendon and muscles[14–18], associated with histological changes such as impaired connective tissues or myosteatosis. The decrease in muscle-tendon unit performance has an impact on mobility of the older adults. For example, older adults redistributed lower extremity joint moment and power compared with young people in walking[19,20], so-called biomechanical plasticity. The scientific community[19,21–28] most often points to a reduction in mechanical power generated by the plantar flexor muscles during the push-off phase of walking as the hallmark biomechanical ageing features of gait.

The decline in mobility and functional capacity in older adults is influenced by multiple factors, including muscle weakness, changes in connective tissue properties, and neurodegenerative processes. While the reduction in ankle muscle contractile performance significantly affects mobility[29], other neuromuscular factors also play a crucial role[30,31]. In particular, age-related changes in spinal motoneuron activity impact force production and movement control. Older adults exhibit altered motoneuron recruitment and firing characteristics[32,33], leading to reduced muscle function. Additionally, age-related modifications in spinal segmental output —such as broader motoneuron activation patterns, increased co-activation, and differential impairment of caudal versus rostral segments—may contribute to changes in locomotor coordination and efficiency[34–36]. Decoding these neuromuscular changes is essential for understanding their contribution to gait adaptations in aging and may be essential for linking the modification of output with neuromuscular degeneration and the firing characteristics of motoneurons.

High physical activity level is crucial for enhancing physical fitness by increasing muscle strength, and cardiopulmonary function and stimulating metabolic activity[37–42], decreasing risk of disability[43]. Regular physical activity plays a role in increasing/maintaining muscle mass, strength, power, and functional capacity in older adults, but it also modify age-related changes in motor unit structure and function across the life span[31,44]. For example, exercise promotes various metabolic responses and morphological reconfigurations in the neuromuscular junction of older adults[45], decelerating or even reversing

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neuromuscular junction degeneration[46]. On the contrary, less regular physical activity among older adults may exacerbate age-related differences between young and older adults, for example in muscle activation[47]. However, enhancing physical capacity alone may not be sufficient to mitigate the age-related decline of the neuro-muscular system. Indeed, increasing the physical activity of older adults via resistance training fails to directly translate to improved propulsive power generation in walking[48]. Similarly, Brach et al. (2013) assert that multicomponent impairment-based walking exercises, while beneficial for strength, flexibility, and endurance, don't always lead to better walking ability. Also, a higher level of physical activity in older adults did not mitigate the age-related modification of kinematic coordination and distal-to-proximal redistribution[49], referring to the shift in mechanical work and power generation from distal joints (such as the ankle) to more proximal joints (such as the hip) during walking[25], suggesting that there remains a gap in understanding the biomechanical adaptations that physical activity levels may induce in gait mechanics across aging[48].

While a large body of literature has focused on the role of physical activity in preserving mobility, it is still difficult to disentangle the influence of biological aging from that of physical activity on the subsequent changes in the neuromuscular system. To address this question, we recorded data from both older and younger participants, divided according to their weekly physical activity levels. After evaluating the static balance, isometric strength and the mechanical properties of ankle and knee extensors, we then compared the mechanics and neuromuscular control of gait. The purpose of this study was to shed light on contributors to the age-related changes in biomechanical, physiological, and motor control adaptations of gait. We first hypothesized that a clear effect of aging on muscular mechanical properties, maximal isometric forces and neuromuscular control of gait would be observed. Also, we tested the hypothesis that more active older individuals exhibit a "slowing down" in the natural aging process of the neuromuscular system.

Results

Some small differences in subject characteristics were found between groups, presented in Table 1. Subjects physically more active were slightly younger than less active ones ($F_{1,55} = 2144.0$, $p < 0.001$; $F_{1,55} = 6.0$, $p = 0.017$). Older adults were on average shorter than young adults ($F_{1,55} = 8.6$, $p = 0.004$). Regarding the subject's weight, no significant differences were observed between groups ($p > 0.743$). However, the BMI was slightly lower in more active individuals ($F_{1,55} = 4.0$, $p = 0.049$), while age ($F_{1,55} = 0.3$, $p = 0.579$) had no effect. More importantly, no effects of age ($F_{1,55} = 0.9$, $p = 0.334$) or interaction ($F_{1,55} = 0.4$, $p = 0.511$) were observed on the classification using the estimated MET-min/week.

Muscular properties

The maximal isometric torques were affected by age (Fig. 1B). Indeed, both the knee and ankle extensor strength were smaller in older adults compared to young participants (knee: $F_{1,55} = 27.5$, $p < 0.001$; ankle: $F_{1,55} = 26.9$, $p < 0.001$). No difference was observed in knee extensor torque as a function of physical activity level ($F_{1,55} = 2.7$, $p = 0.107$). However, an effect of level of activity was observed on plantar flexor torque ($F_{1,55} = 6.2$, $p = 0.020$), with the more active participants being stronger.

When comparing the mechanical properties of knee and ankle extensors, the stiffness of both *rectus femoris* and *gastrocnemius medialis* decreased with age (RF: $F_{1,326} = 101.8$, $p < 0.001$; GM: $F_{1,326} = 5.5$, $p = 0.019$). Instead, the tissue dampening increased for both muscles with age (RF: $F_{1,326} = 199.0$, $p < 0.001$; GM: $F_{1,326} = 169.9$, $p < 0.001$). The physical activity level significantly influenced both stiffness (RF: $F_{1,326} = 76.4$, $p < 0.001$; GM: $F_{1,326} = 21.7$, $p < 0.001$) and tissue dampening (RF: $F_{1,326} = 6.8$, $p = 0.009$; GM: $F_{1,326} = 10.5$, $p = 0.001$). The active participants exhibited higher muscle stiffness and lower tissue dampening. Regarding stiffnesses, the effect of physical activity level was more pronounced in young adults (interaction: RF: $F_{1,326} = 8.1$, $p = 0.005$; GM: $F_{1,326} = 4.9$, $p = 0.027$;).

Static balance

The average centre of pressure (CoP) displacement during standing was compared between groups (Fig. 1C). The lateral displacement of the CoP was not influenced by age or physical activity level (Age: $F_{1,54} = 2.4$, $p = 0.121$; physical activity level: $F_{1,54} = 0.1$, $p = 0.710$). Regarding the anteroposterior displacement of the CoP, greater displacement was observed in older adults ($F_{1,54} = 4.0$, $p = 0.050$), but the level of activity did not affect the static balance ($F_{1,54} = 0.56$, $p = 0.457$).

Gait mechanics

Figure 2 illustrates a typical trace of the centre of mass (CoM) energy fluctuation and the time course of the instantaneous recovery within one stride in each group. Older adults walked with shorter steps ($F_{1,55} = 32.8$, $p < 0.001$). Interestingly, the effect of age was greater for less active participants (physical activity level: $F_{1,55} = 8.4$, $p = 0.005$; interaction: $F_{1,55} = 4.4$, $p = 0.040$), with the less active older adults displaying significantly shorter step lengths compared to the more active ones. Similarly, the stance phase was smaller in older adults ($F_{1,55} = 29.6$, $p < 0.0001$), and in less active participants ($F_{1,55} = 4.1$, $p = 0.045$).

The mechanical work required to sustain the motion of the CoM relative to the surroundings (W_{ext}) was not affected by the age ($F_{1,55} = 3.8$, $p = 0.056$) or physical level of the participants ($F_{1,55} = 2.7$, $p = 0.101$). However, an interaction between these two factors was found ($F_{1,55} = 4.4$, $p = 0.040$), showing that in older adults, the mechanical cost of walking was higher in less active individuals compared to the more active ones (post hoc: $p = 0.019$). Such difference in W_{ext} can partially be explained by a reduction

Table 1 | Subject characteristics grouped based on their age and level of physical activity

variable	Young adults		Older adults		
	Less active	*More active*	*Less active*	*More active*	
n	16	15	14	14	
Age (years)	26.7 ± 4.0	25.5 ± 2.0	73.1 ± 5.5	69.5 ± 2.5	+ £
Height (cm)	178.8 ± 8.6	179.4 ± 8.8	169.7 ± 9.7	174.0 ± 9.3	+
Weight (kg)	73.2 ± 10.8	72.9 ± 8.5	72.2 ± 15.9	76.3 ± 13.6	
BMI (kg m^{-2})	23.1 ± 4.4	22.7 ± 1.9	24.9 ± 3.6	25.0 ± 3.0	
Knee extensor strength (N m)	194.0 ± 48.8	203.7 ± 54.4	119.9 ± 40.5	150.7 ± 37.5	+
Ankle extensor strength (N m)	47.8 ± 25.2	65.0 ± 19.7	30.7 ± 12.6	35.8 ± 10.3	+ £
Met-min/week	1265 ± 965	5185 ± 1471	1473 ± 859	6481 ± 4846	£

Table 1 presents the characteristics of the young and older adult participants, grouped as either less active or more active based on their MET-min/week values. The variables include age (years), height (mt), weight (kg), body mass index (BMI) (kg/mt^2), knee extensor strength, and plantar flexor strength (N.m). The values are reported as means with corresponding standard deviations (±). Significant effects of age, physical activity level (Pal), and their interaction (Age*Pal) are noted with the symbols +, £, and $, respectively.

Values are the mean ± SD. A significant ($p < 0.05$) effect of age, physical activity level, and their interaction are indicated with the symbols +, £, and $, respectively.

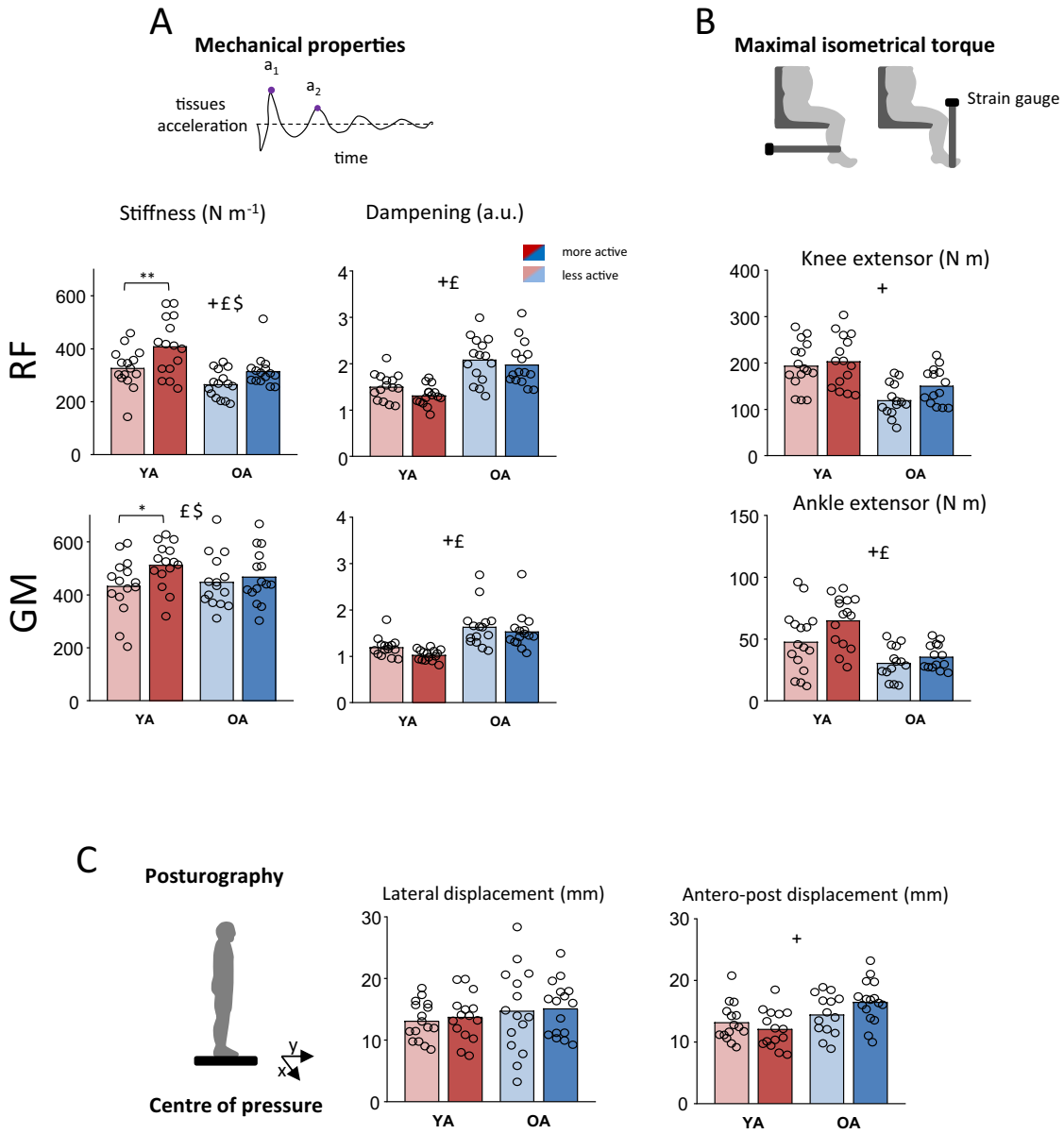

Fig. 1 | Mechanical properties, maximal isometric torque, and posturography. **A** Mechanical Properties: The stiffness (N/m) and damping (a.u.) of the *rectus femoris* (RF) and *gastrocnemius medialis* (GM) muscles were measured using the MyotonPRO device. The upper schematic illustrates the tissue acceleration following the brief impulse applied by the MyotonPRO, which records the subsequent oscillatory response of the tissue. The peak a_1 represents the initial acceleration peak and a_2 represents the decaying oscillations as the tissue returns to its resting state. **B** Maximal Isometric Torque: the schematics illustrate the participant's position and the orientation of the strain gauge fixation, which varies depending on the muscles being assessed. The bar plots represent the maximal isometric torque of knee extensors and ankle extensors for each group **C** Posturography: The CoP displacement was measured in both lateral (X) and anteroposterior (Y) directions. The bar plots indicate the average lateral and anteroposterior displacement in each group of participants. In each subplot, the bars represent the group mean, whereas the open circles indicate individual data. A significant effect of age, physical activity level, and their interaction are indicated with the symbols +, £, and $, respectively. The asterisks indicate post hoc comparisons between groups (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, ****$p < 0.0001$).

of energy transduction between E_k and E_p. Indeed, the *%recovery* was reduced in older adults ($F_{1,55} = 6.3$, $p = 0.014$) in less active participants ($F_{1,55} = 8.8$, $p = 0.004$), with a significant interaction between both fixed factors ($F_{1,55} = 5.4$, $p = 0.023$). In sum, as for W_{ext} no difference was observed between less and more active young adults (post hoc: $p = 0.645$), whereas the less active older adults showed a significantly lower *%recovery* than the more active ones ($p < 0.001$).

The pattern of fluctuation of the CoM power during walking (Fig. 2B) typically presents one major peak of negative power ($\dot{W}^-$) and two major peaks of positive power ($\dot{W}_1^+$ & $\dot{W}_2^+$)[50,51]. The three peaks of power were affected by age ($\dot{W}^-$: $F_{1,55} = 9.5, p = 0.003$; $\dot{W}_1^+$: $F_{1,55} = 14.5, p < 0.001$; $\dot{W}_2^+$: $F_{1,55} = 14.4, p < 0.001$), with greater $\dot{W}^-$ and $\dot{W}_1^+$ and smaller $\dot{W}_2^+$ in older individuals. No significant effect of physical activity level was found (all $p > 0.547$), but an interaction between age and the level of activity was significant for $\dot{W}_1^+$ ($F_{1,55} = 4.9$, $p = 0.030$). The peak of power tended to be greater in older participants less active compared to more active ones, whereas no differences were observed between young adults less or more active (post hoc: $p = 0.243$).

Gait kinematics

The average lower-limb joint angles (hip, knee and ankle), as well as two orientation angles (trunk and foot), were measured for each group of participants (Fig. 3A). In Fig. 3B, we have highlighted 3 parameters affected by age: the foot angle at touchdown, the mean trunk angle, and peak knee

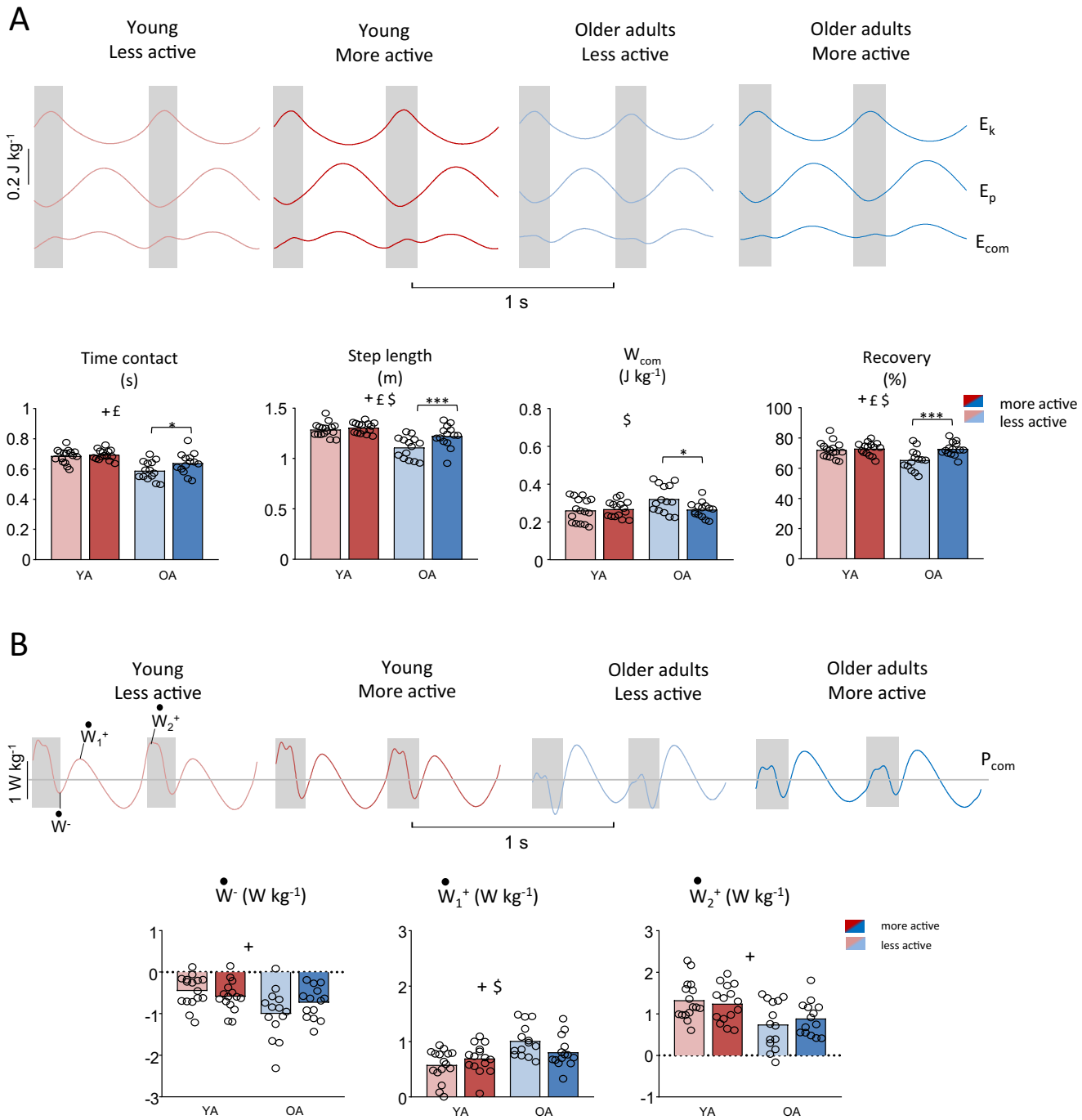

Fig. 2 | Mechanics of walking. A Mean traces of kinetic energy (E_k), potential energy (E_p), and total mechanical energy (E_{com}) versus time across one representative stride for each group of participants. At the bottom, the bar plots indicate from left to right the average stance duration (s), step length (m), external work to sustain the motion of the CoM (J kg^{-1} m^{-1}), and the %recovery. **B** Mean time curves of the external power of the centre of mass (P_{com}) in each group of participants. Each trace displays one major negative peak (W^-, collision) and two major positive peaks, W^+_1 (weight acceptance) and W^+_2 (propulsion), during a stride. The bar plots represent the average value of the three peaks of power. In all panels, the grey shaded areas indicate the double-contact phases. The length of the trace along the x-axis represents the mean stride time for each group. Other information as in Fig. 1.

flexion during stance. Indeed, compared to young adults, the foot at touchdown was less dorsiflexed ($F_{1,54} = 4.6$, $p = 0.036$), the trunk was more inclined forward ($F_{1,54} = 28.2$, $p < 0.0001$) and peak knee flexion was greater during stance ($F_{1,54} = 4.7$, $p = 0.033$) in older adults. None of these angles was affected by the level of physical activity (all $p > 0.201$). Nevertheless, an interaction between the effect of age and physical activity level was significant for the foot angle at touchdown ($F_{1,54} = 8.5$, $p = 0.005$). Indeed, the older adults more active had greater ankle dorsiflexion at touchdown compared to less active ones (post hoc: $p = 0.002$), whereas no difference was found between groups of younger adults.

Muscle activity

Figure 4 shows the ensemble averages of rectified electromyographic (EMG) envelopes for each group during one walking stride. When comparing the root mean square (RMS) of each EMG, an average greater activation was observed in older adults for all knee extensor and flexor muscles (*vastus*

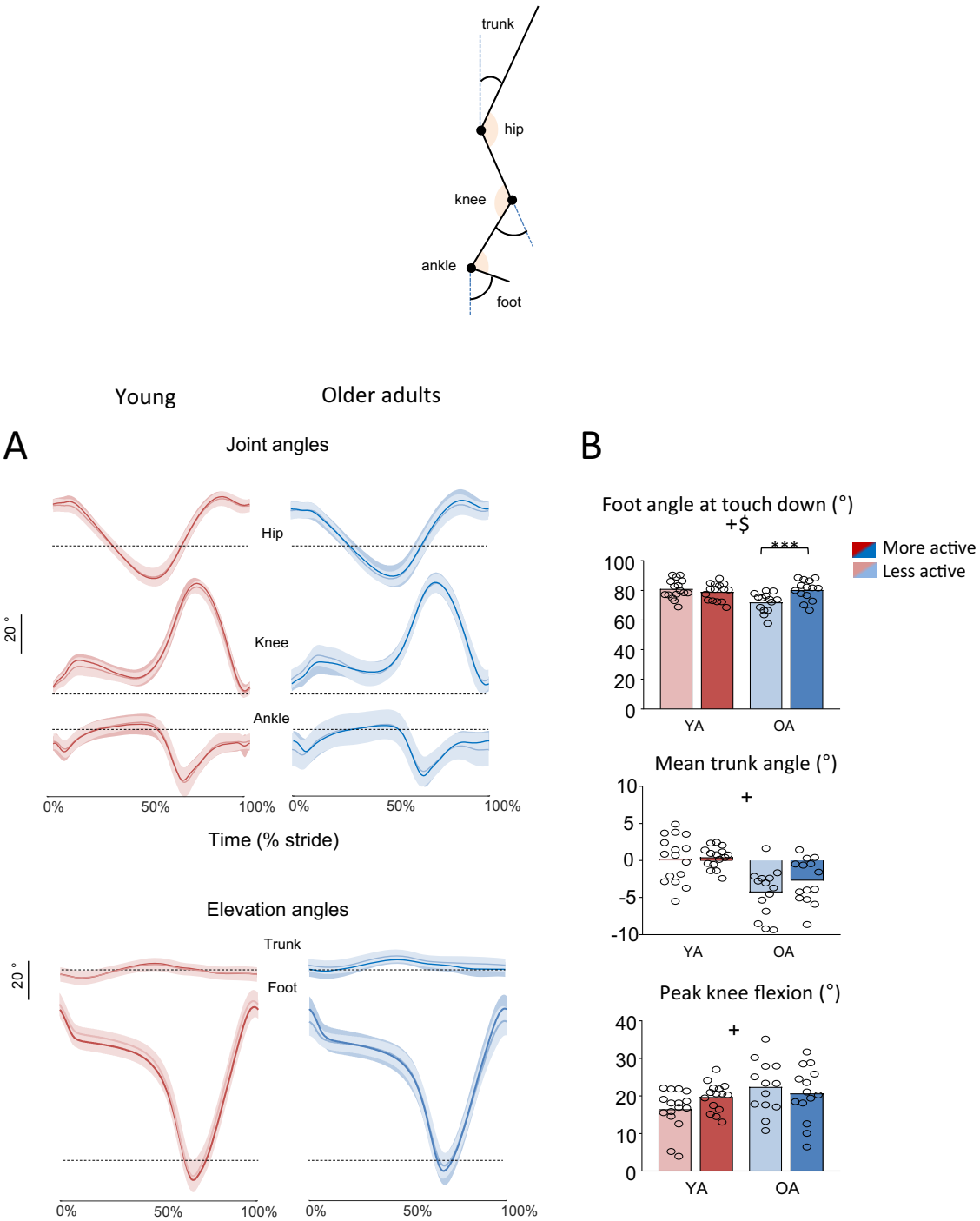

Fig. 3 | Kinematics of walking. A The stickman illustrates the different joint angles (hip, knee, and ankle) as well as the two orientation angles (trunk and foot) measured in each participant. The mean traces and standard deviations (shaded areas) of each angle over a stride are presented. **B** The bar plots present the average foot angle at touch down, the mean trunk angle over a stride, and the peak knee flexion during stance in each group. Other indications as in Fig. 1.

medialis, vastus lateralis, rectus femoris, semitendinous, biceps femoris), and also for *tibialis anterior*, and *gastrocnemius lateralis* (for all $F > 6.2$ and $p < 0.016$). Also, an effect of physical activity was observed for some muscles (*vastus lateralis*: $F_{1,53} = 4.8$, $p = 0.032$; *tibialis anterior*: $F_{1,55} = 4.0$, $p = 0.049$; *gastrocnemius medialis*: $F_{1,55} = 6.1$, $p = 0.016$), with a higher activation in active individuals. Moreover, for the *erector spinae*, an interaction between age and physical activity level was found ($F_{1,54} = 5.0$, $p = 0.029$), with lower activation in less active older adults compared to their more active peers (post hoc: $p = 0.013$). For the *tibialis anterior*, post hoc analysis revealed that older adults in the more active group had higher activation than their less active counterparts ($p = 0.015$), while no differences were found in younger adults ($p = 0.785$).

Regarding the duration of EMG activation, measured based on the full width at half maximum (FWHM) (Fig. 4B), longer bursts were observed in older adults for a great majority of muscles, erector spinae (ES), tensor fasciae latae (TFL), the gluteus medius (GMED), rectus femoris (RF), tibialis anterior (TA), semitendinosus (ST), long head of biceps femoris (BF), soleus (SOL) (for all $F > 6.2$ and $p < 0.015$). Instead, no effect of physical activity level (or interaction) was found (for all $F < 3.3$ and $p > 0.071$). The timing of activation was also affected by age. Indeed, the ES, GMED and BF were

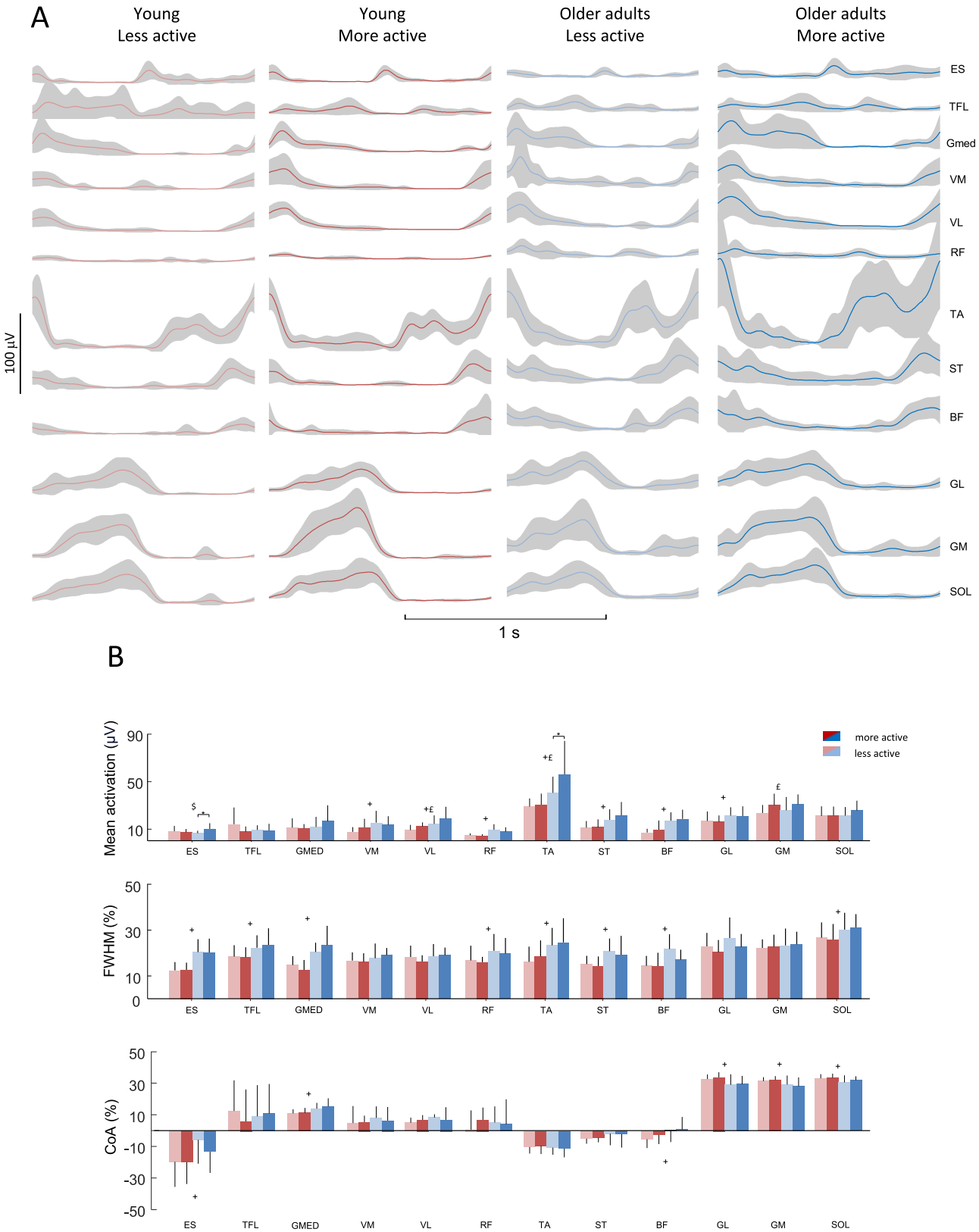

Fig. 4 | Electromyography (EMG) activation patterns. A Ensemble-averaged EMG curves of the *erector spinae* (ES), *tensor fasciae latae* (TFL), *gluteus medius* (Gmed), *vastus medialis* (VM), *vastus lateralis* (VL), *rectus femoris* (RF), *tibialis anterior* (TA), *semitendinosus* (ST), *biceps femoris* (BF), *gastrocnemius lateralis* (GL), *gastrocnemius medialis* (GM), and *soleus* (SOL). The coloured lines represent the ensemble-averaged EMG traces for each group, while the shaded grey areas indicate the standard deviation. **B** The bar plots represent, from top to bottom, the root mean square (in µV), the full-width half maximum (FWHM, in % of stride), and the centre of activation (CoA, in % of stride) for each muscle and each group. The bars correspond to the mean values, with vertical lines above each bar representing one standard deviation. Other indications as in Fig. 1.

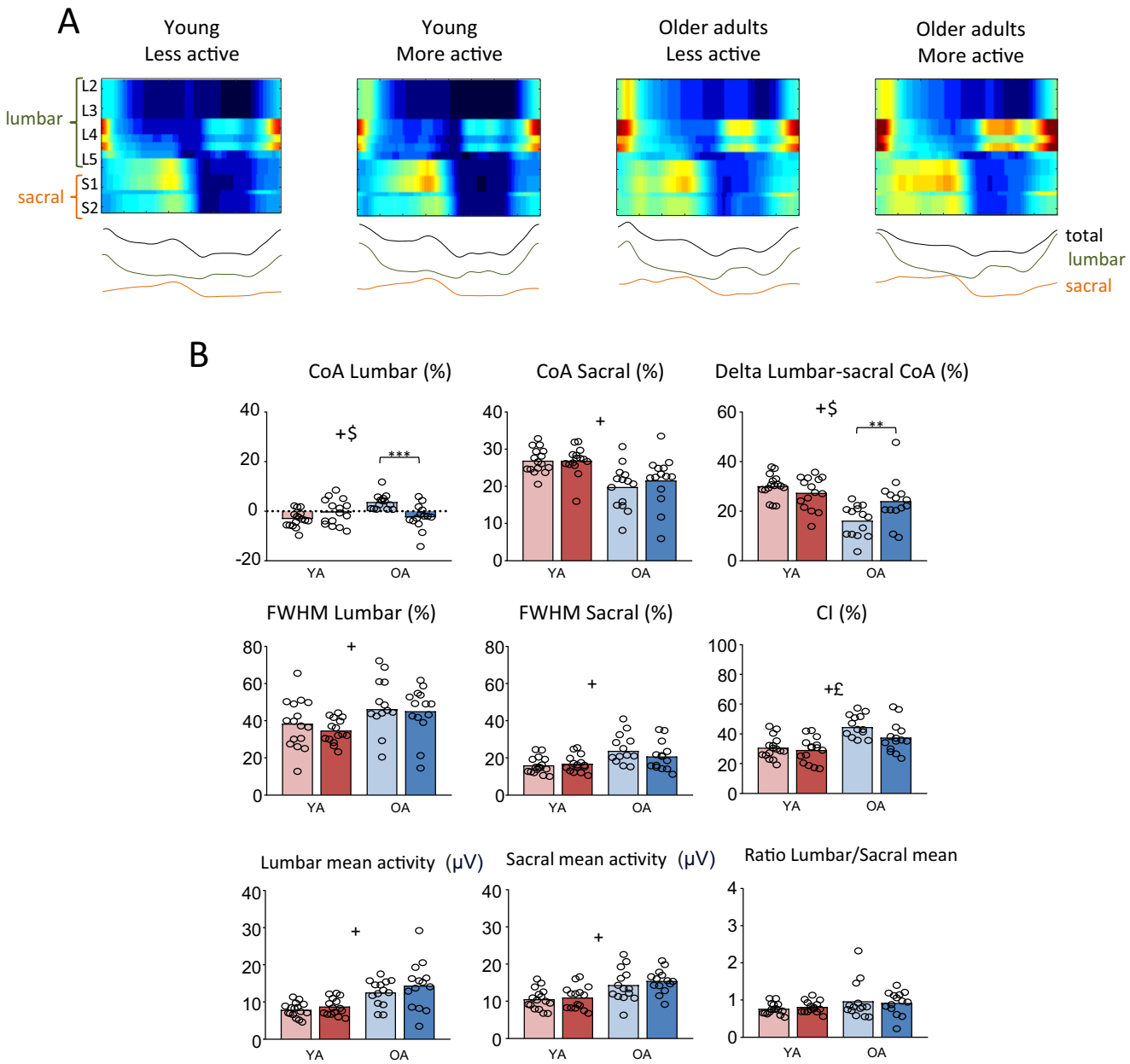

Fig. 5 | Spinal motor outputs. A Unilateral spatiotemporal spinal motor outputs computed from ensemble-averaged EMGs for all participants of each group. The total, lumbar, and sacral activation levels are also depicted as traces below the heat maps for each group, relative to the gait cycle. **B** Bar plots show the centre of activation (CoA, in % of stride) for the lumbar and sacral segments and the delta between lumbar and sacral CoA. Also, the full-width half maximum (FWHM, in % of stride) for both spinal regions, and the coactivation index (CI, %) between lumbar and sacral activations are presented. Finally, the mean activation levels (in μV) of lumbar and sacral outputs, along with the ratio between lumbar and sacral mean activation are displayed. Other indications as in Fig. 1.

activated later in the stride in older adults (ES: $F_{1,54} = 7.1$, $p = 0.010$; GMED: $F_{1,54} = 12.8$, $p = 0.001$; BF: $F_{1,53} = 6.5$, $p = 0.013$), whereas the ankle extensors were activated earlier in stance (gastrocnemius lateralis: $F_{1,55} = 10.4$, $p = 0.002$; GM: $F_{1,55} = 7.7$, p = 0.007; SOL: $F_{1,54} = 5.8$, $p = 0.019$). Again, no effect of physical activity level (or interaction) was found on the timing of activation (for all $F < 1$ and $p > 0.319$).

Spinal maps

The Fig. 5A presents the EMG signals mapped onto the estimated rostrocaudal location of the motor neuron (MN) pools in the spinal cord. The timing of activation of the lumbar and sacral motor pools, estimated using the centre of activity (CoA), was different between young and older participants (lumbar: $F_{1,55} = 4.8$, $p = 0.032$; sacral: $F_{1,55} = 22.0$, $p < 0.001$). Indeed, the lumbar CoA occurred later whereas the sacral CoA occurred earlier. As a result, the delta between lumbar and sacral activations was reduced in older adults ($F_{1,55} = 23.4$, $p < 0.001$). While no effect of physical activity was observed on individual muscle activation patterns (Fig. 5), an interaction was found between age and level of physical activity for the lumbar CoA ($F_{1,55} = 15.6$, $p < 0.001$), with less active older participants displaying a later activation of the lumbar motor pools than more active elders (post hoc: $p < 0.001$), without difference observed between less and more active young participants (post hoc: $p = 0.082$). In turn, the delta between lumbar and sacral CoA was also smaller for less active older adults than more active ones (interaction: $F_{1,55} = 8.8$, $p = 0.003$; post hoc: $p = 0.003$).

The burst durations of the spinal motor output were also affected by age, in both lumbar ($F_{1,55} = 12.2$, $p = 0.001$) and sacral segments ($F_{1,55} = 7.3$, $p = 0.008$). Most likely due to longer bursts, the co-activation level (CI) between the lumbar and sacral motor pools was greater in older adults

($F_{1,55} = 21.4$, $p < 0.001$). While no effect of physical activity level on burst duration was found in both spinal levels ($p > 0.05$), the co-activation was higher in less active individuals ($F_{1,55} = 4.7$, $p = 0.034$), with a higher CI in less active older adults than more active ones (post hoc: $p = 0.029$).

The mean activity of both the lumbar and sacral segments was higher in older adults ($F_{1,55} = 26.6$, $p < 0.001$; $F_{1,55} = 21.8$, $p < 0.001$), but the ratio between lumbar and sacral activation was similar in both age groups ($F_{1,55} = 3.9$, $p = 0.053$). No significant effect was found for the physical activity level in the mean activation of the spinal segments (lumbar: $F_{1,55} = 0.7$, $p = 0.616$; sacral: $F_{1,55} = 0.7$, $p = 0.384$) or in the ratio between lumbar and sacral activation ($F_{1,55} = 0.002$, $p = 0.966$).

Discussion

In this study, we provide new insights into how a higher level of physical activity can influence the age-related changes observed during walking. Gait in older adults is often characterized by a shorter step length, greater metabolic cost, and distal-to-proximal joint effort redistribution. Our results show that the less active older subjects significantly decrease their step length, increase the power at collision via proximal joints and increase their mechanical work during walking (by about ~20% relative to young adults; Fig. 2A) relative to more active individuals. While we confirm our hypothesis regarding the impact of aging on muscle mechanical properties and maximal strength, relatively few changes are observed between the most active individuals and the more sedentary ones. However, a modification in spinal motor output was observed in less active individuals, showing an accentuation of the previously documented effect of aging.

The metabolic cost of walking is an important variable of daily life that has been studied extensively. In systematic reviews, Das Gupta et al. (2019)[52] and Aboutorabi et al. (2016)[53] showed that the average net cost of walking was ~20% higher in older adults, associated with a loss of independence and an increased risk of morbidity and mortality[5–8]. Previous studies have found that mechanical work does not explain the increase in the metabolic cost of elderly walking since no significant changes have been observed[54,55]. Considering only the effect of age, we did not observe any significant change in mechanical work between younger and older individuals (Fig. 2A). However, considering the level of physical activity, we observed a 20% increase in mechanical work in less active older adults. This change can partly be linked to a decrease in the pendular mechanism of walking in less active older adults (Fig. 2A).

In a previous publication, we reported a similar reduction of energy recovery in older adults unable to accelerate the centre of mass forward and upward before double stance because of a lack of trailing leg push-off at the end of stance[35,56]. Consequently, the centre of mass accelerates after the foot contact of the leading limb[57]. Similarly, our results showed that the phase of negative power ($\dot{W}^-$) occurring after collision with the ground, followed by a first phase of positive power ($\dot{W}_1^+$) were higher in older adults, and specifically in less active ones (Fig. 2B). Both contribute to the vertical redirection of the centre of mass after foot contact (FC), via greater eccentric energy at the knee extensors of the leading limb[58,59] and were designated as the collision phase[51]. Also, a reduction of the second phase of positive power ($\dot{W}_2^+$) was reduced in older adults, suggesting a reduction of ankle propulsion in late stance[19,20]. Such distal-to-proximal redistribution with age has garnered considerable scientific attention for nearly 50 years[19,21–27], most often consider as the hallmark biomechanical ageing features of gait.

While one might expect to observe a change in propulsive force in more active individuals, the age-related reduction of propulsion was similar between groups. Similarly, it has been shown that resistance training consistently improves muscle strength and mitigates sarcopenia but fails to directly translate to improved propulsive power generation in walking[48]. Instead, the impact of physical activity level was mainly observed during the collision phases (Fig. 2B). Such increase has been associated with deviations from the specific adaptations of the locomotor apparatus to bipedal gait, including the erect posture of the trunk[60], straight leg during stance[61,62], and heel-to-toe rolling pattern of the foot[63,64]. Our results showed that less active older adults reduced the heel-to-toe rolling pattern, by decreasing dorsal flexion at foot contact (Fig. 3B). Also, less active individuals tend to increase knee flexion during stance and to increase the average trunk flexion (Fig. 3). All these modifications of kinematic strategy have been related to a greater impact under the leading limb during step-to-step transition[60,64], as in older adults. These alterations could stem from age-related declines in neuromuscular function, and the nervous system may adapt by modifying motor strategies to enhance stability and reduce the risk of falls.

Neural factors are likely to contribute as well to age-related motor impairment[36]. For example, changes in control strategy[65–67] have been reported in older adults. Indeed, compared to young adults, older adults exhibited greater activation of lower-limb muscles during walking[68], and higher co-activation of antagonist muscles across the ankle and knee potentially contributing to joint stiffening[65,69]. In our results, we observed a greater activation with age in *tibial anterioris* and gastrocnemius lateralis, as well as in knee extensors and flexors (Fig. 4). Also, the timing of muscle activation changed with age, being longer for both ankle flexor (TA) and extensor (SOL) muscles, and knee flexors (ST, BF) and extensor (RF), potentially increasing co-activation.

The greater muscle activation could be related to the decline in muscle quality. Aging muscles weaken (Fig. 1B), which results in an increase in activation and thus in more energy consumed per amount of force produced[70,71]. In addition, following the principle of motor unit recruitment, motor unit recruitment progresses from slow-twitch to metabolically more expensive fast-twitch fibres at higher activations[72]. While the effect of age is in accordance with previous published results, little to no modifications were observed in older adults based on their physical activity level, except for EMG amplitude. Indeed, some muscles (TA, vastus lateralis, GM and ES) were more active in less active individuals.

Interestingly, insights may be gained by looking into the recruitment and organization of motoneurons during locomotion. The spinal maps, assessed by mapping muscle activity onto the rostrocaudal location of motoneurons, have already revealed specific characteristics of normal aging[34–36,73,74]. As compared to young adults, the motoneurons activation is significantly wider in older adults and the delta between lumbar and sacral activation is reduced. Indeed, the lumbar and sacral activity centres are closer together (Fig. 5), inducing a higher co-activation. Such modification has been related to the lack of late push-off prior to toe-off from the trailing leg[35,36,74]. As for kinematics, it's interesting to note that we observed a deviation from the two features of motoneuron activation that have been associated with the most energy-efficient bipedal gait: (1) distinct bursts of activity in the lumbar and sacral segments and (2) short burst durations relative to gait cycle duration[74].

While many age-related changes in gait patterns have been related to physiological deficits in peripheral system function[75], our results suggest that changes in the way the central nervous system coordinates the movements also contribute. In addition, our results show that age-related changes are amplified in older individuals with lower levels of physical activity. For example, the CoA of lumbar motor pools occurred after touch down in less active older adults, as compared to the other groups for whom the activity on average occurred before touch down. It potentially reflects a lack of anticipation before the collision with the ground, as reported earlier in older adults with a higher risk of falls[76]. Also, greater co-activation is observed between the activity of the two motoneuron pools in older adults less active, compared to active ones (Fig. 5B). This co-activation reflects the widening of spinal motor output, which has been proposed as a conservative strategy aimed at maintaining gait stability[77,78]. This seems to support our hypothesis that engaging in physical activity helps slow down the degradation of the neuromuscular system. Indeed, it's plausible that regular physical activity may mediate the age-related changes in the central nervous system, and in turn affects movement control strategy[78,79].

The modification of control strategy is also coupled with other intrinsic factors, such as weaker muscles and softer muscle-tendon units in older adults[59]. Indeed, both knee and ankle extensor maximal isometric torque was reduced with age (Fig. 1B), and individuals with less physical activity displayed reduced ankle extensor maximal forces. Reduced physical activity

typically results in skeletal muscle atrophy and an associated reduction of maximal voluntary force[80]. Numerous alterations in the physiological properties of the locomotor apparatus have been observed with aging[81]. Using a predictive neuromechanical model to investigate the physiological origins of age-related gait changes, Song and Geyer (2018) showed that the loss of muscle strength and muscle contraction speed dominantly contribute to the reduced walking economy[82]. The smaller ankle maximal force of less active individuals may exacerbate age differences and induce a greater redistribution of lower extremity joint moment and power compared with more active ones. Such results could explain why vigorous exercises, such as power training or running, prevents the age-related deterioration of muscle[83] and, make everyday activities easier[84].

In addition to strength, age-related changes in the mechanical and morphological properties of lower-limb muscle-tendon units have been reported[85]. Our results also showed that the muscle-tendon stiffness, measured via myotonmetry, was affected by age: the stiffness of rectus femoris and gastrocnemius lateralis was reduced and the dampening (oscillation attenuation) was increased in older adults (Fig. 1A). It has been suggested that age-related changes in neuromuscular activity (higher coactivation) reflect a strategy of stiffening the limb during stance likely to compensate the reduced muscle-tendon stiffness, to utilize tendon elasticity effectively[86]. Indeed, it is suggested that the muscle fascicle behaviours can be related to the motor strategy of older adults to stiffen joints and stabilize motor output in an effort to compensate for reduced muscle strength and for increased tendon compliance and joint laxity[87]. Interestingly, a higher level of physical activity in older adults reduced the differences between young and older adults, suggesting a positive impact of the level of activity (Fig. 1A). In addition to the change in strength, this effect of physical activity may contribute to the modification of motor strategy observed between less and more active older individuals, in particular the reduction of co-activation between lumbar and sacral segments (Fig. 5).

When comparing older adults according to fall history (non-fallers versus fallers groups), modification of gait and neuromuscular system between groups was similar to the one observed in the present study between less and more active older adults. Indeed, fallers showed shorter stride length[88], increased cost of walking, higher co-activation of lower-limb muscles[69] and modified motor control strategy[89]. In addition, muscle stiffness of the *gastrocnemius medialis* was significantly lower in the fallers group[90] and older adult fallers had 28% lower knee extensor strength[69]. Here, we did observe a more variable static balance between young and older participants (Fig. 1C), but no effect of physical activity level. However, given that fallers are presumably less active due to their mobility limitations and fear of falling, it is plausible that their reduced activity levels directly influence these neuromuscular deficits. These findings strengthen our results by highlighting that physical activity plays a crucial role in mitigating age-related changes in gait and neuromuscular function. This reinforces the idea that maintaining or increasing physical activity in older adults can help preserve gait mechanics, and muscle function, and potentially reduce the risk of falls.

The primary limitation of this study is that the level of physical activity was assessed using a self-reported questionnaire, which is inherently less accurate than objective measures such as VO_2 max[91], daily step count[92], or accelerometer-based activity tracking[93]. These more precise methods could provide a deeper understanding of the relationship between physical activity and gait parameters. However, we believe that our approach also serves as a strength of the study. Despite using a simple classification of activity levels, we observed significant modifications in gait and neuromuscular function related to physical activity. In addition, the individuals classified as 'less active' performed an average of 1473.6 METs min/week, exceeding the World Health Organization recommendations[94]. This reinforces the robustness of our findings and supports the main conclusion of the paper, suggesting that even small increases in physical activity could have meaningful effects on gait. For example, transitioning from a lower activity level to a more active group, with just a few additional hours of sports per week, could result in significant improvements in gait mechanics and mobility for less active older adults.

Another limitation of the study is the use of a treadmill. Walking on a treadmill may potentially reduce gait variability as compared to over-ground gait[95]. However, treadmill walking has been commonly used to investigate age-related modification of gait[34,56,76], and spatiotemporal, kinematic, kinetic, muscle activity, and muscle-tendon outcome measures are largely comparable between motorized treadmill and overground locomotion[96].

In conclusion, the recognition that the mechanical cost of walking is not purely age-dependent, but rather a consequence of reduced physical activity and subsequent skeletal muscle changes, underscores the vital importance of maintaining physical activity across the lifespan to mitigate the effects of aging on mobility. Indeed, our findings demonstrate that the level of physical activity significantly influences age-related changes in gait and neuromuscular function. While some of these changes are an inherent part of the aging process[97], a substantial portion can be attributed to the decline in physical activity levels with advancing age[98]. Our results underline the critical role of regular physical activity in preserving gait mechanics and muscle function in aging. Therefore, incorporating structured training programs that enhance physical activity levels could be an effective strategy to improve walking performance, reduce walking costs, and delay mobility impairments in older adults[99]. Such interventions[100] may ultimately contribute to reducing the risk of falls and promoting greater independence as people age.

Methods

Participants

Fifty-nine healthy young and older adults (♂ = 39, ♀ = 20) participated in this study. Both age groups were divided into two groups (less and more active), according to the median level of physical activity, measured through the Global Physical Activity Questionnaire (GPAQ) (Fig. 6). A modified z-score using the median and Median Absolute Deviation (MAD) was applied to provide a robust classification of participants into "Less Active" and "More Active. The four groups' characteristics are presented in Table 1. Sample size was based on an a priori power analysis (effect size = 0.4, power >80%) from Dewolf et al. [34], based on unpublished correlation between physical functioning and step period. Our test indicated that 12 subjects per group would be sufficient. The inclusion criteria were the following: ability to walk a kilometre, no locomotor system injury complaints and no previous history of neurological disorders. All participants provided informed consent, and the procedures used for this study were approved by the ethics committee of the Université Catholique de Louvain (Belgian Registration Number: B403201524765) and adhered to the Declaration of Helsinki.

Experimental procedure and data collection

Postural control and gait analyses were conducted using an instrumented treadmill (H/P Cosmos-Stellar treadmill, Germany, belt surface: 1.6 ×0.65 m) equipped with four force transducers (Arsalis®, Belgium). The transducers were placed under the body of the treadmill, the force transducers measured the three components of ground reaction force (GRF) exerted by the treadmill belt under the foot[101]: F_v, F_f, and F_l, respectively the vertical, fore-aft, and lateral components of the GRF. Data were sampled at a frequency of 1000 Hz.

Electromyographic (EMG) muscle activity from 12 muscles on the right side of the body was captured at a frequency of 2048 Hz using a Delsys Trigno Wireless System (Boston, MA). The muscles registered were the ES at the L2 level, the TFL, the Gmed, the vastus medialis (VM), the vastus lateralis (VL), the RF, the TA, the ST, the BF, the GL, the GM, and the SOL. Prior to electrode placement, the skin was prepared by shaving and cleaning with fine sandpaper, ether, and alcohol following guidelines from SENIAM (the European project on surface EMG—seniam.org). To ensure accurate placement, the muscle bellies were located by palpation, and electrodes were aligned with the main direction of the muscle fibres (66). Electrode placement and signal quality were verified through visual inspection of EMG

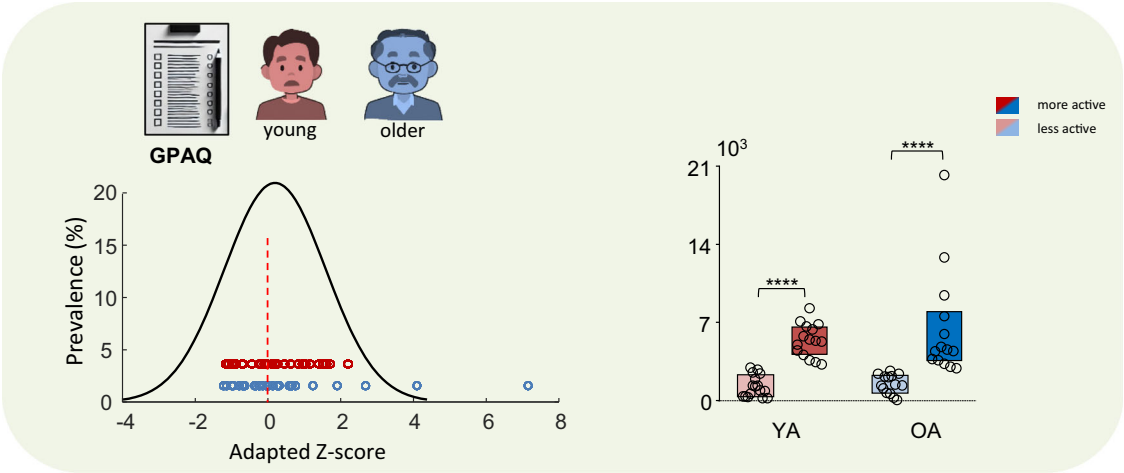

Fig. 6 | Group classification based on physical activity levels. Left panel: Distribution of physical activity levels in MET-min/week in young (red dots) and older adults (blue dots), classified using the Global Physical Activity Questionnaire (GPAQ). Older adults had a broader distribution of activity levels compared to the younger group. An adapted Z-score was applied to normalize activity levels. In this way, participants with a positive/negative adapted Z-score were classified as more/less active. *Right panel:* Boxplot displaying the average MET-min/week scores per group. The statistical significance of the differences between groups was assessed using the Kruskal-Wallis test. The asterisks indicate post hoc comparisons between groups (*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, ****$p < 0.0001$).

signals while participants contracted each muscle. EMG data and kinetic measurements were synchronized using a Delsys Trigger Module. Bilateral, full-body three-dimensional kinematics were recorded using a Qualisys system (Gothenburg, Sweden) equipped with 14 Oqus 600+ cameras and 4 Miqus M3 cameras placed around the treadmill. Participants were equipped with 20 retro-reflective markers, placed bilaterally on specific anatomical landmarks: neck, shoulders, wrists, posterior superior iliac spines, anterior superior iliac spines, greater trochanters, knees, malleoli, elbows, and the fifth metatarsal bones, according to the Qualisys sports marker set disposition.

The participants were first asked to stand on an instrumented treadmill to perform a static balance test. They stand with their feet together, eyes closed, for 30 s each. During the test, they were instructed to maintain their arms relaxed at their sides, avoid compensatory movements, and stand as still as possible. The forces exerted by the ground on the feet of the participant during standing were measured on the instrumented treadmill. From the GRF, the lateral and fore–aft positions of the centre of pressure (CoP) were computed as follows:

$$CoP_x = \frac{-M_y - hF_x}{F_z}$$

$$CoP_y = \frac{-M_x - hF_y}{F_z}$$

Where F_x, F_y, and F_z are the lateral, fore-aft, and vertical components of the GRFs; M_x and M_y are the moment components in the force transducer coordinate system; and h is the vertical distance between the force transducers and the tread surface[102]. The range of CoP_x and CoP_y oscillation was then determined.

Following the balance test, biomechanical properties of the *gastrocnemius medialis* (MG) and *rectus femoris* (RF) were measured using a handheld, non-invasive MyotonPRO device (Myoton AS, Tallinn, Estonia). Specifically, we measured these muscles 'dynamic stiffness and dampening (logarithmic decrement) by applying a mechanical impulse and recording the muscle's response[103]. We measured the biomechanical properties of the *rectus femoris* (RF) and *gastrocnemius medialis* (GM) at locations where reliability has been previously confirmed. Specifically, the RF was measured at two-thirds of the distance between the anterior superior iliac spine (ASIS) and the superior pole of the patella[104] and the GM was measured at the most reliable site identified by Nguyen et al. [105]. Five successive measurements were taken for each muscle, while participants were standing still. Subsequently, the participants were asked to walk wearing their shoes on an instrumented treadmill at 1.11 m s^{-1} (4 km h^{-1}). The selected walking speed was similar to the one used in our previous studies on older adults[34,35,56,106] and was reported as the comfortable and economical average walking speed based on the net energy consumption[61]. Before starting data collection of walking, participants familiarized themselves with treadmill walking through multiple trials with verbal feedback from the experimenter. After the familiarization period, we gradually ramped up the treadmill to 4 km h^{-1} and then waited at least 30 s before starting the kinematics, GRF and electromyographic data acquisition. On average, 28.1 ± 2.9 (mean ± SD) strides were analysed per participant.

After completing the walking trial, the participants performed Isometric Maximal Voluntary Torque (IMVT) measurements for the knee and ankle extensors. The isometric strength tests were performed using a 1D strain gauge (Arsalis®, Belgium) to measure torque[107], on a custom-designed table equipped with adjustable straps. The IMVT protocol began with a warm-up, during which participants performed incremental repetitions. Following the warm-up, the participants were instructed to exert their maximum effort for 6 s. Three trials were recorded, and the highest torque was analysed. The IMVT data were sampled at 500 Hz. For the knee extensor torque measurements, participants were seated with their hips, knees and ankles positioned at a 90° angle, secured with adjustable straps, and a rigid rope was fixed horizontally between the ankle and the gauge fixed on the wall[108]. For ankle extensor torque, the participants were seated in the same position, but with the shank fixed vertically using adjustable straps. A rigid rope was fixed vertically between the fifth metatarsal and the strain gauge, fixed on the wall. Participants were instructed to avoid trunk flexion to ensure accurate torque measurements. The protocol and setup were adapted from Dragoi et al. [109].

Data analysis

Division of the stride. The first instant of FC and the toe-off (TO) events were estimated from the displacement of the centre of pressure on the belt[110]. The FC and TO were automatically identified based on the computation of d_{path}, which represents the difference between the CoP trajectory and a straight-line approximation connecting its initial and final positions within a step cycle[110]. A stride was delimited by two successive right FCs. A step was defined as the interval between one leg's FC and the contralateral leg's FC. Stance phases were measured as the time between FC and TO of the same leg.

Posture. The lateral (CoP_x) and anteroposterior (CoP_y) displacement of the centre of pressure (CoP) were analysed for 30 s standing with closed eyes. A 4th-order low-pass Butterworth filter was applied with a 20 Hz cut-off frequency[111]. Specifically, the ranges of CoP displacement in both directions (CoP_x and CoP_y) were determined as two standard deviations (± 1 s.d.) of the time series, following the approach described by Dewolf et al. [111], providing a measure of the postural oscillation around the average position of the CoP.

Walking kinetics. Data were recorded at a sampling rate of 1000 Hz. The fore-aft and vertical velocity of the CoM were determined from the fore-aft and vertical components of the GRF using the procedure described in detail in Dewolf et al. (2016)[112]. In short, the fore-aft acceleration of the CoM was calculated as $a_f = F_f/m$, where m is the subject's body mass. The vertical acceleration of the CoM was calculated as $a_v = (F_v\text{-}m\,g)/m$, where g is the acceleration of gravity. The vertical (V_v) and the forward velocity (V_f) of the CoM were calculated by time-integration of a_v and a_f, respectively, plus an integration constant, which was computed so that the average velocity over a stride was equal to zero. The vertical and forward displacements of the CoM (S_v and S_f, respectively) were then computed by time-integration of V_v and V_f.

The energy of the CoM (E_{com}) was computed as the algebraic sum at each instant of its gravitational potential energy ($E_p = m\,g\,S_v$) and its kinetic energy ($E_k = ½\,m\,(V_f^2 + V_v^2)$). The work done to move the CoM relative to the surroundings (W_{com}) was then computed as the sum of the positive increments of E_{com}[50]. The energy transduction between E_p and E_k was estimated from the relative amount of energy saved over a step (%*recovery*):

$$\%\textbf{\textit{recovery}} = \frac{W_k + W_p - W_{com}}{W_k + W_p} * 100$$

where W_k and W_p correspond to the sum of the positive increments of E_k and E_p, respectively. The external power P_{com} was computed as $P_{com} = dE_{com}/dt$. During a walking stride, the P_{com} time-curve typically shows one major negative peak (W^-) and two major positive peaks (W^+_1 and W^+_2), known as collision and propulsion, respectively[51]. The maximum values of these peaks were measured for all trials.

Kinematics. Kinematic data were recorded at a sampling rate of 240 Hz and later oversampled during post-processing to 1 kHz, to match the kinetic sampling frequency. The elevation angle of each segment (trunk, thigh, shank, and foot), defined as their orientation relative to the vertical[102,113] were computed, with 0° corresponding to alignment with the vertical axis during walking. Joint angles for the hip, knee, and ankle were calculated based on the relative orientation between adjacent segments. Each stride was interpolated over 400 points, time-normalizing the data across all trials for each subject.

EMG. The 12 raw EMG signals were resampled at 1000 Hz to match the sampling frequency of the kinetic data. The signals were high-pass filtered at 30 Hz, then rectified, and subsequently, low-pass filtered using a zero-lag 4th-order Butterworth filter at 10 Hz. Additionally, a semi-automatic artefact detection procedure was implemented. A notch filter at 50 Hz was applied to remove frequency-related artefacts, such as power line interference and its harmonics. Then, gait cycles containing evident artefacts, such as signal loss or saturation due to sensor detachment, were manually excluded following visual inspection. Finally, an outlier detection method was applied: individual gait cycles were flagged for further review if their correlation coefficient with the ensemble-averaged EMG envelope fell below 0.6, indicating substantial deviation from the typical signal pattern[114]. The time scale was normalized by interpolating each gait cycle to 400 points. The root mean square (RMS) of each EMG signal was then computed as a measure of overall muscle activation amplitude across the gait cycle. For each condition, the FWHM was determined as the duration during which the EMG activity exceeded half of its maximum value[78,115]. The centre of activity (CoA) of each EMG signal was measured as an estimation of the timing of activation. The CoA during the gait cycle was calculated using circular statistics[116] and plotted in polar coordinates (polar direction denoted the phase of the gait cycle, with angle α that varies from 0 to 360°). The CoA of the EMG waveform was calculated as the angle of the resultant vector (i.e., the first trigonometric moment) pointing to the centre of mass of the circular distribution of EMG activity relative to the gait cycle[115], computed as:

$$A = \sum_{i=1}^{400} (\cos \alpha_i XEMG_i),$$

$$B = \sum_{i=1}^{400} (\sin \alpha_i XEMG_i),$$

$$CoA = \tan^{-1}(B/A)$$

where EMG_i represents the EMG amplitude at each phase of the gait cycle. The CoA was preferred, as identifying a clear peak of activity was impractical for most muscles[115].

The EMG activity was then mapped onto the rostro-caudal positions of the MN pools in the human spinal cord, ranging from segments L2 to S2. This mapping followed the myotomal charts of Kendall et al. [117], based on the approximate rostro-caudal location of MN pools innervating different muscles in the human spinal cord. In general, each muscle is innervated by several spinal segments. To reconstruct the output pattern of any given spinal segment S_j, all rectified EMG waveforms corresponding to that segment were averaged as:

$$S_j = \frac{\sum_{i=1}^{n_j} k_{ij} \cdot EMG_i}{n_j},$$

where n_j is the number of EMG_i waveforms corresponding to the j^{th} segment, k_{ij} is the weighting coefficient for i^{th} muscle (from Kendall's chart). The assumption implicit in both methods is that the rectified EMG provides an indirect measure of the net firing of MNs of that muscle in the spinal cord. To compute the total motor output for each condition, the motor output patterns across the gait cycle were summed across the lumbar, sacral and all spinal segments. The mean activation of the lumbar (L2 to L5) and sacral (S1 to S2) segments was computed by averaging the motor output patterns for each region. Finally, the FWHM and the CoA were calculated for both lumbar and sacral segments. The co-activation index (CI) was assessed between the lumbar and sacral segments using the following formula[118,119]:

$$CI = \frac{\sum_{j=1}^{400} \left\{ \left[lumbar_j + sacral_j \right] / 2 \right\} x \left[Lumbar_j / sacral_j \right]}{400},$$

where sacral and lumbar represent the mean activation of the segments during the stride. The CI was then averaged over the entire gait cycle (j = 1:400), providing an estimate of the co-activation over the entire cycle. High CI represent a high level of activation of both spinal segments across a large time interval, whereas low CI indicate either a low-level activation of both segments or a high-level activation of one segment along with low-level activation of the other one. Spinal-level co-activation provides a functional representation of neuromuscular output, as previously used to assess age-related co-activation[35].

IMVT. A custom-made MATLAB script was utilized to accurately determine the peak force generated during the 6-second maximal voluntary contraction (MVC) trial. The script processed the raw data, applying a low-pass filter at 20 Hz. After filtering, the peak torque was identified as the maximum force maintained for at least half a second. This approach avoids the detection of a short peak of force, providing a

precise measurement of the participant's maximal torque output during the isometric test. The lever arms were measured using anthropometers.

Statistics

For each dependent variable (stance duration, step length, external work of the CoM, recovery, peak negative power, peak positive power 1 and 2 of the CoM, amplitude of CoP_x and CoP_y, elevation angles, joint kinematics, foot angle at touchdown, peak knee flexion, muscle activation metrics (RMS, FWHM, CoA), spinal motor outputs, co-activation index, myoton properties, and IMVT torque measurements), the normality of the residuals was visually assessed using QQ plots. For variables that did not meet the normality assumption, a log10 transformation was applied, and normality was confirmed afterwards. A mixed-effects model was performed with age (young *vs.* older adults) and physical activity level (less *vs.* more active) as fixed effects. A post-hoc Fisher LSD multiple comparisons test was used to evaluate differences between age groups and levels of physical levels. The interaction between the fixed effect was reported if it was significant. All statistical analyses were conducted using SPSS 23 (IBM, USA) with an alpha level of 0.05.

Data availability

The data that support the findings of this study are available from the corresponding author, AHD, upon reasonable request.

Code availability

The code used for data processing and analysis in this study is available upon reasonable request from the corresponding author.

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Author contributions

The experiments were conducted within the laboratory of physiology and biomechanics of human locomotion—UCLouvain. All authors (A.D. and M.N.) contributed to the conception of the work, the acquisition, analysis and interpretation of data and to writing of the work. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Competing interests

The authors declare no competing interests.

Additional information

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