Maximal shortening velocity during plantar flexion: Effects of pre-activity and initial stretching state

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We investigated the effects of the initial length of the muscle-tendon unit (MTU) and muscle pre-activation on muscle-tendon interactions during plantarflexion performed at maximal velocity. Ultrasound images of *gastrocnemius medialis* were obtained on 11 participants in three conditions: (a) active plantarflexion performed at maximal velocity from three increasingly stretched positions (10°, 20°, and 30° dorsiflexion), (b) passive plantarflexion induced by a quick release of the ankle joint from the same three positions, and (c) pre-activation, which consisted of a maximal isometric contraction of the plantarflexors at 10° of dorsiflexion followed by a quick release of ankle joint. During the active condition at maximal velocity, initial MTU stretch positively influenced ankle joint velocity (+15.3%) and tendinous tissues shortening velocity (+37.6%) but not the shortening velocity peak value reached by muscle fascicle. The muscle fascicle was shortened during the passive condition; however, its shortening velocity never exceeded peak velocity measured in the active condition. Muscle pre-activation resulted in a considerable increase in ankle joint (+114.7%) and tendinous tissues velocities (+239.1%), although we observed a decrease in muscle fascicle shortening velocity. During active plantarflexion at maximal velocity, initial MTU length positively influences ankle joint velocity by increasing the contribution of tendinous tissues. Although greater initial stretch of the plantarflexors (ie, 30° dorsiflexion) increased the passive velocity of the fascicle during initial movement, its peak velocity was not affected. As muscle pre-activation prevented reaching the maximal muscle fascicle shortening velocity, this condition should be used to characterize tendinous tissues rather than muscle contractile properties.

KEYWORDS
elasticity, gastrocnemius, passive velocity, plantar flexion, tendon, ultrasound, unloaded shortening

1 | INTRODUCTION

The capacity of the human muscle to produce maximal shortening velocity is one of the main factors determining maximal power output and, in turn, performance in numerous motor tasks. Using ultrafast ultrasound technology, Hauraix et al[1] recently assessed, for the first time in vivo, maximal ankle joint angular velocity and fascicle shortening velocity of *gastrocnemius medialis* during very fast dynamic plantar flexion (ie, maximal velocity plantar flexion performed on a specific rotational low inertia footplate with no external resistance). However, to measure the actual maximal shortening velocity of fascicles, it is mandatory to clearly document the contributions of elastic elements to the MTU shortening

Nicolas Beaumatin and Hugo Hauraix have contributed equally to this work.

process. This would provide a better understanding of the role of muscle-tendon interactions and how rapid concentric contractions are performed in vivo. Thus, three additional issues remained to be examined.

First, in the above-mentioned maximal velocity protocol,[1] the authors reported a large contribution of tendinous tissues (ie, around 40%) to the total muscle-tendon unit velocity. In the absence of a stretch-shortening cycle and considering the ankle positioned at −20° before the maximal velocity plantar flexion (0° being the neutral position, positive/negative correspond to plantar/dorsiflexion), the authors attributed this contribution to the initial stretched state of tendinous tissues.[2] This participation should be influenced by the amount of stretching of tendinous tissues and would hence directly influence the maximal joint velocity reached. Currently, no study has examined the influence of the initial ankle position on the magnitude of the contribution of these passive structures and on the fascicle-tendon interactions during fast angular motions.

Second, the maximal fascicle shortening velocity measured during the maximal velocity protocol, which was significantly related to the maximal joint velocity,[1] can be considered to nicely reflect the maximal velocity contractile property of the muscle (ie, the cross-bridge cycling rate). However, some in vitro studies have demonstrated that, when the muscle fiber is very stretched before the contraction, elastic elements present in the sarcomere can also substantially participate to shorten the muscle fiber and induce an increase of the unloading shortening velocity.[3-6] Then, in vivo, an important initial stretching state of the muscle-tendon unit would induce a significant contribution of sarcomere elastic elements to the shortening velocity of muscle fascicles during the subsequent ballistic contraction. This consideration strengthens the need to study the influence of the initial ankle position on the muscle fascicles' passive velocities in vivo to better understand how they contribute to the fascicle shortening velocity.

Third, a maximal recruitment of motor units requires approximately 300 ms,[7] while a maximal velocity plantar flexion performed against very low external resistance could only last 100-150 ms.[8] Therefore, some authors have suggested that the muscle should be isometrically pre-activated[9,10] to obtain a more complete recruitment. In line with this, a higher maximal ankle velocity was reported in movements in the presence of pre-activity[11] (up to 1300°/s) compared to without pre-activity[1] (700-800°/s). However, the pre-activity level should largely influence both the muscle fascicles' and tendinous tissues' initial lengths and their contributions to the subsequent maximal velocity plantar flexion. This point is crucial because, while pre-activity would maximize the contribution of tendinous tissues, its effect on fascicle shortening velocity has never been appraised.

Therefore, the first aim of this study was to investigate the influence of the initial prestretching of the muscle-tendon unit during an active maximal velocity plantar flexion. We hypothesized that the increased initial dorsiflexion angle would: (a) increase the joint velocity by enhancing the contribution of the passive tendinous tissue structures; and (b) increase the muscle fascicle shortening velocity due to the increased influence of elastic structures included in bundles of muscle fibers. The second aim was to examine the influence of muscle pre-activity on muscle fascicle-tendon interactions. We hypothesized that this pre-activity would improve joint velocity while failing to improve muscle fascicle shortening velocity.

2 | METHODS

2.1 | Participants

Eleven healthy males (22.4 ± 3.7 years, 179.8 ± 8.0 cm, 75.0 ± 10.5 kg) volunteered to participate in the study. All participants were engaged in a physical activity, from recreational to high-level competition (eg, sprinting, running, soccer, basketball, and swimming). The participants were fully informed about the nature and aim of the study before giving their written consent to be included in the study. They had no history of neurological or musculo-skeletal pathology. Approval for the project was obtained from the local ethics committee. This study was conducted according to the Declaration of Helsinki.

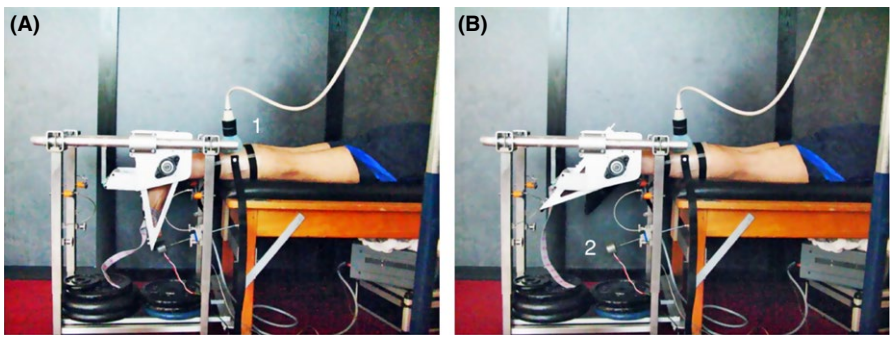

FIGURE 1 Specific ergometer used for the maximal velocity plantar flexion protocol with the ankle in the starting dorsiflexion position (A) and at the end of the movement (B). The ultrasound probe (1) was placed over the *gastrocnemius medialis* muscle belly. The pedal was held by the electromagnet (2) prior to the release. Here, the starting position was set at −10°

2.2 | Ergometer

High-velocity plantar flexions were performed on a specific ergometer (Bio2M, Compiègne, France Figure 1) composed only of a bench and a light weight rotational pedal,[12] to reduce the moment of inertia as much as possible (0.0327 Nm s^2/rad,[13]). The moment of inertia of the foot was calculated using the parallel axis theorem proposed by de Leva[14] and amounted to an average 0.065 Nm s^2/rad. Additional details of these procedures are provided as Data S1. Ankle angle was measured with an optical absolute encoder. An electromagnet was used and adjusted to maintain the starting position in different dorsiflexion angles: −10°, −20°, and −30° (0° = neutral position, positive/negative correspond to plantar/dorsiflexion). The ankle position signal was sampled at a frequency of 1000 Hz using a 16-bit A/D converter (PowerLab 16/35, ADInstruments, Dunedin, New Zealand).

2.3 | Ultrasound measurements

An ultrasound scanner (Aixplorer, Supersonic Imagine, Aix-en-Provence, France) was used to observe fascicle behavior of *gastrocnemius medialis* during plantar flexions with a frame rate of 2000 Hz. A linear probe (5-12 MHz, 55 mm) was placed on the skin surface over the muscle belly at 30% of the distance between the top of the lateral malleolus and the lateral femoral epicondyle, vertically to the muscle fascicles and perpendicular to the skin. This probe was securely attached to the right leg with custom-made equipment to avoid any displacement of the device during the protocol (Figure 1). Ultrasound measurements were synchronized with mechanical data using an external trigger of the ultrasound scanner recorded by the acquisition system of the mechanical signals (PowerLab, ADInstruments, Dunedin, New Zealand).

2.4 | Protocol

Before completing a standardized warm-up, the subjects laid on the bench in a prone position with legs fully extended (Figure 1). The right ankle was firmly attached and secured on the pedal platform, and the ankle's rotation axis was adjusted to the axis of the pedal. Warm-up was composed of five submaximal isometric plantar flexions, three maximal voluntary contractions (MVC), and several trials of maximal velocity plantar flexions to become familiarized with future tasks.

First, subjects performed two trials of the pre-activity maximal velocity condition with a starting position fixed at −10° (pre-active). From visual feedback, the subjects performed isometric plantar flexions at 80% of MVC. Once torque was stable (approximately 3-4 seconds), the electromagnet was switched off manually to apply a quick release of the joint.[11] A 0.5 seconds ultrasound acquisition was automatically triggered by a trigger-out signal from the magnet.

Participants then performed two trials of active plantar flexions at maximal velocity without pre-activity (active) and two trials of passive plantar flexions induced by a quick release of ankle joint (passive) at three different starting angles (−10, −20 and −30°) in a randomized order, with one minute of rest between each trial. For the active trials, after a 3-seconds countdown, the subjects were instructed to contract "as fast as possible" throughout the range of motion (until 56°, Figure 1). The torque produced by the electromagnet was adjusted to compensate the passive torque produced in each starting position and to minimize the potential isometric phase prior to the movement of the pedal. A trial was discarded if an initial torque >5% of MVC was observed. For the passive trials, a full release of the pedal was performed by the shutdown of the electromagnet's power. The subjects were instructed to relax as much as possible and were not informed about the release instant to minimize any unintentional muscle pre-activity. If there was any suspicion of active contraction, the trial was carried out again.

2.5 | Data analysis

Data processing was fully described in a previous study.[1] Briefly, raw data were analyzed using custom MATLAB scripts (MathWorks, Inc, Natick, MA, USA). Ankle joint angle was low-pass filtered by a zero phase second-order Butterworth filter with a cutoff frequency of 150 Hz. At the end of the experiment, shank and *gastrocnemius* length were measured with the knee angle at 90° and ankle angle in neutral position. The instantaneous length of the *gastrocnemius medialis* muscle-tendon unit (L_{MTU}) was calculated using the anthropometric model proposed by Grieve et al[15] based on knee (considered as constant throughout the experiment, 0°) and ankle angle measurements and shank and gastrocnemius length (for more details, see Data S1).

Ultrasonic raw data were used to create B-mode images by applying a conventional beam formation (ie, applying a time-delay operation to compensate for travel time differences). Typical video of B-mode images is available in Videos S1-S3 for each condition tested for starting angle at −10°. The MATLAB software developed by Cronin et al[15] was used to automatically track the behavior of muscle fascicles, superficial, and deep aponeuroses. When the muscle fascicle was not fully visible, muscle fascicle length (L_F) was extrapolated.[1,9,16] Muscle fascicle length was low-pass filtered by a zero phase second-order Butterworth filter with a cutoff frequency of 40 Hz. The tendinous tissue lengths (ie, tendon and aponeurosis) were considered as the difference between muscle-tendon unit length and horizontal muscle fascicle

length. Initial and final values of fascicle and muscle-tendon lengths were also calculated for each trial. The shortening velocity of muscle fascicles, tendinous tissues, and the muscle-tendon unit was calculated using first derivation. Data were analyzed between the beginning of the movement (ie, when the pedal started to rotate) and the end of plantar flexion. Data were then interpolated through the range of motion over 100 equally spaced points.

2.6 | Statistical analysis

All data being normally distributed (Shapiro-Wilk's test), two-way ANOVAs (condition × starting position) were performed to assess the statistical changes of initial fascicle length, final fascicle length, angle for the peak fascicle shortening velocity, peak joint velocity and peak shortening velocities of fascicles, tendinous tissues, and muscle-tendon unit (2 conditions: active and passive; 3 starting ankle's angle positions: −10, −20, and −30°). A Tukey post-hoc analysis was conducted when appropriate. The effect of pre-activity was separately tested using paired t tests for initial and final ankle angle, fascicle and tendinous tissues lengths, peak joint velocity and peak shortening velocities of fascicles, tendinous tissues, and muscle-tendon unit during the 100° condition performed with (pre-active at −10°) and without (active at −10°) pre-activity.

3 | RESULTS

3.1 | Effect of initial prestretching

Average patterns of the ankle joint, muscle-tendon unit, muscle fascicle, and tendinous tissues length changes in all conditions are depicted in Figure 2. Average initial and final values of muscle fascicle and MTU lengths during active and passive conditions performed at −10, −20, and −30° are shown in Table 1. Two-way ANOVA showed a significant main effect of condition and starting position on initial value of fascicle length ($P = .015$ and $P = .002$, respectively). The condition × starting position interaction was not significant ($P = .892$). Significant effects were found for the final fascicle length for condition ($P < .001$) and starting position ($P = .023$) without significant condition × starting position interaction ($P = .442$). Initial fascicle length values were increased from −10° to −30° for both active and passive conditions (+0.8 cm, Table 1).

Two-way ANOVA showed a significant main effect of starting position on initial value for MTU length ($P < .001$). There was an average 1.2 cm additional lengthening of MTU with the increase of initial dorsiflexion from −10° to −30° ($P < .001$, Table 1). There was no significant condition effect ($P = .559$) or condition × starting position interaction ($P = .899$). Significant main effects were observed for the final MTU length for condition ($P < .001$) and starting

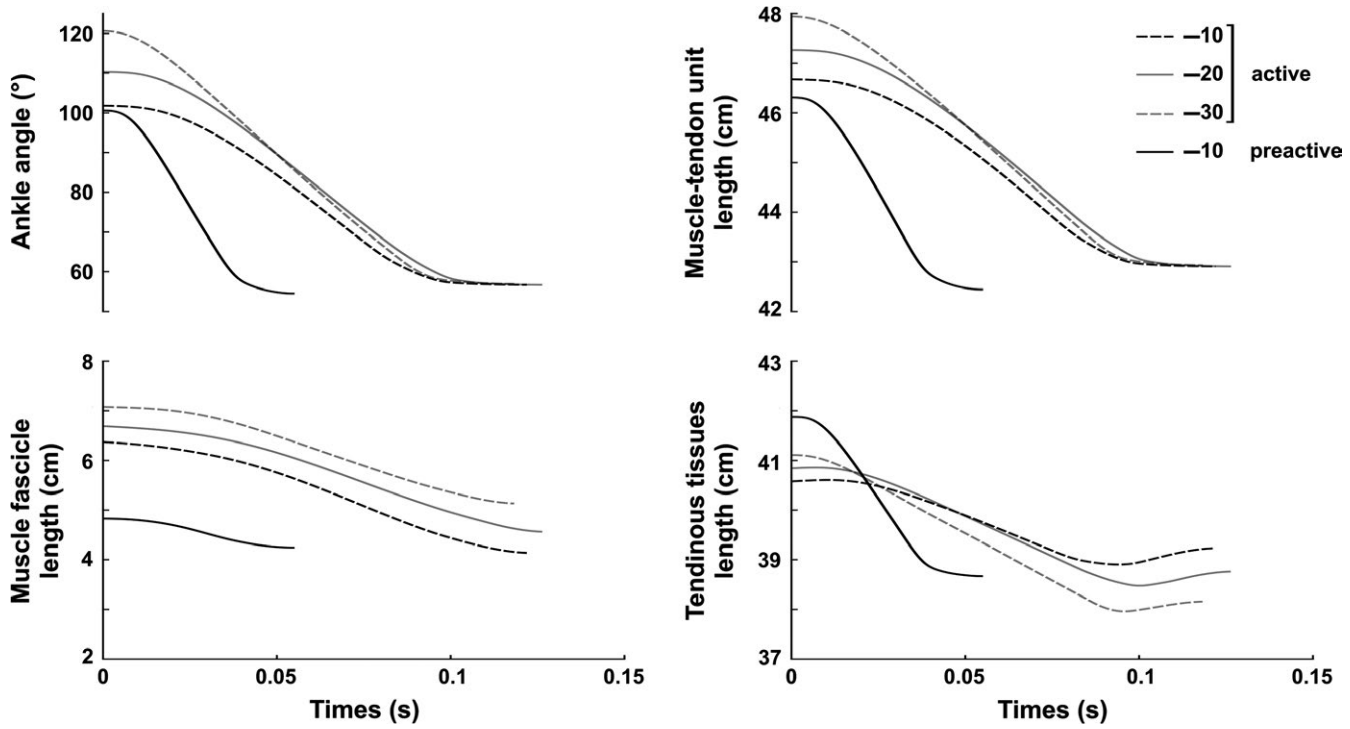

FIGURE 2 Mean changes in ankle angle (A), muscle-tendon unit length (B), muscle fascicle length (C), and tendinous tissue length (D) during maximal velocity plantar flexions performed against no external load and without pre-activity (active) at three different initial ankle angles (−10°, −20°, and −30°) and performed with pre-activity (a near maximal isometric contraction at −10° followed by a quick release of ankle joint, pre-active). Profiles are displayed as mean (without SD, for the sake of clarity). Negative values correspond to dorsiflexion position in °

position ($P = .039$) with a significant condition × starting position interaction ($P = .038$).

Average patterns of the ankle joint, muscle fascicles, and tendinous tissues shortening velocity are presented in Figure 3. We observed that values obtained during passive conditions were systematically lower than during active conditions, except for fascicle velocity over the first 20° in the −30° condition. The range of motion in passive conditions decreased with the reduction in ankle starting angle. Tendinous tissue velocity remained positive during the whole movement, which means that no lengthening of the tendinous tissues was induced during the plantar flexion.

TABLE 1 Summary of initial and final lengths of muscle fascicle (L_F) and muscle-tendon unit (L_{MTU}) for active (maximal velocity plantar flexions performed against no external load) and passive (passive release) conditions for the three initial ankle angles (−10, −20 and −30°). Values are means ± SD, in centimeters

Length (cm)		Initial		Final	
Condition	Starting angle (°)	Active	Passive	Active	Passive
L_F	−30	7.1 ± 1.0	7.6 ± 1.3*	5.1 ± 1.1	6.4 ± 1.7*
	−20	6.7 ± 0.8	7.1 ± 1.0*	4.6 ± 1.0	6.1 ± 0.9*
	−10	6.4 ± 1.2[a]	6.7 ± 1.2*[,a]	4.1 ± 1.1[a]	5.9 ± 1.4*[,a]
L_{MTU}	−30	47.9 ± 2.9	47.9 ± 2.9	43.0 ± 2.7	44.0 ± 3.5*
	−20	47.2 ± 2.9[a]	47.2 ± 2.9[a]	43.0 ± 2.7	44.4 ± 3.0*
	−10	46.6 ± 2.9[a,b]	46.6 ± 2.9[a,b]	43.0 ± 2.7	44.9 ± 3.1*

*$P < .05$ represents a significant differences from active condition.
[a,b]$P < .05$ represent significant differences from −30° to −20°, respectively.

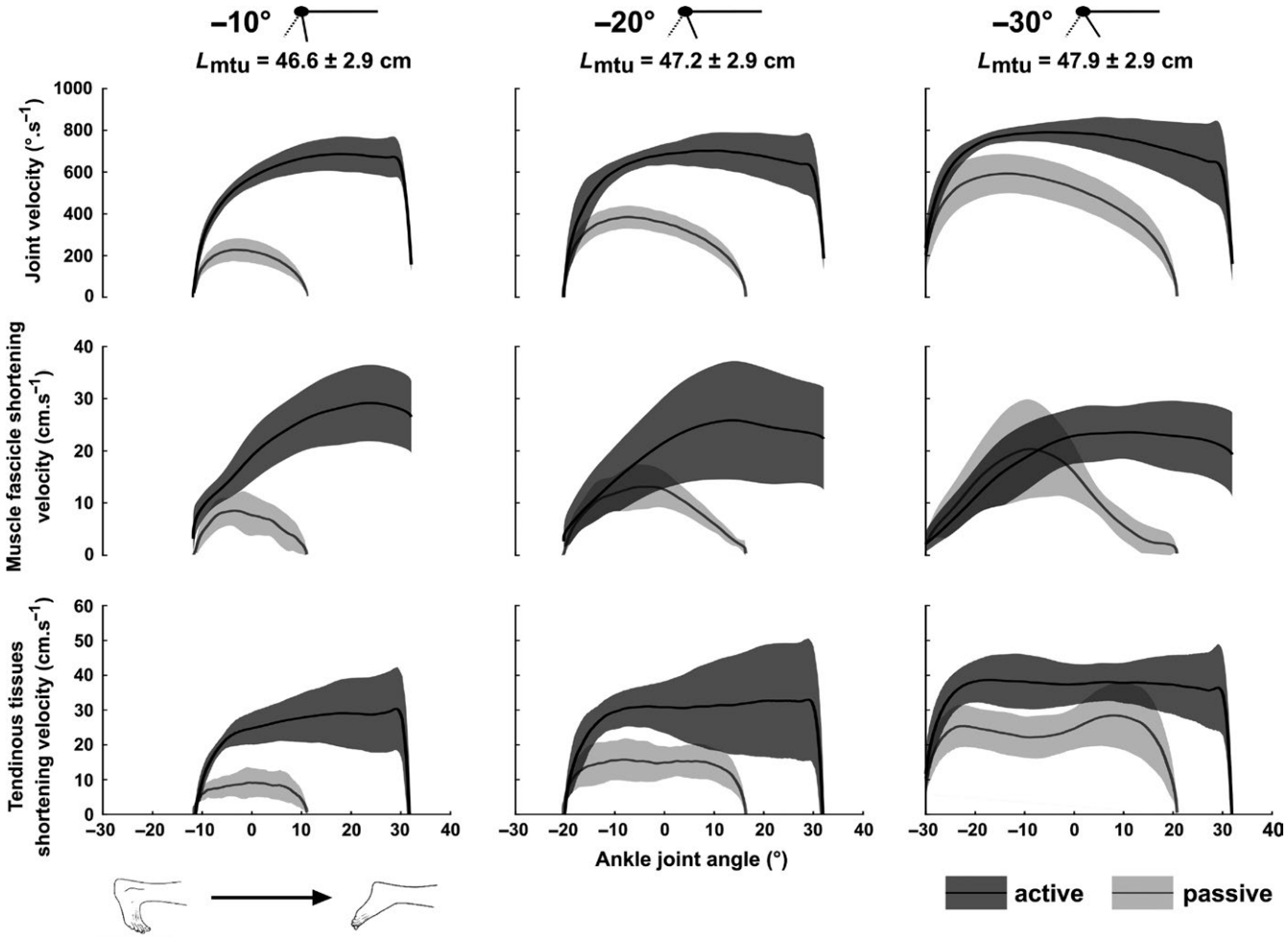

FIGURE 3 Mean patterns (±SD) of angular velocity and muscle fascicle and tendinous tissue shortening velocities during maximal velocity plantar flexions performed against no external resistance (active, dark gray) and passive release (passive, light gray) conditions in three imposed initial starting positions of the ankle (−10, −20, and −30°). Mean value of the initial Muscle-tendon unit (MTU) length in each condition is mentioned at top

The two-way ANOVA demonstrated a significant main effect of both condition and starting position on peak joint velocity ($P < .001$) and a significant condition × starting position interaction ($P < .001$). During active conditions, peak joint velocity was significantly higher at −30° than at the two other starting positions ($P = .003$, Figure 4). In passive conditions, peak joint velocity significantly increased with the increase in the starting position angle ($P < .001$). Regardless of the starting position tested, peak joint velocity was achieved later during active conditions in comparison with passive conditions (93.1°, 76.2°, and 69.3° vs 104.1°, 96.7°, and 92.9° for the starting angles −30°, −20°, and −10°, respectively, $P < .001$, Figure 4). The two-way ANOVA demonstrated a significant main effect of condition ($P < .001$), and a significant condition × starting position interaction ($P < .001$) for peak fascicle shortening velocity. Starting position did not affect peak fascicle velocity during active conditions ($P = .677$, Figure 4). However, it increased significantly with the increase in starting position in passive conditions ($P = .035$ and $P = .007$). The peak shortening velocities reached by muscle fascicles were always higher in active compared to passive conditions, regardless of the starting angle tested ($P < .001$).

We observed significant effects of condition and starting position on tendinous tissues shortening velocity. Although close to significance level, the interaction condition × starting position was not verified ($P = .058$). The values of maximal shortening velocity of tendinous tissues increased with the increase in starting position, regardless of the condition tested (Figure 4, $P < .001$). We obtained lower values in passive than in active conditions ($P < .001$).

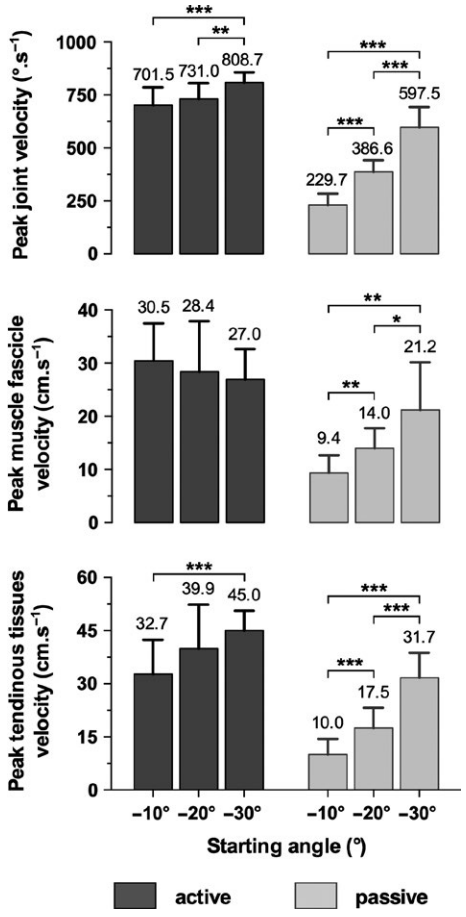

FIGURE 4 Effects of the Muscle-tendon units (MTU's) initial prestretch on ankle joint, muscle fascicle, and tendinous tissues peak velocities for active (maximal velocity plantar flexions performed against no external resistance) and passive conditions for the three initial ankle angles. Values are means ± SD. *$P < .05$, **$P < .01$, and ***$P < .001$ for significant differences between the starting positions

3.2 | Effect of muscle pre-activity

Initial muscle fascicle length was significantly lower during pre-active conditions compared to -10° active conditions (4.8 ± 1.2 cm vs 6.4 ± 1.2 cm, $P < .001$, Figure 2). Conversely, the initial tendinous tissue length was higher in pre-active compared to active conditions (41.9 ± 3.2 cm vs 40.6 ± 2.9 cm, $P < .001$, Figure 2). Average joint, fascicle, and tendinous tissue velocities in pre-active and active conditions performed with a starting ankle angle of −10° are presented in Figure 5. We observed that values obtained in pre-active conditions were systematically higher than active conditions for joint and tendinous tissues velocities. Maximal values of joint and tendinous tissues velocities also occurred earlier for pre-active conditions than active conditions ($P < .001$). Conversely, fascicle velocity always appeared higher during active than pre-active conditions.

Paired *t* tests demonstrated significant differences between pre-active and active conditions (Figure 6): muscle pre-activity induced significant increases in peak joint and tendinous tissue velocities (+804.8 ± 177.0°/s and +78.2 ± 11.5 cm/s, respectively, $P < .001$). Active conditions elicited higher peak fascicles velocity than the pre-active condition (30.5 ± 8.5 cm/s vs 21.7 ± 12.1 cm/s, $P < .001$).

4 | DISCUSSION

The present study has three major findings. First, the initial MTU stretching state prior the contraction greatly influenced the peak joint velocity achieved during active maximal velocity plantar flexion by enhancing the tendinous tissue (but not fascicle) shortening velocity. Second, passive elements, within the muscle fascicles marginally contributed to the shortening velocity, and this influence was limited to the beginning of the shortening phase when fascicles were highly prestretched (ie, −30° of ankle angle). Third, the presence of pre-activity preceding the maximal velocity contraction substantially increased the maximal joint velocity exclusively through the increase in tendinous tissue's shortening

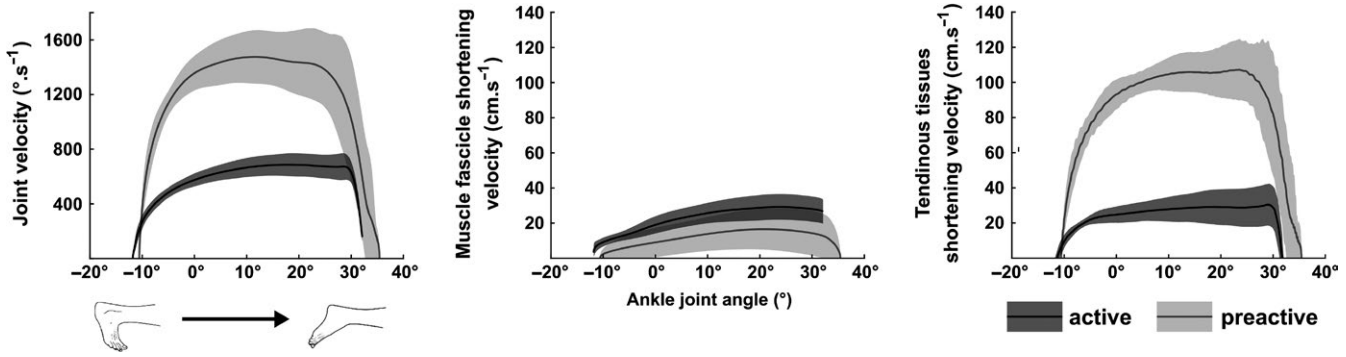

FIGURE 5 Mean patterns (±SD) of ankle joint, fascicle, and tendinous tissues shortening velocities measured during active (dark gray) and pre-active (light gray) conditions (initial ankle angle = −10°). See caption of Figure 2 for details of abbreviations

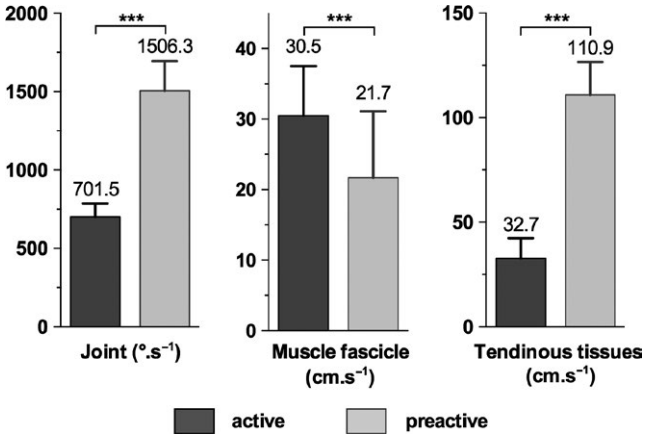

FIGURE 6 Effects of muscle pre-activity on ankle joint, muscle fascicle, and tendinous tissues peak velocities reached during active and pre-active. See caption of Figure 2 for details of abbreviations. Values are means ± SD. ***$P < .001$ for significant differences between active and pre-active conditions

velocity. This muscle pre-activity prevents the muscle fascicles reaching maximum shortening velocity.

4.1 | Effect of initial prestretching

In accordance with our hypothesis, the peak joint velocity reached during the active conditions was largely influenced by MTU length (ie, MTU prestretch) by approximately +4 and +15% at −20° and −30°, in comparison with the −10° condition, respectively. This is explained by the greater initial length of the tendinous tissues at −30° compared to −20° and −10° (Table 1). This induced an increase in tendinous tissue contribution to MTU velocity, of 51% at −10°, 54.8% at −20°, and 60.1% at −30° (Figure 3). These mean contributions were calculated for a range of motion between 0° and 20° of ankle angle, in the same manner as reported by Hauraix et al.[1] In the current study, although peak MTU shortening velocity increased with the starting dorsiflexion angle, this was not the case for the peak shortening velocity of muscle fascicles. The peak muscle fascicle shortening velocity was always reached during the second part of the movement (Figure 3) when fascicles operated at almost the same length whatever the starting angle position, that is, just around the optimal length [5-6 cm[17]]. This result is consistent with in vitro studies showing that the maximal fiber shortening velocity (V_0 measured by the slack test method) is not influenced by sarcomere length within a relatively large range around the optimal length (between 1.65 and 2.7 μm[3]). Therefore, from a practical point of view, a starting ankle position angle between −10° and −30° can be used to consistently determine the maximal muscle fascicles shortening velocity of *gastrocnemius medialis* using the protocol proposed by Hauraix et al.[1]

Given that an extreme dorsiflexion position could also induce an important lengthening of sarcomere-based passive structures, a "passive" velocity may contribute to fascicle shortening velocity and contribute to the ankle velocity. Indeed, in vitro studies show an increase in elastic energy release resulting from an increased passive tension in these noncontractile structures of the fibers under stretching.[3,5,6] Thus, passive conditions were examined for the three starting angle conditions to isolate this participation. In accordance with our hypothesis, a shortening velocity of the muscle fascicles also exists during these passive releases and directly depends on the amount of the initial prestretching (Figures 3 and 4). At the anatomical level, the specific contributions of structures to the passive shortening of muscle fascicles remain to be clarified. Minajeva et al[4] showed that, from a specific stretching state of the sarcomere, the titin protein allows the creation of a passive shortening velocity in isolated myofibrils. In addition, it is quite probable that extrasarcomeric collagenous tissues such as perimysium and endomysium also contribute significantly.[18] Assuming that these structures are placed in parallel with the contractile elements in a Hill-type model, their contributions to the muscle fascicles velocity during an active plantar flexion would be effective only if they reach higher values than the contractile component. In the current study, it is interesting to note that the peak muscle fascicle shortening velocity achieved

in passive conditions never exceeded those reached in active conditions (Figure 4). Moreover, it is important to note that the peak fascicle shortening velocity in active conditions was systematically reached later in the movement. As the fascicles are too short to create a passive force by in-parallel structures at these angles[19] (ie, 10°-20° of plantarflexion, Figures 2 and 3), we can assume that it did not influence the peak fascicle shortening velocity reached in this phase. These results demonstrate that the parallel elastic elements of the muscle fascicle would not contribute to the maximal shortening velocity of the plantarflexors in vivo. Thus, the methodology proposed in the present study and previous recent work to measure the maximal fascicle shortening velocity[1] can be considered as reliable to evaluate a maximal velocity representing the muscle "contractile" property.

For extreme sarcomere lengths (ie, longer than 2.7 μm, ~130% of the optimal length), Edman et al[3] showed that the maximal fiber shortening velocity is higher than the velocity reached around the optimal length, demonstrating the involvement of the sarcomere's elastic elements. Despite a comparable initial stretch of muscle fascicles (~120%-140% of L_0 in the 120° condition, considering 5 cm < L_0 < 6 cm, Hoffman et al[17]), in vivo results obtained in the current study are not in agreement as they did not demonstrate an increase in peak shortening velocity of the fascicles. This is certainly partly explained by the residual resistance due to the moment of inertia of the system (ie, approximately 0.1 Nm s^2/rad). Although this inertia was kept as small as possible, the ankle joint acceleration is limited at the beginning of movement, and a torque is inevitably produced by the muscle. That means that the first degrees of rotation did not fully correspond to an actual unloaded condition, explaining the inability of muscle to reach a peak shortening velocity earlier. It is therefore possible that the present setup failed to completely unload the muscle fibers immediately after the onset of contraction when elastic elements would possibly exceed the unloaded active shortening velocity.[4] Notwithstanding, the rate of fascicle shortening velocity rise in this early phase of contraction at the extreme angle (ie, −30°) was slightly higher in passive than in active conditions (Figure 3, right middle panel). This can be explained by: (a) an incomplete recruitment of motor units in this phase in active condition performed without pre-activity; but also (b) the fact that the contractile system would produce a substantial braking force when the speed of shortening induced by the passive structures in the fascicles is very high.[3]

4.2 | Effect of muscle pre-activity

During a ballistic maximal contraction, it may be difficult to maximally recruit all muscle fibers due to the short duration of the movement itself (ie, 100-150 ms). Numerous have authors suggested to pre-activate the muscle in an isometric condition[9,10] to facilitate a complete recruitment during the subsequent contraction. As expected and previously reported,[11] the maximal joint velocity during the condition with muscle pre-activation was significantly higher than during active condition (1506 vs 701°/s, respectively, Figure 6). However, the results of the present study demonstrate that this difference is fully explained by the storage of elastic energy in tendinous tissue during the isometric contraction and its release during the quick release movement. This experimental condition elicited more than 3 times the tendinous tissue shortening velocity reached in active condition (111 vs 33 cm/s), while muscle fascicle shortening velocity was reduced by 1.4 on average (20 vs 30 cm/s). This result showed that the presence of pre-activation fails to enhance the muscle fascicle shortening velocity and significantly limits the ability of muscle fascicles to maximally shorten.

While this result remains unexplained, it should be noted that muscle fascicle operated at a shorter length range in pre-active condition (4.8-4.2 cm) compared to the active condition (6.4-4.1 cm), because of different initial states between both conditions. We propose two main hypotheses to explain why such initial states with short fascicle lengths may prevent the ability to reach high shortening velocity. First, a short muscle length was demonstrated as a limiting factor to reach the maximal unloaded shortening velocity.[3] However, this was only found for very short lengths for isolated frog muscle fibers (<1.65 μm of sarcomere length, ie, <75%-80% of optimal length). In the present study, we estimated the initial fascicle length at 80%-95% of the optimal length (considering 5 cm < L_0 < 6 cm,[17]) in the condition with muscle pre-activity. Therefore, it is not clear whether the short fascicle lengths alone can explain our results. Second, the dynamic changes in *gastrocnemius medialis* muscle geometry may limit fascicle shortening velocity in vivo. Indeed, the pre-activity induces increases in both the muscle thickness and pennation angle. Thus, the 2D fascicle motion during the maximal velocity plantar flexion should be very different between both conditions. For instance, the increase in thickness at high pennation angle in the in pre-activity condition may lead to a resistance to fascicle shortening inside the muscle, and to a reduction in the muscle fascicles shortening velocity.

Fascicle-tendon interactions observed in the pre-activity condition induce fascicle shortening, muscle thickening, and tendon stretch. This behavior is very efficient to produce high ankle velocity during the subsequent maximal velocity plantar flexion thanks to the release of tendon elastic energy. However, it also prevents the achievement of the highest muscle fascicle shortening velocity. This finding increases the relevance of the quick release method (using pre-activity) to describe the properties of series elastic element rather than the muscle's contractile properties.[11] Finally, although the issue of a maximal recruitment in the absence of pre-activity is not completely resolved, we can observe that: (a) the peak

value of the muscle fascicle shortening velocity is always reached before the end of the movement; and (b) this value is similar, regardless of starting angle, while the total time of contraction differed. These findings show that the additional time for fully activating the muscle in a more dorsiflexed starting position does not lead to an increase of the velocity. As a whole, it allows us to speculate that a maximal recruitment may be likely possible in the classical active maximal velocity condition despite an absence of pre-activity (provided a starting angle at least equal to 10° of dorsiflexion).

5 | CONCLUSION AND PERSPECTIVES

The present study demonstrated that the initial stretching state of the muscle strongly influences the peak angular velocity of the ankle joint and shortening velocity of the tendinous tissues, but does not increase muscle fascicle velocity during a maximal velocity plantar flexion with no external resistance. In addition, the passive velocity due to elastic elements of sarcomeres exists but is marginal in vivo and limited to the initial range of movement for conditions with an initial large MTU stretch (ie, extreme dorsiflexion starting angle). Overall, this process should not influence the peak muscle fascicle shortening velocity, which is achieved in the second phase of the maximal velocity plantar flexion. Finally, our findings highlight that muscle pre-activity enhances joint velocity due to a large increase in tendinous tissue contribution while maximal muscle fascicle shortening velocity is reduced. Therefore, maximal contraction against no external resistance performed in a pre-activated condition can be confidently considered as a relevant procedure to appraise the series elastic properties,[11] while contractions performed without pre-activity[1] are confirmed as an effective method to assess "active" maximal fascicle shortening velocity.

The method presented here to estimate maximal velocity of the fascicle might be easily reproduced to further characterize the mechanical properties of other pennate muscles, as was recently done for *vastus medialis*.[20] Moreover, in the area of training and rehabilitation prescription, different methods have been considered with the aim of increasing maximal power by improving maximal velocity capabilities. For instance, ballistic, high-acceleration movements with minimal[21] or even negative[22] external loads have been recently proposed for squat exercise. One of the practical perspectives of this work would be to investigate whether such specific training can actually enhance the maximal fascicle shortening velocity of the lower limb muscles and/or participate to improve the maximal power by altering the tendinous tissue properties and optimizing muscle-tendon interaction.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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