Human medial gastrocnemius force–velocity behavior shifts with locomotion speed and gait

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Humans walk and run over a wide range of speeds with remarkable efficiency. For steady locomotion, moving at different speeds requires the muscle–tendon units of the leg to modulate the amount of mechanical power the limb absorbs and outputs in each step. How individual muscles adapt their behavior to modulate limb power output has been examined using computer simulation and animal models, but has not been studied in vivo in humans. In this study, we used a combination of ultrasound imaging and motion analysis to examine how medial gastrocnemius (MG) muscle–tendon unit behavior is adjusted to meet the varying mechanical demands of different locomotor speeds during walking and running in humans. The results highlighted key differences in MG fascicle-shortening velocity with both locomotor speed and gait. Fascicle-shortening velocity at the time of peak muscle force production increased with walking speed, impairing the ability of the muscle to produce high peak forces. Switching to a running gait at 2.0 $m{\cdot}s^{-1}$ caused fascicle shortening at the time of peak force production to shift to much slower velocities. This velocity shift facilitated a large increase in peak muscle force and an increase in MG power output. MG fascicle velocity may be a key factor that limits the speeds humans choose to walk at, and may explain the transition from walking to running. This finding is consistent with previous modeling studies.

muscle mechanics | biomechanics | preferred transition speed

Ankle plantar–flexor muscles are a vital source of mechanical power for human locomotion (1, 2). During walking, plantar–flexor muscles provide body weight support, contribute to propulsion, and accelerate the limb into swing (3). In running, the ankle acts in a spring-like manner, absorbing energy in plantar–flexor muscle–tendon units during early stance and providing energy to accelerate the body in late stance (1). The mechanical work required to produce whole-body movement during walking and running varies between gaits and across speeds (4, 5). Thus, the plantar–flexors may need to adjust their mechanical work output with gait and speed to meet the changing demands on their contribution to total mechanical work.

A recent experimental study in humans used an inverse-dynamics approach to examine how the mechanical power outputs of muscles acting at the hip, knee, and ankle joint were modulated for walking and running at a range of speeds (6). It was found that positive power output at the ankle, in conjunction with the knee and hip, increased with walking speed. Also, Hansen et al. (7) showed that at walking speeds above those preferred, the net positive work done at the ankle increased. When switching from walking to running gait, the relative contribution of ankle positive power output to total positive power output also increased (6). It was inferred from these data that plantar–flexor muscle mechanics were adjusted to accommodate faster walking speeds and then again with the switch to running gait. If this is truly the case, then it may have implications for locomotor economy and motor control/feedback mechanisms. For example, it might help to explain why humans prefer to walk at moderate speeds [$\sim$1.2 $m{\cdot}s^{-1}$ (8)], and why we switch to running at speeds where it would consume less metabolic energy to walk [$\sim$ 2.0 $m{\cdot}s^{-1}$ (9)]. However, an inherent limitation of inverse-dynamics analyses is the inability to determine the contributions of individual muscles to joint mechanics. Furthermore, the major plantar flexors (gastrocnemius and soleus) have highly pennate fascicles that insert on the calcaneous through the very compliant Achilles tendon and aponeurosis. This architecture decouples the actions of the muscle fibers from the mechanics of the whole muscle–tendon unit (10), and thus the separate contributions of fascicles and elastic tissues are hard to determine. Therefore, to truly understand how plantar–flexor muscle mechanics are adjusted with speed and gait, studies at the muscular level are necessary.

Modeling studies of human locomotion have provided insight into the changing function of the plantar–flexor muscle–tendon units with increasing locomotion speed. In support of the inferences made above, modeling studies of human walking have predicted that increasing speed requires increased work from the plantar flexors (11). However, similar models also showed that as walking speed increased, so did plantar–flexor fiber-shortening velocities, and this impaired plantar–flexor force production (12). Because of this result, Neptune and Sasaki (12) proposed that degeneration of plantar–flexor muscle fibers' ability to produce force with walking speed was a factor in triggering the transition from walking to running. These authors showed in their modeling studies that human plantar–flexor muscle fiber-shortening velocity decreased after switching to a running gait at the preferred transition speed. This improvement in contractile conditions allowed the plantar flexors to produce much greater forces when running compared with walking at the same speed (12). Although modeling results have provided excellent new insights, the need has been recognized for in vivo evidence to support their findings (13, 14).

Support for modeling studies comes from in vivo data recorded in animal models. Using sonomicrometry and tendon buckle transducers, Prilutsky et al. (15) showed that the velocity of cat gastrocnemius fibers increased with walking speed, impairing gastrocnemius force production at faster speeds. In running guinea fowl (16) and turkeys (17), ankle extensors operate at low fascicle-shortening velocities, supporting the notion that running gait allows distal leg muscles to operate under favorable conditions for force production. However, these animal data come from varied species and may not translate well to humans. Also, the techniques used for measuring muscle mechanics are too invasive for routine use in humans.

The purpose of this study was to investigate the effects of locomotor speed and gait on the mechanics of the human medial gastrocnemius muscle–tendon unit in vivo. We aimed to acquire in vivo evidence to provide support for and help validate previous findings from modeling studies (5, 11, 12, 18). To achieve this, we combined ultrasound imaging of medial gastrocnemius (MG) muscle fascicles and an inverse-dynamics analysis during walking and running. Based on data from human modeling (12)

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and animal models (15), we examined two hypotheses: (*i*) MG fascicle velocity would increase with walking speed, impairing MG force production at faster speeds; and (*ii*) switching from fast walking to a running gait would slow down MG fascicle velocity and facilitate greater MG force production.

Results

The considerable decoupling of the length change of MG fascicles from that of the whole MG muscle–tendon unit (MTU) during the stance phase of walking was highlighted by their respective length change patterns (Fig. 1*A* and Fig. S1). This decoupling was facilitated by the lengthening (~15 mm) of the series elastic element (SEE) during early to midstance and its recoil in late stance (Fig. 1*A* and Fig. S1). As a result, MG fascicles were able to operate at low velocities relative to the whole MTU throughout stance (Fig. 1*B* and Fig. S1).

For all walking speeds, MG fascicle force peaked in late stance (50–60% of stride time; Fig. 1*B* and Fig. S1), coinciding with when MG fascicles were shortening at greater velocities than during the majority of the stance phase (Fig. 1 and Fig. S1). Peak MG fascicle force (F_{MGmax}) during stance was significantly (P = 0.01) less for walking at 2.0 m·s^{-1} than for walking at 1.25 m·s^{-1} (Fig. 2). This coincided with a significant (P = 0.01) increase in MG fascicle velocity at the time of F_{MGmax} (V_{MGmax}) (Fig. 2). Average MG fascicle velocity ($\bar{V}_{MG}$) during stance, average MG fascicle force ($\bar{F}_{MG}$) during stance, MG fascicle length at initial ground contact (L_i), and change in MG fascicle length (ΔL) were not different between walking speeds of 1.25 m·s^{-1} and above (Table 1).

During running at speeds of 2.0 m·s^{-1} and above, F_{MGmax} and $\bar{F}_{MG}$ increased significantly (P = 0.01) compared with walking at 2.0 m·s^{-1} (Fig. 2 and Table 1). This coincided with a significant (P = 0.01) reduction in V_{MGmax} (Fig. 2), although $\bar{V}_{MG}$ was not different (Table 1). Under all running conditions, the timing of F_{MGmax} (~ 20% of stride time; Fig. 1*G* and Fig. S2) was such that it coincided with some of the lowest MG fascicle velocities that occurred during the stance phase (Fig. 1*F* and Fig. S2). Similar to walking, the lengthening of the SEE during stance decoupled the MG fascicle length changes from that of the MTU (Fig. 1*E* and Fig. S2) and allowed the MG fascicles to shorten at lower velocities than the MTU and SEE during late stance (Fig. 1*F* and Fig. S2).

Increasing walking speed from 0.75 m·s^{-1} to 1.25 m·s^{-1} resulted in an increase in average positive power produced by the MTU ($\bar{P}^{+}_{MTU}$) (P = 0.001) brought about by an increase in average positive power produced by the MG fascicles ($\bar{P}^{+}_{FAS}$) (P = 0.017; Fig. 3). There was no change in $\bar{P}^{+}_{MTU}$ or $\bar{P}^{+}_{FAS}$ across walking speeds from 1.25 to 2.0 m·s^{-1}, and the average positive power produced by the SEE ($\bar{P}^{+}_{SEE}$) was constant across all walking speeds (Fig. 3). For running, there was no difference in $\bar{P}^{+}_{FAS}$, $\bar{P}^{+}_{SEE}$, and $\bar{P}^{+}_{MTU}$ between any speeds (Fig. 3). However, running at 2.75 m·s^{-1} and 3.25 m·s^{-1} involved significantly (P = 0.01) more $\bar{P}^{+}_{MTU}$ and $\bar{P}^{+}_{SEE}$ than any of the walking speeds (Fig. 3). In Fig. 3*C*, the total average positive power provided by the fascicles and the SEE is broken down into the percentages provided by the SEE and fascicles.

Discussion

We examined two hypotheses: (*i*) MG fascicle velocity would increase with walking speed, impairing MG force production at faster walking speeds; and (*ii*) switching from fast walking to a running gait would slow down MG fascicle velocity and facilitate greater MG force production. The data provide support for both hypotheses, as V_{MGmax} increased at faster walking speeds, and this was associated with decreasing F_{MGmax}. Furthermore, changing to a running gait at 2.0 m·s^{-1} resulted in a reduction in V_{MGmax} and large increases in F_{MGmax}.

Overall patterns of MG fascicle length change across the walking gait cycle showed relatively isometric behavior during midstance followed by shortening in late stance (Fig. 2). This was associated with considerable stretch and recoil of the SEE (Fig. 2). This is in good agreement with studies using similar measurement techniques (19, 20) and modeling studies (12, 21) at normal walking speeds. There was a trend for increases in fascicle-shortening velocity with walking speed (Fig. 2 and Table 1) that agreed well with data from modeling studies of human walking over a similar range of speeds (12), providing good support for the predictions of this model. These increases in fascicle velocity may be important for MG force production during fast walking.

The MG is required to produce large forces late in the stance phase of walking to contribute to forward propulsion (3), accelerating the leg into the next step. The ability of MG to produce these forces is dependent on several factors. The maximum force a muscle fascicle can produce for a given activation is determined by its length and velocity. Given that in the present results there was no change in L_i or ΔL between walking speeds, it can be assumed that the fascicles remained on the same part of their force–length relationship (although where this is in relation

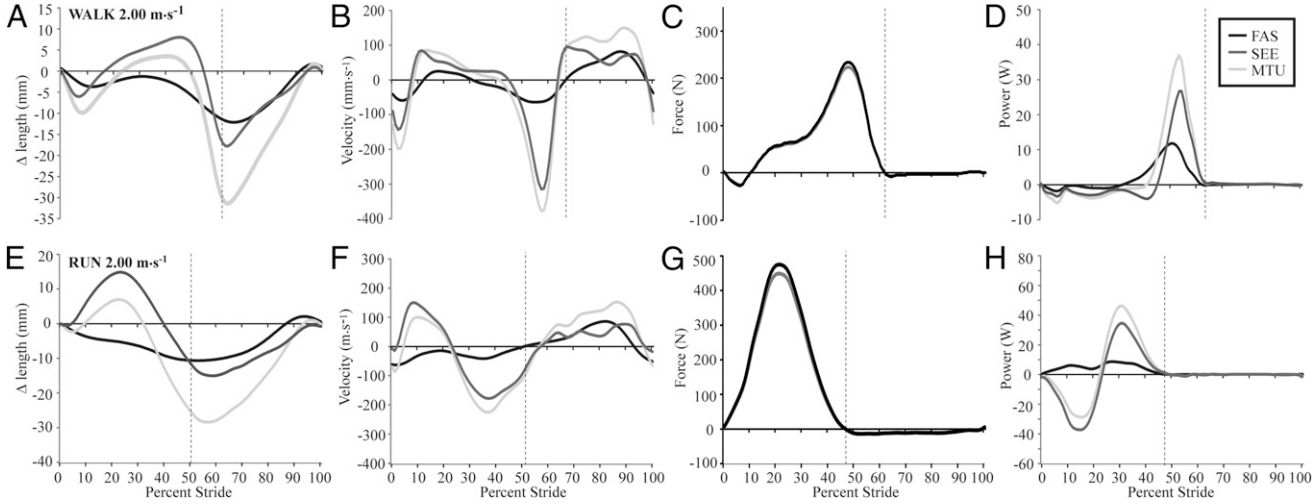

Fig. 1. Group mean medial gastrocnemius fascicle (black), series elastic element (dark grey), and muscle-tendon unit (light grey): length changes (*A* and *E*), velocities (*B* and *F*), forces (*C* and *G*; MTU force not plotted for clarity), and powers (*D* and *H*) during walking at 2.0 m·s^{-1} (*A–D*) and running (*E–H*) at 2.0 m·s^{-1}. All data are normalized to 101 points over an entire stride. The dashed vertical lines indicate the average time of toe-off for the analyzed limb. Error bars were omitted for clarity. These data are representative of trends for all speeds within each gait, and similar plots for all other speeds are included in Figs. S1 and S2.

Table 1. Group mean (±SEM) values for L_i, ΔL, $\bar{V}_{MG}$, $\bar{F}_{MG}$, and the maximum length of the SEE (Max L_{SEE})

	Walk				Run			
Speed ($m \cdot s^{-1}$)	0.75	1.25	1.75	2.0	2.0	2.25	2.75	3.25
L_i (mm)	47 ± 2	50 ± 2	50 ± 2	49 ± 2	48 ± 2	47 ± 2	46 ± 3	47 ± 2
ΔL (mm)	−7 ± 1*	−14 ± 1	−14 ± 1	−13 ± 1	−13 ± 2	−13 ± 1	−14 ± 2	−13 ± 2
$\bar{V}_{MG}$ ($mm \cdot s^{-1}$)	11 ± 2*	25 ± 3	33 ± 3	35 ± 2	34 ± 4	31 ± 4	27 ± 6	28 ± 4
$\bar{F}_{MG}$ (N)	171 ± 18*	195 ± 17	157 ± 9	130 ± 5*	219 ± 14†	200 ± 14†	199 ± 18†	187 ± 19†
Max L_{SEE} (mm)	373 ± 25	379 ± 22	374 ± 20	374 ± 22	381 ± 19†	384 ± 21†	383 ± 17†	382 ± 23†

*Significantly different from walking at 1.25 $m \cdot s^{-1}$ ($P = 0.001$).
†Significantly different from walking at 2.00 $m \cdot s^{-1}$ ($P = 0.002$).

to the shape of this relationship cannot be confirmed). Therefore, the reduction in MG force observed at fast walking speeds cannot be due to changes in the operating length of the fascicles. However, there was a significant increase in V_{MGmax} for walking at 2.0 $m \cdot s^{-1}$ compared with 1.25 $m \cdot s^{-1}$ that coincided with the aforementioned significant reduction in F_{MGmax} and $\bar{F}_{MG}$. This would be expected, given that muscle fibers are less able to produce force at faster shortening velocities (22). This result agrees well with the findings of modeling studies that also showed a reduction in gastrocnemius muscle forces at faster walking speeds that was partly due to increased gastrocnemius fiber-shortening velocities (12). Thus, our in vivo data support the previous suggestion from modeling studies that plantar–flexor muscles may suffer from less favorable contractile conditions at faster walking speeds and thus are unable to produce the same forces as at slower speeds (12, 15). Gastrocnemius is important in providing power for propulsion during walking, which involves rapid shortening of the whole MTU to produce high angular velocities of the ankle. Although the compliant Achilles tendon has been shown to lower the required shortening velocity of muscle fascicles during push-off at preferred walking speeds (19, 20), it may be unable to do so sufficiently at fast walking speeds.

Neptune and Sasaki (12) have suggested that the inability of the plantar–flexor muscles to provide push-off force might be a determinant of the preferred walk-to-run transition speed (PTS) that typically occurs close to 2.0 $m \cdot s^{-1}$. These authors found that a switch to running at the PTS improved muscle contractile conditions by slowing MG fiber velocity and shifting fiber length close to $\mathbf{L}_0$. The present data strongly support this idea, with fascicle velocity at the time of peak MG force being significantly reduced at all running speeds compared with walking at 2.0 $m \cdot s^{-1}$ (Fig. 2). For running, the fascicle velocities at peak force were close to isometric or even slightly lengthening. This was associated with a doubling of peak force values when switching from walking at 2.0 $m \cdot s^{-1}$ to running. To confirm that this increase in force was due to the reduction in fascicle velocity, muscle activation data are required, and activation was not measured in this study. However, Neptune et al. (11) also saw a large increase in force when transitioning to running, and this was achieved with relatively small increases in muscle activation because of greatly improved contractile conditions. Furthermore, published electromyography (EMG) data at the walk-to-run transition showed that average rectified MG EMG only increased by ~20% from fast walking speeds to slow running (23, 24). Given that the current study showed MG force to nearly double when transitioning from walking to running, it would seem likely that an improvement in contractile conditions would be necessary with such a small increase in activation.

The present data for human MG fascicles during running also agree well with previous in vivo measurements made on humans at one speed (7.5 $km \cdot h^{-1}$) that show the fascicles shortening throughout the stance phase (20). Muscle fascicle data for humans running at multiple speeds are scarce. Ishikawa and Komi (25) showed that MG fascicle length shortened with increasing speed, but their data were for large increments in speed, with all but one speed being faster than in the present study. Also, the runners in Ishikawa and Komi's data were instructed to switch from heel to toe running when increasing speed from the slowest speed (2.0 $m \cdot s^{-1}$) to the next fastest (3.5 $m \cdot s^{-1}$). None of the runners in the present study were toe strikers at the speeds performed. In the absence of comparable human data, some parallels can be drawn with in vivo data from other bipedal runners. Our data showed no change in fascicle velocity (average

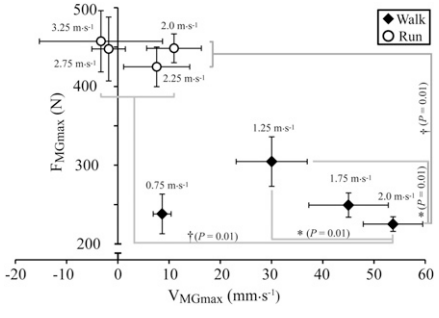

Fig. 2. Group mean (±SEM) F_{MGmax} plotted against V_{MGmax}. Walking (filled diamonds) and running (open circles) speeds are indicated for each data point. *Statistically significant difference from walking at 1.25 $m \cdot s^{-1}$. †Statistically significant difference from all running speeds (based on the repeated-measures ANOVA and post hoc Tukey's HSD).

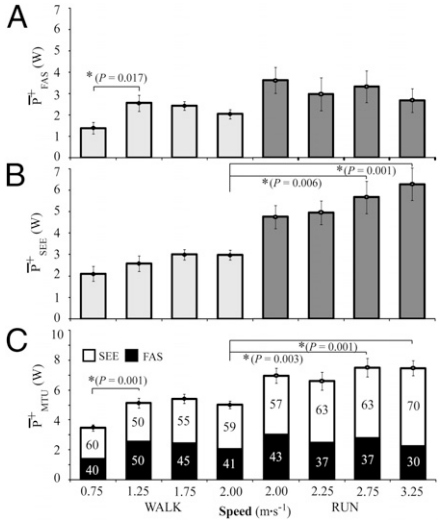

Fig. 3. (*A*) Average fascicle positive power ($\bar{P}^{+}_{FAS}$). (*B*) Group mean (±SEM) medial gastrocnemius average series elastic element power ($\bar{P}^{+}_{SEE}$). (*C*) Average positive MTU power divided into percentage contributions of $\bar{P}^{+}_{FAS}$ and $\bar{P}^{+}_{SEE}$. *Statistically significant difference.

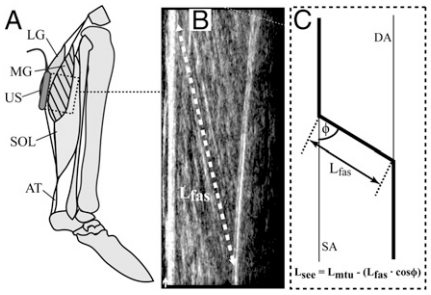

Fig. 4. (*A*) A schematic showing the muscles of the triceps surae (MG, LG, SOL), the Achilles tendon, and the placement of the ultrasound probe (US). (*B*) An example ultrasound image obtained of the medial gastrocnemius. (*C*) The geometry of the fascicle, superficial aponeurosis (SA), and deep aponeurosis (DA), including the formula for calculating series elastic element length based on fascicle length (L_{fas}), pennation angle (ϕ), and muscle–tendon unit length (L_{mtu}).

or at the time of peak force) with increasing running speed (Fig. 2 and Table 1). Running guinea fowl similarly show no change in MG fascicle velocity with running speed (16). These data support the assumption of Kram and Taylor (26) that muscles are working on a similar part of their force–velocity relationship across all steady-state running speeds. As such, our data help reinforce Kram and Taylor's hypothesis that the time course of generating force and the cost of supporting body weight during locomotion are the major determinants of the metabolic cost of running. However, the present data are only for one muscle with a relatively small physiological cross-sectional area. Data for more muscles involved in locomotion are required to relate fascicle velocities to the total metabolic cost of locomotion.

The force production of a muscle fascicle in conjunction with its length change determines its average power output or how much work it does. The length change of MG fascicles was consistent across all walking speeds, and so one might have expected $\bar{P}^{+}_{FAS}$ to have varied in a similar manner to $\bar{F}_{MG}$ with walking speed. However, although $\bar{P}^{+}_{FAS}$ followed a similar trend to $\bar{F}_{MG}$, the reduction in $\bar{P}^{+}_{FAS}$ from walking at 1.25 m·s^{-1} to 2.0 m·s^{-1} was not significant. In fact, $\bar{P}^{+}_{FAS}$ did not significantly change even when switching from a walk to a run. This was despite increased $\bar{P}^{+}_{MTU}$ between walking and running. Almost all of the increase in $\bar{P}^{+}_{MTU}$ when switching from walking to running came from increased $\bar{P}^{+}_{SEE}$. It seems that the increases in MG muscle force production that occurred with adopting a running gait facilitated a greater stretch of the SEE, which reached greater maximum lengths in running than for walking (Table 1). Consequently, the SEE could then return more energy as a contribution to increase $\bar{P}^{+}_{MTU}$ (Fig. 3 *B* and *C*). Fig. 3*C* shows that energy recycled by the SEE was responsible for 50–70% of $\bar{P}^{+}_{MTU}$ in walking and running. This agrees well with estimates made using exoskeletons (27) and modeling studies (11) during walking as well as ultrasound-based measurements during hopping (28, 29). This supports the growing evidence suggesting that the elastic recoil of the Achilles tendon and aponeurosis is responsible for a large proportion of the positive work done by the plantar–flexor MTUs during human locomotion.

Although it is not possible to determine from the present data, it is interesting to consider where the values calculated for fascicle force, length, and velocity during locomotion lie on the force–length or force–velocity relationships of human MG muscle. Length did not change between conditions and thus it was not important for the findings of this study, but it would be of interest to know which part of the force–length relationship humans are operating on during locomotion. The velocities reported were in the region of −0.01 to 0.06 m·s^{-1}. The resting length ($\mathbf{L}_0$) for MG is not known from the present study's data, but using a literature value of 55 mm (30) the velocity ranges from −0.2 to 1.1 $\mathbf{L}_0$·s^{-1}. This is well below the reported range of values for the maximum shortening velocity (V_{max}) of MG, which is 10–12 $\mathbf{L}_0$·s^{-1} (31). It seems that during walking and running, human MG operates at low percentages of its V_{max}, which agrees with recent human modeling data (21) and lateral gastrocnemius (LG) data recorded in vivo in running turkeys (17). Also, the estimated maximum force the human MG can produce when fully activated and isometric has been calculated to be 1,500 N (31), which is well above any of the forces calculated in the present study. Therefore, it seems that during walking and steady-state running, MG is working well within its performance limits and presumably at relatively low activations. Perhaps this is not surprising given that in these activities, humans are moving well below maximal running velocities and generating ground reaction forces far less than those produced during sprinting or other maximal explosive tasks.

This presumed submaximal activation of MG suggests that the lowering of fascicle velocity between walking at 2.0 m·s^{-1} and running is not a necessity but a preference. Higher muscle forces could be achieved at the fascicle velocities observed for fast walking if a greater volume of muscle were recruited by increases in activation. However, increasing the volume of active muscle would increase metabolic energy consumption (32). Running at the preferred transition speed actually requires greater MG activation than walking at the same speed (12, 23). However, the greater activation required to run may result in proportionally greater increases in force (and work) owing to the reduced fascicle velocity in running. Also, slower muscle fiber-shortening velocities require less metabolic energy (33). Therefore, the fascicle velocity-related benefits of running might outweigh the negative consequences of the greater activation required. The argument presented here comes with a caveat, that we do not know where on its force–velocity relationship soleus (SOL) is operating. Soleus is considerably larger than MG, likely accounting for a greater proportion of plantar–flexor force production, and is also important for producing forward propulsion (3). If soleus were operating nearer its force-producing limit at the PTS, then this might suggest that switching to running to lower soleus fascicle velocity makes the decision a necessity. Neptune and Sasaki (12) showed in their modeling data that soleus and gastrocnemius forces were reduced when walking at speeds above the PTS despite increasing activation. In the same study, these authors presented experimental data that anterior–posterior ground reaction forces decreased for walking above the PTS despite increasing plantar–flexor EMG signals. Therefore, although our data suggest MG never approaches its theoretical limits, it might be that soleus does and therefore the transition becomes necessary.

To draw more certain conclusions about the influence of plantar–flexor muscle mechanics on the gait transition, future studies should aim to include measurements of both soleus and lateral gastrocnemius function. This could aid more confident determination of force distribution between the plantar flexors. The current approach based on physiological cross-sectional area (PCSA) has often been used (34–36), but relies upon the assumption that relative muscle activation level is similar in each of the plantar flexors (e.g., MG, LG, and soleus are all activated at 70% of their maximum). In the present study, it is assumed that this holds across speeds (e.g., if MG relative activation increases by 10% from one speed to the next, so does soleus and LG relative activation). Experimental EMG and modeled activation data support this assumption, indicating that the relative activations of gastrocnemius and soleus increase in the same manner across fast (60–120% of PTS) walking speeds and slow running speeds (12). Other studies that report relative activations of both soleus and gastrocnemius across speed (24) indicate that the relative activations of the two muscles are slightly offset (e.g., during walking at 2.0 m·s^{-1}, gastrocnemius was active to 40% of maximum and soleus was at 45–50%). However, this offset was similar across all walking speeds (i.e., always 5–10% greater in soleus), and shifted slightly in favor of gastrocnemius with the transition to running (i.e., for running at 2.0 m·s^{-1},

soleus was 55% active and gastrocnemius was 60%). This shift would have meant that in the present study the increase in force due to MG observed for the transition to running at 2.0 $m \cdot s^{-1}$ would have been underestimated. Thus, even a model that accounts for such changes in relative muscle activation should find that force production by MG increases with the transition to running [as was the case for Sasaki and Neptune (12)].

Combining imaging (i.e., fascicle length, velocity) and EMG data from all of the triceps surae with a force-sharing model that accounts for differences in activation, force–length, and force–velocity between the different muscles is a promising approach that could lead to improved estimates of muscle-level mechanics, in vivo. However, this approach would require careful determination of force–length and force–velocity relationships as well as "true" maximum activations for each muscle involved. We note that although the force estimates in this study should be considered cautiously in light of the aforementioned limitations (i.e., force sharing), the observed changes in MG force with speed and gait were still what would be expected based on the force–length and force–velocity behaviors we acquired directly from ultrasound images.

Our length and velocity measurements based on ultrasound images are not entirely immune from potential error. For example, misalignment of the image plane with the fascicular plane (37) could cause projection errors in our fascicle length measurements. To combat this possibility, in the present study care was taken to align the ultrasound probe such that fascicles could be visualized from the deep to the superficial aponeurosis. This approach has been shown to be accurate in cadavers (38) and repeatable in vivo (39). The sampling rate of 50 Hz for ultrasound images might be considered low for some of the analyzed speeds. Too low of a sampling rate could have attenuated fascicle velocity peaks. However, if this were the case, careful analysis of Figs. 1 and 2 reveals that larger peaks in velocity would only strengthen the current result. Greater peaks could have increased V_{MGmax} for fast walking speeds and reduced V_{MGmax} for running gait [at the time of peak force, velocity was decreasing in magnitude for running but increasing for walking (Fig. 1)].

This study shows in vivo in humans that the shortening velocity of MG fascicles increases with walking speed and that this impairs the muscle's ability to produce force. Switching to a running gait reduced fascicle velocity at the time of peak force and correspondingly increased peak and average muscle force production and recycling of energy in the series elastic structures. Thus, we confirm suggestions from modeling studies that muscle fascicle velocity may be a primary trigger in the walk-to-run transition. Future studies should attempt to extrapolate these findings to other muscles that power locomotion and directly link muscle contractile behavior to metabolic costs.

Materials and Methods

Experimental Protocol. Ten healthy individuals [six male (mean ± SD, age = 25 ± 5; height = 1.76 ± 0.1 m; mass = 77 ± 12 kg) and four female (age = 24.5 ± 5.6; height = 1.6 ± 0.2 m; mass = 67 ± 5 kg)] gave written informed consent to participate in this study. Ethical approval for all experimental procedures was granted by an institutional review board at the University of North Carolina, Chapel Hill, and all procedures were in line with the Declaration of Helsinki.

All of the experimental trials took place on a split-belt treadmill that was instrumented with two separate force platforms, one under each belt (Bertec). Participants completed four walking trials and four running trials. Walking trials were at 0.75, 1.25, 1.75, and 2.0 $m \cdot s^{-1}$. Running trials were at 2.0, 2.25, 2.75, and 3.25 $m \cdot s^{-1}$, providing a range of speeds with an overlap at 2.0 $m \cdot s^{-1}$ for comparison of walking and running at the same speed. Each trial lasted 7 min and participants were allowed to self-select their stride frequency and length.

Kinematics and Kinetics. An eight-camera motion analysis system (Vicon) was used to capture the positions of 22 reflective markers attached to the pelvis and right leg (modified Cleveland Clinic marker set). Raw marker positions were filtered using a second-order low-pass Butterworth filter with a cutoff of 10 Hz. A static standing trial was captured, and the positions of markers on segment endpoints were used to calibrate a four-segment (pelvis, thigh, shank, and foot) model for each subject using established inertia parameters (40). Clusters of three or four markers on rigid plates were attached to the pelvis, thigh, and shank segments to track segment motion during walking and running. For the foot, a cluster of three markers was attached directly to the participants' shoe. Joint angles for the hip, knee, and ankle were computed in three dimensions as the orientation of the distal segment with reference to the proximal segment and differentiated to calculate joint velocities.

Force data were recorded during walking and running, using the two force platforms embedded in the treadmill. For walking trials, participants were required to walk with each foot hitting its ipsilateral force platform, so as to separate out left- and right-limb contributions during double support. Raw analog force platform signals were filtered with a similar filter to marker data but with the cutoff set to 35 Hz. Inverse-dynamics analyses (1) were then used to compute net joint moments, which were multiplied by joint velocities to calculate joint powers at the hip, knee, and ankle. Kinematics and kinetics were calculated for the right leg only, and it was assumed that the left leg behaved symmetrically over a number of gait cycles. All kinematics and kinetics calculations were performed using Visual3D software (C-Motion).

Determination of Gastrocnemius Muscle Parameters. Medial gastrocnemius muscle fascicle length during walking and running was measured from B-mode ultrasound images (41). A linear ultrasound transducer (LV7.5/60/96Z; Telemed) operating at 8.0 MHz was placed over the midbelly of the MG and aligned so that MG fascicles could be visualized from deep to superficial aponeuroses (Fig. 4). Images were sampled at 50 Hz, and a pulse from the ultrasound system that was high (3–5 V) during recording and low (0 V) before and after was used to trigger collection of all other data synchronously. To obtain fascicle length from each image, a custom MATLAB (MathWorks) program was used to digitize the points of attachment of a fascicle on the superficial and deep aponeuroses, and the length was calculated as the distance between these two points. Pennation angle was defined as the angle between the digitized fascicle and the superficial aponeurosis (Fig. 4). The instantaneous length of the whole MG muscle–tendon unit was calculated from ankle and knee joint angles using the equations of Hawkins and Hull (42). To obtain a value for the length of the series elastic element (SEE), the length of the fascicle was multiplied by the cosine of pennation angle and subtracted from the MTU length (41) (Fig. 4). This ignores any angle between aponeurosis and external tendon of MG that could result in slight underestimation of the SEE length. Initial fascicle length (L_i) was taken as the length of the fascicle at heel strike, and fascicle length change (ΔL) was calculated relative to L_i.

Calculation of Gastrocnemius Muscle Kinetics. Muscle and tendon forces could not be measured directly, and so were estimated using inverse-dynamics analysis combined with the measured muscle parameters. Achilles tendon (AT) force was calculated as the net flexion–extension ankle moment divided by the moment arm of the AT per previously published methods (28, 29). The instantaneous moment arm of the AT was calculated as the first derivative of MG MTU length with respect to ankle angle (30, 43). The force attributable to MG was estimated by multiplying the AT force by the relative PCSA of MG within the plantar flexors (44), which was taken as 0.159 (equation 1 in ref. 45). This force was considered the force in the SEE of the MG (F_{SEE}). To estimate the force in the muscle fascicles of MG (F_{MG}), F_{SEE} was divided by the cosine of the MG pennation angle (equation 2 in ref. 35). This approach to calculating muscle force does not account for any contribution of antagonistic dorsiflexors to the net ankle moment, although this was assumed not to be significant during the stance phase, when tibialis anterior is minimally active (23), and the key variables in this study were determined. This PCSA-based approach also assumes similar relative activations among plantar flexors across conditions, which is considered in detail in the *Discussion*.

The velocities of the MG fascicles, MTU, and SEE were calculated as the first derivative of their lengths with respect to time. The power output of the fascicles, SEE, and MTU were then calculated as the product of their respective forces and velocities (Eqs. **3–5**). Positive work done by fascicles, SEE, and MG was estimated by integration of positive portions of each component's power curve. Periods of positive power during each trial were integrated by the trapezium method and summed, and then divided by the number of strides taken to calculate average positive work done per stride. These values were divided by stride time to convert to average positive powers for fascicle ($\bar{P}^{+}_{FAS}$), SEE ($\bar{P}^{+}_{SEE}$), and MTU ($\bar{P}^{+}_{MTU}$). These average powers were considered indicative of the fascicle and tendon interaction. The ideal (most efficient) scenario would be for $\bar{P}^{+}_{FAS}$ to be zero

(i.e., the fascicle is always isometric) and all of $\bar{P}^{+}_{MTU}$ to be supplied by $\bar{P}^{+}_{SEE}$ (i.e., from recoil of the SEE).

$$F_{SEE} = F_{AT} \cdot 0.159, \quad [1]$$

where F_{SEE} is the force due to the medial head of gastrocnemius, F_{AT} is the force due to all plantar flexors, and 0.159 is the relative physiological cross-section of the MG within the plantar flexors (compared with 0.57 for soleus, 0.065 for lateral gastrocnemius, and the remainder due to other plantar flexors).

$$F_{MG} = F_{SEE} \cdot (cos\phi)^{-1}, \quad [2]$$

where F_{MG} is the force in the muscle fascicles and ϕ is the pennation angle (in radians).

$$P_{MTU} = F_{SEE} \cdot V_{MTU}, \quad [3]$$

where P_{MTU} is MTU power and V_{MTU} is MG MTU velocity.

$$P_{SEE} = F_{SEE} \cdot V_{SEE}, \quad [4]$$

where P_{SEE} is SEE power and V_{SEE} is SEE velocity.

$$P_{FAS} = F_{MG} \cdot V_{FAS}, \quad [5]$$

where P_{FAS} is MG fascicle power and V_{FAS} is MG fascicle velocity.

The key outcome variables identified at each speed were the maximum force produced by the fascicles (F_{MGmax}); velocity of the fascicle at the time of F_{MGmax} (V_{MGmax}); average shortening velocity of the MG fascicle ($\bar{V}_{MG}$); average force from the MG fascicles during ground contact ($\bar{F}_{MG}$); $\bar{P}^{+}_{MTU}$; $\bar{P}^{+}_{FAS}$; and $\bar{P}^{+}_{SEE}$.

Statistical Analysis. Each individual's fascicle length, velocity, and kinetics data were averaged over at least five strides. Data presented in graphs and tables are group means and SEMs unless otherwise stated. A repeated-measures ANOVA with a Bonferroni adjustment was used to test for differences across all speeds in L_i, ΔL, $\bar{P}^{+}_{FAS}$, $\bar{P}^{+}_{SEE}$, $\bar{P}^{+}_{MTU}$, $\bar{V}_{MG}$, $\bar{F}_{MG}$, F_{MGmax}, and V_{MGmax}. Where a significant F ratio was found for the effect of speed on a variable, Tukey's honestly significant difference (HSD) was used to assess between which speeds a significant difference existed; P values reported for pairwise comparisons represent the P value of a paired t test between the two speeds. For all statistical tests, an α level of 0.05 was set as a priori for statistical significance.

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Supporting Information

Farris and Sawicki 10.1073/pnas.1107972109

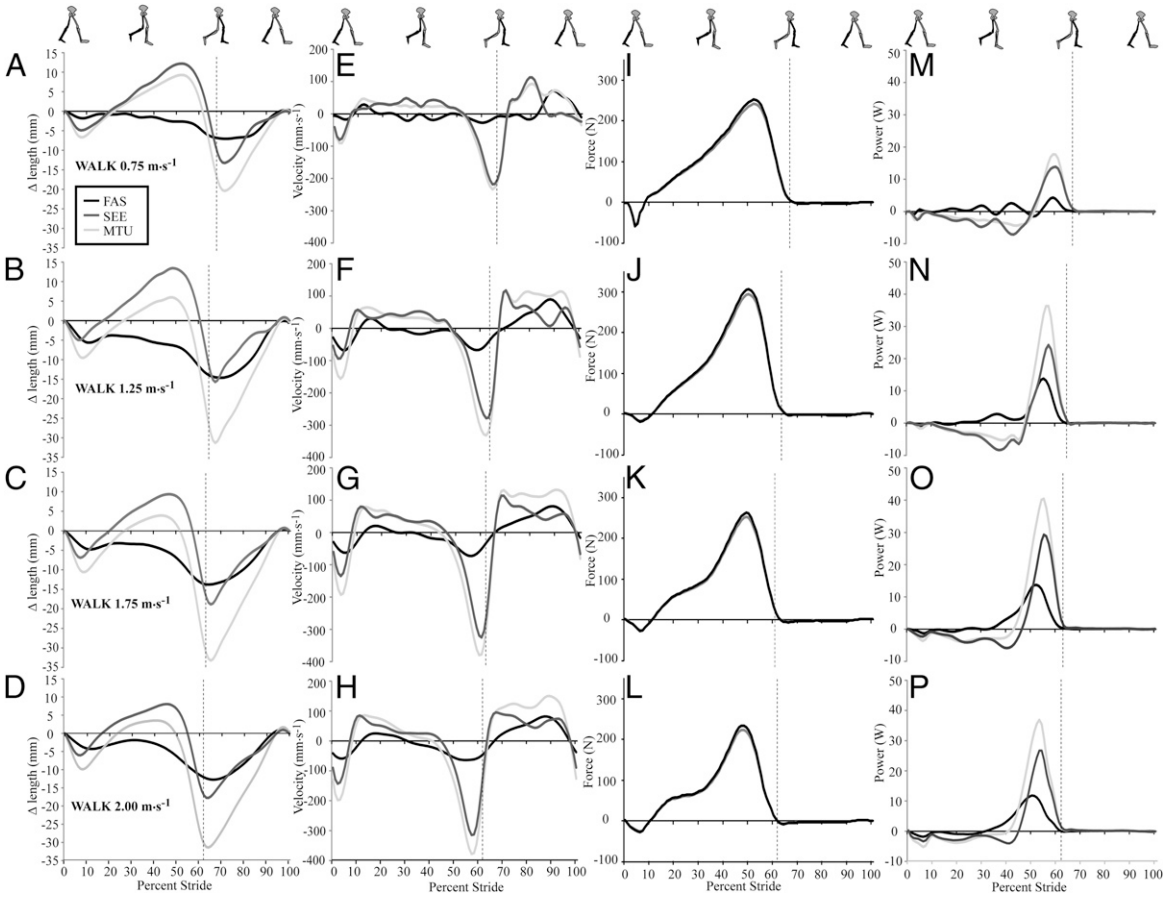

Fig. S1. Group mean medial gastrocnemius fascicle (black), series elastic element (dark gray), and muscle–tendon unit (light gray): length changes (*A–D*), velocities (*E–H*), forces (*I–L*) (MTU force is not plotted for clarity), and powers (*M–P*) during walking at different speeds (first row, 0.75 m·s^{-1}; second row, 1.25 m·s^{-1}; third row, 1.75 m·s^{-1}; fourth row, 2.0 m·s^{-1}). All data are normalized to 101 points over an entire stride. The dashed vertical lines indicate the average time of toe-off for the analyzed limb. Error bars are omitted for clarity.

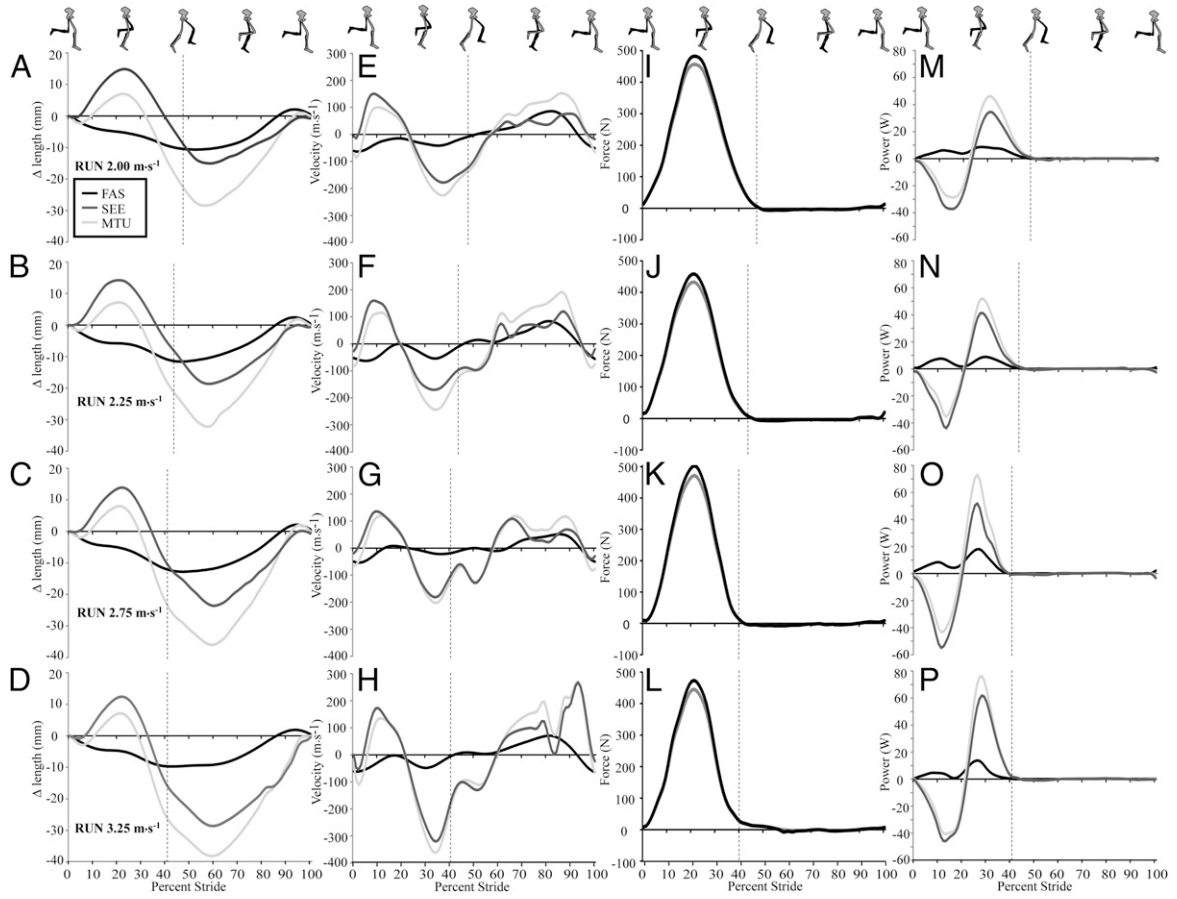

Fig. S2. Group mean medial gastrocnemius fascicle (black), series elastic element (dark gray), and muscle–tendon unit (light gray): length changes (*A–D*), velocities (*E–H*), forces (*I–L*) (MTU force is not plotted for clarity), and powers (*M–P*) during running at different speeds (first row, 2.0 $m{\cdot}s^{-1}$; second row, 2.25 $m{\cdot}s^{-1}$; third row, 2.75 $m{\cdot}s^{-1}$; fourth row, 3.25 $m{\cdot}s^{-1}$). All data are normalized to 101 points over an entire stride. The dashed vertical lines indicate the average time of toe-off for the analyzed limb. Error bars are omitted for clarity.

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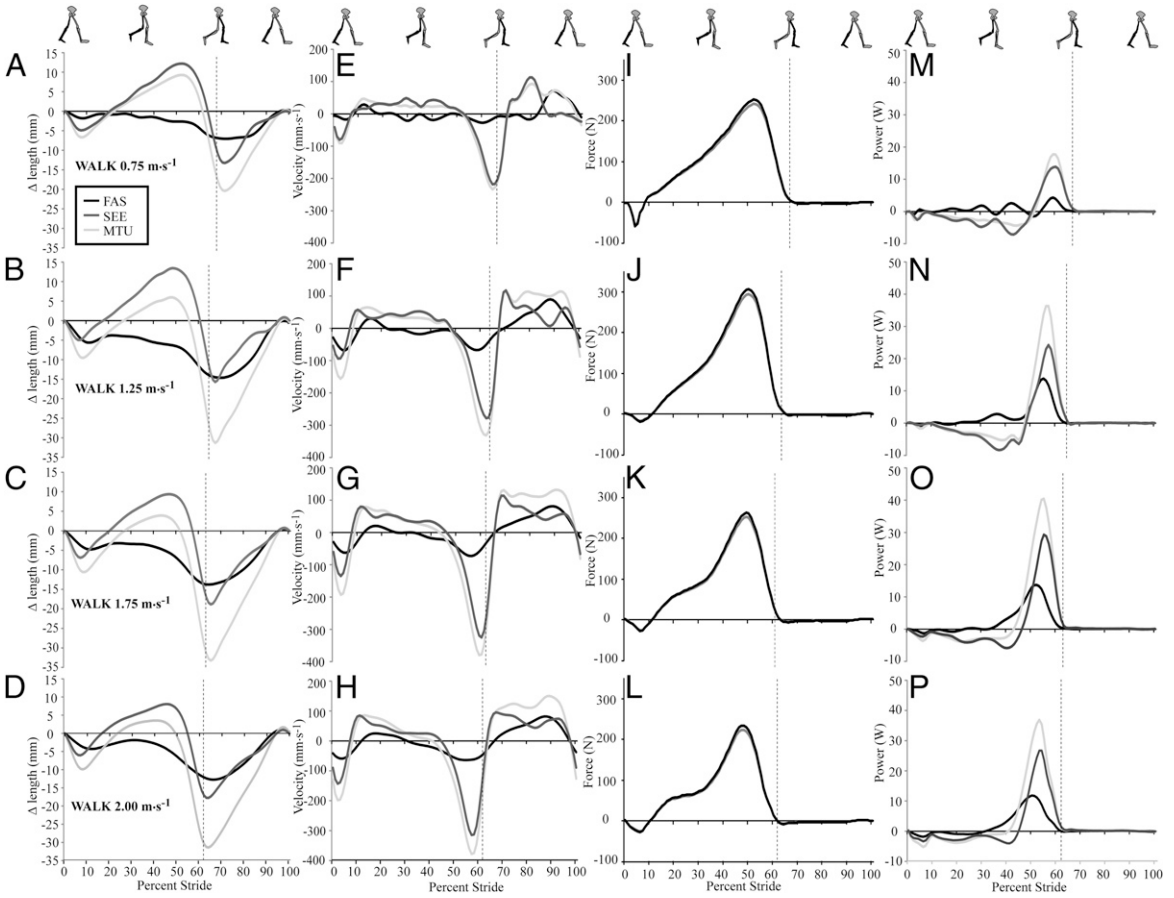

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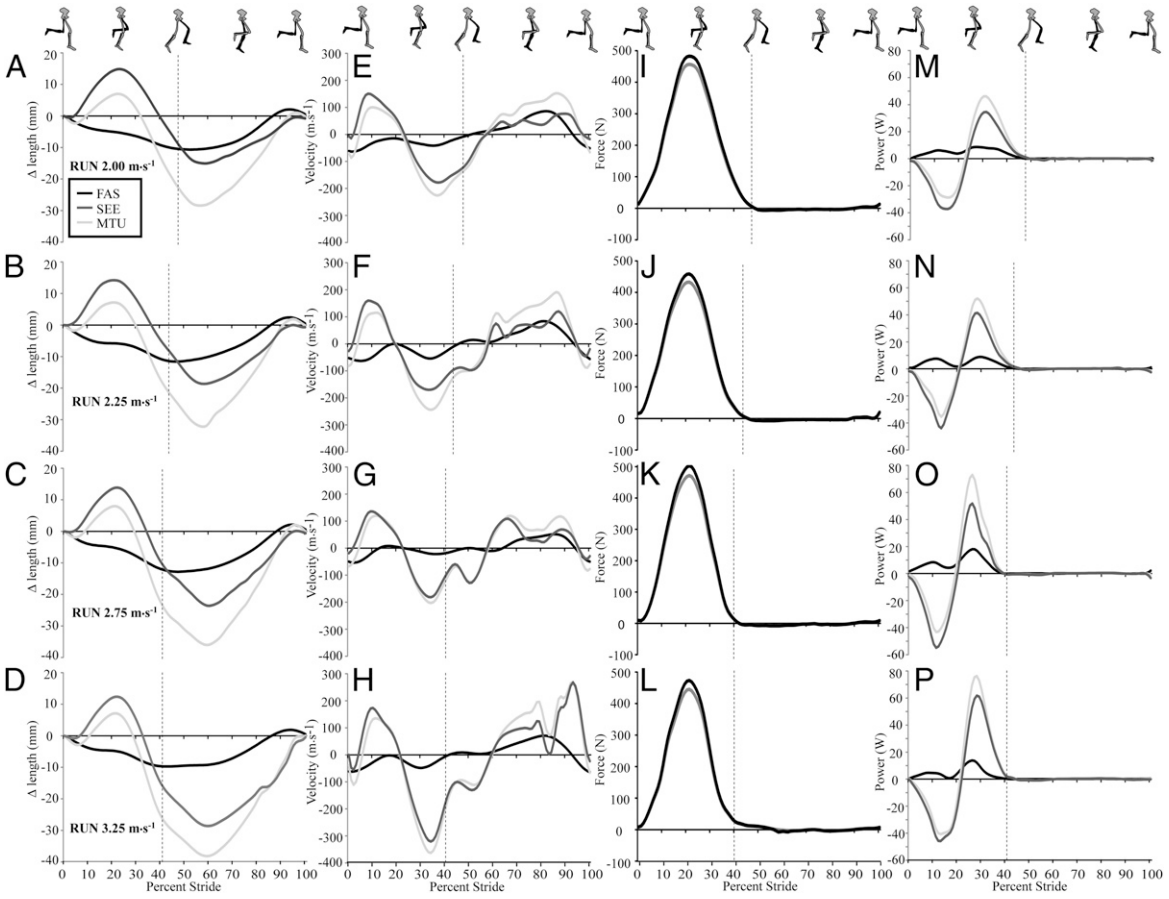

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