Energy cost and muscular activity required for leg swing during walking

Jinger S. Gottschall and Rodger Kram

Department of Integrative Physiology, University of Colorado, Boulder, Colorado

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Gottschall, Jinger S., and Rodger Kram. Energy cost and muscular activity required for leg swing during walking. J Appl Physiol 99: 23–30, 2005; doi:10.1152/japplphysiol.01190.2004.—To investigate the metabolic cost and muscular actions required for the initiation and propagation of leg swing, we applied a novel combination of external forces to subjects walking on a treadmill. We applied a forward pulling force at each foot to assist leg swing, a constant forward pulling force at the waist to provide center of mass propulsion, and a combination of these foot and waist forces to evaluate leg swing. When the metabolic cost and muscle actions were at a minimum, the condition was considered optimal. We reasoned that the difference in energy consumption between the optimal combined waist and foot force trial and the optimal waist force-only trial would reflect the metabolic cost of initiating and propagating leg swing during normal walking. We also reasoned that a lower muscle activity with these assisting forces would indicate which muscles are normally responsible for initiating and propagating leg swing. With a propulsive force at the waist of 10% body weight (BW), the net metabolic cost of walking decreased to 58% of normal walking. With the optimal combination, a propulsive force at the waist of 10% BW plus a pulling force at the feet of 3% BW the net metabolic cost of walking further decreased to 48% of normal walking. With the same combination, the muscle activity of the iliopsoas and rectus femoris muscles during the swing phase was 27 and 60% lower, respectively, but the activity of the medial gastrocnemius and soleus before swing did not change. Thus our data indicate that ~10% of the net metabolic cost of walking is required to initiate and propagate leg swing. Additionally, the hip flexor muscles contribute to the initiation and propagation leg swing.

Address for reprint requests and other correspondence: J. S. Gottschall, Emory Univ. School of Medicine, Dept. of Physiology, Whitehead Research Bldg., Atlanta, GA 30322 (E-mail: jgottsc@emory.edu).

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and waist force trial would indicate which muscles are responsible for leg swing during normal walking.

METHODS

Overview. We completed pilot and experimental protocols. For the pilot protocol, each subject walked with only a forward pulling force at each foot. For the experimental protocol, each subject walked with forward pulling forces at each foot, a constant forward force at the waist, and a combination of these foot and waist forces.

Ground reaction forces. Subjects performed each protocol on a newly developed dual-belt force treadmill. The right treadmill is mounted on a force platform and is essentially a hybrid of the designs of Belli et al. (4) and Kram et al. (16). We collected right leg ground reaction forces (GRF), both horizontal and vertical components, for 10 s at 1,000 Hz. The GRF data were filtered by using a fourth-order Butterworth low-pass filter with a cutoff frequency of 20 Hz. We used the GRF records to detect kinematic events and to quantify the effects of the external force devices. For each trial, we calculated the vertical and horizontal force peaks and impulses per step.

Swing assist apparatus. We applied independent anterior pulling forces to the distal dorsum of each shoe during the swing phase (Fig. 1). Nylon cords, clipped to each shoe, led to rubber tubing that was stretched over low-friction pulleys. The magnitude of this external swing assist (ESA) force was adjusted with the subject standing in place at the middle of the treadmill with the cords taut and the cable clamps flush with a stopper plate. We monitored the desired nominal ESA with a force transducer (Omega Engineering, Stamford, CT) in series with the rubber tubing. We adjusted the tension of the rubber tubing to the nominal force by stretching the tubing with a hand winch. Subjects walked at the same location on the treadmill so that the magnitudes of the ESA remained consistent from stride to stride. By design, the ESA force was not constant throughout the stride. During the stance phase, as the foot moved backward, the rubber tubing stretched so that the ESA force increased and was greatest at the end of stance. At the onset of the swing phase, the device pulled the leg anteriorly, allowing the rubber to shorten and the ESA force decreased (Fig. 2). After midswing, a cable clamp attached to each cord reached a stopper plate, and the ESA applied to the foot decreased to zero. The cable clamp minimized the need to recruit muscles eccentrically at the end of swing. The external swing apparatus was a modification of the device utilized by Modica and Kram (21).

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Horizontal pulling apparatus. We applied a propulsive force using a waist belt worn near the center of mass (Fig. 1). Our previous study determined that a magnitude of 10% BW optimally reduces the metabolic cost of walking (10). The waist belt was connected to lengths of rubber tubing that were stretched over several low-friction pulleys. The rubber tubing was stretched to approximately two to three times its resting length so that small changes in length did not substantially change the applied force. Thus an almost constant applied horizontal force (AHF) was employed. We monitored the AHF with a force transducer in series with the rubber tubing. The magnitude of the AHF was adjusted by altering the number of rubber tubing elements in parallel and by stretching the tubing with a hand winch. Subjects viewed a digital display of the AHF and maintained the desired level of force by walking at the appropriate position on the treadmill. AHF was maintained within ±5% of the desired value of 10% BW (9.5–10.5%). The horizontal pulling apparatus was similar to the device utilized by Gottschall and Kram (10).

Pilot protocol. Four subjects performed a standing trial and six walking trials at 1.25 m/s. All trials lasted 7 min with a 3-min rest between each trial. The subjects walked with no external force for the initial and final walking trials. We then applied anterior pulling forces at the feet equal to 1, 3, 5, or 7% BW. During these pilot trials, we measured GRFs (Fig. 3) and metabolic energy consumption but not EMG. Analysis of the horizontal GRF data indicated that the pulling forces at the feet inadvertently provided whole body propulsion, illustrated by a decrease in the horizontal propulsive impulse (Fig. 3B). To account for this propulsive force, during the experimental protocol, we applied an optimal propulsive force at the waist combined with pulling forces at the feet.

Experimental protocol. Five men and five women volunteered [age 27.79 yr (SD 3.30), mass 68.93 kg (SD 7.45), height 1.78 m (SD 0.03)]. All of these healthy subjects gave written, informed consent according to the University of Colorado Human Research Committee approved protocol.

Each subject performed a standing trial followed by seven walking trials at 1.25 m/s. We familiarized the subjects to dual-belt force treadmill walking within 7 days of data collection. Both the standing and walking trials lasted 7 min with 3 min between each trial. We determined stride frequency (strides/min) during the first walking trial, and subjects matched a metronome set to their frequency for the remaining trials. The subjects walked normally with no applied external forces at both the beginning and the end of the experiment. We then applied various combinations of external force as a percentage of BW; at each foot, at the waist, or a combination of both these foot and waist forces in a random order. More specifically we applied 1) 3% BW pulling force at each foot 2) 10% BW propulsive force at the waist, and 3) 10% BW propulsive force at the waist simultaneous with 1, 3, or 5% BW pulling force at each foot. For clarity and brevity, we subsequently omit the reference to BW when referring to the external force conditions.
Energy measurements. We measured the rates of (V\(\dot{O}_2\)) and carbon dioxide production (V\(\dot{CO}_2\)) using an open-circuit respirometry system (Physio-Dyne Instrument, Quogue, NY). Before beginning the experimental trials, we measured standing metabolic rate. For all trials, we allowed 3 min for the subjects to reach steady state and then calculated the average V\(\dot{O}_2\) (ml O\(_2\)/s) and V\(\dot{CO}_2\) (ml CO\(_2\)/s) for the subsequent 3 min. We calculated metabolic rate (W/kg) using a standard equation (5). Lastly, we subtracted the standing value from the experimental values to derive net metabolic rate.

EMG measurements. We measured EMG signals by using a tele-metered amplifier system (Noraxon, Scottsdale, AZ). Before electrode placement, we prepared the shaved skin with fine sandpaper and alcohol. We placed bipolar silver-silver chloride surface electrodes (1-cm-diameter disks) over eight muscles [iliopsoas (Ilio), gluteus maximus (GM), biceps femoris (BF), RF, vastus lateralis (VL), anterior tibialis (AT), MG, and soleus (Sol)] of the right leg according to the recommendations by Cram and Kasman (6). For the Ilio, we placed the electrodes lateral to the femoral pulse yet medial to the RF and inferior to the inguinal ligament. For the GM, we placed the electrodes on the superior, central third of the muscle. For the BF, RF, AT, and MG, we placed the electrodes over the approximate center of the muscle belly. For the VL and Sol, we placed the electrodes over the distal third of the muscle lateral to the RF and GM, respectively. The interelectrode distance was 2 cm. The EMG amplifier had a gain of 1,700. We verified that the cross talk between muscles was negligible with a series of contractions suggested by Winter et al. (32) and Cram and Kasman (6). Each subject’s electrodes remained in place for all trials without being removed or replaced.

The muscle verification tests were extremely important for the Ilio. Cram and Kasman (6) define this hip flexor as quasi-specific for surface measurements; in short, the Ilio EMG signal can be corrupted by cross talk from other hip flexors. Because of this possibility, we performed both a cross talk test and a walking task to ensure proper placement and minimal interference. To evaluate cross talk, we employed the method suggested by Cram and Kasman. Subjects knelt on their hands and knees, in a quadrupedal stance, on the floor. After lifting their knee slightly off the ground, they completed a hip flexion motion while flexing the knee and a hip extension motion while extending the knee. When the electrodes are properly placed on Ilio, we expect to record burst of activity only during the hip flexion portion of the exercise. If we observed these proper bursts, the subjects walked normally on a treadmill for 3 min. We examined the Ilio EMG patterns and compared them with surface electrode measurements collected by Sutherland et al. (27). If either of these tests yielded questionable electrode placement, we performed the electrode placement procedure again.

We processed the EMG data in two stages. First, we determined when in a stride the muscles were active and, second, we quantified the magnitude of activation. After data collection at a rate of 1,000 Hz, we band-pass filtered the data to retain frequencies between 10 and 500 Hz. For the temporal analysis, we digitally filtered the rectified signals with a low-pass filter with a cutoff frequency of 7 Hz to generate a linear envelope (31). We created a MatLab program to determine the difference between the baseline and muscle activity. We defined a muscle as active if the EMG linear envelope exceeded a threshold of two standard deviations above the baseline mean for at least 100 ms. Using this stipulation, acceptable Ilio data were obtained for eight subjects. For the amplitude analysis, we full-wave rectified the band-pass filtered signals, calculated the mean EMG amplitude (mEMG) for specific portions of the step, and averaged 10 consecutive steps for each condition. For each subject, we normalized the mEMG for each experimental condition on the basis of the mEMG for the normal walking condition.
Statistical analysis. Energy and EMG data from this study were analyzed across all conditions by using a repeated-measures, six-level design (ANOVA). We performed Newman-Keuls post hoc tests to analyze the differences between each experimental condition. Significance was defined as $P < 0.05$.

RESULTS

Ground reaction force. The GRF and impulse results established the biomechanical effects of our external force devices. The external force devices affected both braking and propulsive horizontal GRF peaks and impulses compared with normal walking (Fig. 3B). Compared with normal walking, with 3% ESA only, the braking force peak increased by an insignificant 8% ($P = 0.29$), whereas the propulsive peak decreased by 17% ($P = 0.0005$). Thus the ESA alone inadvertently aided forward propulsion, and we accounted for this aiding force by incorporating the AHF device. The AHF alone caused the horizontal braking forces to increase by 92% and the propulsive forces to decrease to just 28% of normal. When we combined the 10% AHF and the 3% ESA, the braking force increased by 95% and the propulsive force decreased to 31% of the values for normal walking. Thus the similarity of the changes of these horizontal GRF variables allowed us to fairly compare the differences in metabolic cost and muscle activity between AHF only with AHF and ESA combined conditions.

Compared with normal walking, during the ESA-only trials, the vertical force peaks were not different ($P = 0.76$). In contrast, compared with normal walking, with 10% AHF, the first vertical force peak increased by 27% ($P < 0.0001$) and the second vertical force peak decreased by 15% ($P < 0.001$; Fig. 3A). However, the vertical force peaks during the combined AHF and ESA trials were not different from the peaks during the AHF-only trials ($P = 0.49$). Furthermore, neither of the external force devices affected the vertical impulse ($P = 0.56$). Thus, on the basis of the vertical GRFs, it was reasonable to compare these trials.

Energy. Our most important findings were that, compared with normal walking, net metabolic rate decreased during the AHF-only trials and further decreased during the AHF and ESA combined trials ($P < 0.001$). With 10% AHF only, the net metabolic rate of walking decreased to 58% of normal walking. With 10% AHF and 3% ESA, the net metabolic rate of walking further decreased to 48% of normal walking, the minimum for all the trials (Fig. 4). With 10% AHF and 5% ESA, net metabolic rate was greater than with 10% AHF and 3% ESA, resulting in a J-shaped curve, suggesting that 10% AHF and 3% ESA was the optimal combination.

During the pilot trials, with 5% ESA only, the net metabolic rate of walking decreased to 85% of normal walking, the minimum value for the ESA-only trials. With 1 and 3% ESA, net metabolic rate decreased to 96 and 91%, with 7% ESA, the net metabolic rate of walking decreased to 89% of the normal walking metabolic rate, which was greater than with 5% ESA.

Temporal muscle activity. We evaluated the EMG activity (Fig. 5) of each muscle on the basis of the temporal patterns of the normal walking trials (Fig. 6). The GRF data showed that the average stance phase comprised 0–62% of the stride. We utilized the temporal patterns and kinematic events to divide the stride into the following segments for the specified muscles: third quarter of stance (32–52%; MG and Sol), late stance (56–62%; Ilio, GM, BF, RF, and TA), early swing (62–67%; Ilio and RF), and late swing (90–100%; GM, RF and TA).

Mean muscle activity. During all the combined AHF and ESA trials, the Ilio mEMG, during both the swing initiation and propagation phases, was significantly lower than the AHF-only trials (Fig. 7). With 10% AHF only, the Ilio mEMG values during early swing and late swing were not different from normal walking ($P = 0.75$ and $P = 0.90$, respectively). During late stance, when 1, 3, and 5% ESA was combined with the 10% AHF, the Ilio mEMG values were 71, 75, and 80% lower, respectively, than during the AHF-only trials (all values, $P < 0.05$). During early swing, when 1, 3, and 5% ESA was combined with the 10% AHF, the Ilio mEMG was 75, 73, and 63% lower, respectively, compared with AHF-only trials (all values, $P < 0.05$). The Ilio mEMG during all the ESA trials was significantly different from both normal walking and the AHF-only trials, but the Ilio mEMG values during the ESA trials were not significantly different from each other.

Similar to the Ilio, during all the combined ESA and AHF trials, the RF mEMG during both the swing initiation and propagation phases was significantly lower compared with the AHF-only trial (Fig. 7). With 10% AHF only, the RF mEMG during early swing ($P = 0.88$) and late swing ($P = 0.92$) was not different from normal walking. During late stance, when 1, 3, and 5% ESA were combined with the 10% AHF, the RF mEMG was 62, 63, and 64% lower, respectively, compared with AHF-only trials (all values, $P < 0.05$). During early swing, when 1, 3, and 5% ESA was combined with the 10% AHF, the RF mEMG was 49, 40, and 37% lower, respectively, compared with AHF-only trials (all values, $P < 0.01$). The RF mEMG values during all the ESA trials were significantly different from both normal walking and the AHF-only trials, but the RF mEMG values during the ESA trials were not significantly different from each other.
As we have reported previously (10), during the AHF-only trials, the MG mEMG was lower during the propulsive phase of stance; however, the Sol mEMG did not change compared with the normal walking trials (Fig. 8). With 10% AHF only, the MG mEMG was 55% lower than the normal walking magnitude ($P < 0.0001$), but the Sol mEMG did not differ compared with normal walking ($P = 0.16$). Moreover, when ESA was combined with AHF, the mEMG of neither the MG nor the Sol differed from the AHF-only trial.

We also used EMG to evaluate whether the external force devices caused unintended increases in muscle activity. We compared the activity during normal walking with the experi-

Fig. 5. Raw electromyography during normal walking vs. percentage of stride.

**Fig. 6.** Temporal electromyography during normal walking vs. percentage of stride. Solid bars, activity. Values are means ± SD.

Fig. 7. Mean electromyograph vs. applied external force relative to normal walking for the iliopsoas and rectus femoris during late stance (A) and early swing (B). Values are means ± SD. *$P < 0.05$, **$P < 0.01$ from normal walking.
apparatus may not entirely remove the need to generate swing forces. In our pilot trials, with 5% ESA only, metabolic cost decreased to a minimum of 85% of that during normal walking, suggesting that the metabolic cost of leg swing is $\sim 15\%$. However, the decrease in the horizontal GRFs and impulses

mental conditions for the BF, VL, GM, and TA. The BF and VL mEMG did not change during late stance but did increase during late swing (Fig. 9; all values, $P < 0.05$). The GM mEMG did not change significantly during swing initiation or swing termination. The TA mEMG was significantly higher during swing initiation, propagation, and termination (all values, $P < 0.05$).

DISCUSSION

Energy. The metabolic cost of walking decreased when AHF and ESA were provided. With 10% AHF only, metabolic cost decreased to 58% of that during normal walking. With 10% AHF and 3% ESA, metabolic cost further decreased to 48% of that during normal walking. Thus we conservatively infer that generating forces to initiate and propagate leg swing constitutes $\sim 10\%$ of the net metabolic rate of normal walking.

Our conclusion that swing is 10% of the total cost of swinging the legs during walking is only an estimate. The GRF data indicated that our external force devices change the magnitude and phase of the horizontal impulses. Specifically, the horizontal braking force increased, whereas the propulsive force decreased. These results indicate that the percentage of concentric and eccentric muscle contractions also changed during a stride. However, because eccentric contractions require less metabolic energy, we assume that our estimation is reasonable. In addition, the greater metabolic cost with the combined 10% AHF and the 5% ESA, compared with the combined 10% AHF and the 3% ESA may be due to the increased muscular effort for arresting the protraction at the end of swing (1).

The energetic cost of initiating and propagating leg swing in normal walking may be slightly greater than 10% because our

Fig. 8. Mean electromyograph vs. applied external force relative to normal walking for the medial gastrocnemius and the soleus during the third quarter of stance. Values are means $\pm$ SD. $*P < 0.01$. †$P < 0.0001$ from normal walking.

Fig. 9. Mean electromyograph vs. applied external force relative to normal walking for the gluteus maximus and biceps femoris during late stance (A) and late swing (B). Values are means $\pm$ SD. $*P < 0.01$. †$P < 0.05$ from normal walking.
demonstrated that the ESA device provided a modest amount of forward propulsive force. Therefore, 15% is an overestimate of the metabolic cost of swing. We accounted for these forward propulsive forces by combining the optimal AHF and ESA conditions. We conclude that the metabolic cost of initiating and propagating leg swing comprises between 10 and 15% of the net cost of human walking at 1.25 m/s.

This estimate can be put in context with the broad range of estimates from previous studies. Griffin et al. (12) applied loads to the waist and surmised that swing cost was negligible. However, they acknowledged that the cost of leg swing could be as much as 20% of the net metabolic rate. Marsh et al. (17) measured the blood flow to the individual muscles during walking in guinea fowl and estimated that the swing muscles consume 26% of the total limb energy. Unfortunately, their results are difficult to translate to walking humans because of the differences in leg anatomy. Overall, our approximation seems to be a reasonable value, and our methodology provides the most direct measure of the cost of leg swing in walking humans.

These results allow us to comprehensively partition the net metabolic cost of walking into discrete parts. Grabowski et al. (11) recently employed an upward vertical force to simulate reduced gravity and added mass at the waist to evaluate the metabolic costs of supporting weight and performing propulsive work on the center of mass. They concluded that the cost of supporting BW is ~28% of the net metabolic cost of walking and that the cost of propulsion is ~48%. In a previous study, we (10) applied an optimal external horizontal force that provided propulsion, and metabolic cost decreased by 47%, which is not significantly different from the present study estimate of 42%. Similarly, from the data of Donelan et al. (7), we calculated that the step-to-step transition work, performed to restore and redirect the center of mass velocity, comprises 47% of the net metabolic cost of walking. The similarity of these values for the cost of propulsion, obtained in three different ways, increases our confidence in this estimate. In a different study, Donelan et al. (8) attached rubber tubing that pulled bilaterally on the waist of subjects walking on a treadmill and deduced that lateral stabilization comprises ~6% of the net metabolic cost of normal walking. Thus we can partition the energetic cost of walking as follows: supporting BW = 28%, generating propulsion = 48%, swinging the legs = 10%, and stabilizing laterally = 6%. Overall, these applied external force experiments can account for 92% of the net metabolic cost of walking. The increased metabolic costs of ventilation and cardiac work could easily account for the remaining 8% (13).

Muscle activity. During all the ESA conditions, the Ilio and RF mEMG decreased during the last portion of the stance phase and early swing. Compared with the AHF-only trials, with 10% AHF and 3% ESA, Ilio and RF mEMG decreased by 27 and 41%, respectively, during swing initiation. Similarly, with this optimal external force combination, Ilio and RF mEMG decreased by 26 and 52%, respectively, during swing propagation. This evidence indicates that both the Ilio and RF initiate and propagate leg swing during walking.

During the ESA conditions, neither the MG mEMG nor the Sol mEMG was significantly affected by the ESA forces. During the AHF conditions, we found that the MG mEMG decreased during AHF-only trials but that the Sol mEMG did not change (10). Similar to previous studies (24, 29), our temporal results for normal walking showed that, on average, neither the MG nor the Sol was active past 52% of the stride. It is possible that the force generated by these ankle extensors at the end of the first half of stance phase can produce forward acceleration of the limb. But, our results suggest that neither the MG nor Sol directly initiates or propagates leg swing.

The BF and GM mEMG during late stance indicated that these muscles are not activated to stretch the elastic cords of the ESA apparatus. Compared with normal walking, BF and GM mEMG did not change during any external force condition. This result, in combination with the decreased metabolic cost during the external force conditions, suggests that the subjects did not expend additional energy to stretch the cords of the apparatus.

TA and VL mEMG increased during the ESA and AHF trials, possibly indicating an effort to control the effects of the external force devices. With the ESA device, during both early swing and late swing, the TA mEMG significantly increased. This increased activity did not occur during the AHF-only condition. During normal walking, the TA is active during swing to ensure that the foot clears the surface. The ESA attachment to the distal dorsum of the shoe apparently required increased TA activity to maintain ankle dorsiflexion during swing. In addition, with the AHF device, during late swing and the first half of stance, the VL mEMG significantly increased. This increased activity did not occur during the ESA-only condition. During normal walking, the VL is activated during late swing and the first half of stance to decelerate the body and resist knee flexion. The AHF device increased the need to generate braking forces, which is likely reflected by the increase in the VL mEMG.

Overall, our temporal EMG data are consistent with the majority of previous studies that focused on muscle activity during normal moderate-speed walking (9, 24), but our conclusions regarding the specific muscles responsible for leg swing do not always agree with their inferences. We concur with Neptune et al. (23) and Nene et al. (22) that the hip flexors are important for swing initiation. However, our interpretation conflicts with prior studies of the role of the ankle extensors and leg swing. Hof et al. (15), Meinders et al. (19), and Neptune et al. (23) stated that the ankle extensors contribute to leg swing initiation. Our EMG results cast doubt on that idea because the ankle extensors were not active during the last quarter of stance and their EMG did not decrease with ESA.

Limitations. Our external assistive force devices and EMG techniques were more than sufficient for addressing our hypotheses, yet our methodology imposed some limitations. The AHF device supplied a constant propulsive force. This assistive device could be improved by applying propulsive force only during the second half of stance. The ESA device effectively aided in leg swing initiation and propagation, but it pulled the foot horizontally and anteriorly. An ideal assistive device might apply this leg swing torque on all the leg muscles, alternatively aided in leg swing initiation and propagation, but it pulled the foot horizontally and anteriorly. An ideal assistive device might apply this leg swing torque on all the leg muscles, alternatively aiding in leg swing initiation and propagation, but it pulled the foot horizontally and anteriorly.
power to resolve all trends in our EMG data. Despite these limitations, our novel assistive devices have aided our understanding of leg swing in walking.

Future studies. Many questions remain about the metabolic cost and muscular activity required for leg swing in walking. For instance, is leg swing more expensive at faster walking speeds? Are there other combinations of AHF and ESA forces that would further decrease metabolic cost and muscular activity? Could a combination of simulated reduced gravity, horizontal propulsive, anterior leg swing, and bilateral stabilizing forces during walking reduce the net metabolic cost and muscular activity close to zero? Can these assistive devices be useful clinically to help rehabilitate patients with neuromuscular deficits? These questions can be further investigated with the help of forward dynamic computer simulations, guided by the present EMG and GRF data.

In summary, our metabolic cost data indicate that ~10% of the net metabolic cost of walking is required to initiate and propagate leg swing, while our EMG data indicate that the hip flexor muscles initiate and propagate leg swing.

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GRANTS
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