RESEARCH ARTICLE

RUNNING HEAD: *Energy needed for stabilizing walking decreases with speed*

Speeding up, not slowing down, decreases the metabolic energy needed to stabilize walking in the sagittal plane

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ABSTRACT

There is a metabolic cost associated with stabilizing walking, but it remains unclear to what extent stabilizing walking in the sagittal plane contributes to this cost. Furthermore, strategies for stabilizing walking in the sagittal plane vary with speed, but it is unclear whether this also leads to a speed-dependent metabolic cost of stabilizing walking. Here, we explored the metabolic cost of stabilizing walking in the sagittal plane across speeds and its relationship with control strategies. To this aim, we applied continuous treadmill belt speed perturbations (standard deviation of 0.13 ms^{-1}) to 22 healthy individuals walking at 0.8, 1.2, and 1.6 ms^{-1}. We evaluated changes in metabolic energy consumption and control strategies between perturbed and unperturbed walking and explored relationships between energy consumption and control strategies. Perturbations induced larger increases in metabolic rate and changes in control strategies at slower than faster walking speeds, suggesting that walking is more robust against perturbations at faster speeds. Perturbations increased the metabolic rate by 16.7% at the slowest versus 4.6% at the fastest walking speed. When perturbed, subjects took shorter, wider, and more variable steps, and variability in ankle muscle activation increased, but most changes were larger at slower speeds. Metabolic rate increased more due to perturbations in individuals who reduced step length more, i.e., relied more on anticipatory adjustments of the walking pattern. Our findings are especially relevant to explain the increased metabolic cost of individuals with mobility impairments, who often walk slower and have altered walking control.

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NEW & NOTEWORTHY

Stabilizing walking requires more active control in the frontal than in the sagittal plane. Nevertheless, we demonstrated that there is a considerable energetic cost associated with stabilizing walking in the sagittal plane, especially at slower speeds. This cost is higher in individuals who adjusted their average walking pattern more when walking was perturbed. Diseases that affect both walking speed and control might therefore have a disproportionately large effect on the metabolic cost of walking.

Keywords: Gait stability; Locomotion; Metabolic energy; Perturbations; Balance control strategies

INTRODUCTION

There is a metabolic cost associated with stabilizing walking. The metabolic cost of stabilizing walking has been studied in the frontal plane by either providing external stabilization or perturbing sensory input using visual perturbations (1–3). Increases in the metabolic cost of walking following perturbations were associated with both anticipatory adjustments in step length or step width (3), and reactive adjustments in foot placement resulting in increased step length and step width variability (2). Healthy adults also rely on reactive ankle torques to move the center of pressure under the foot, i.e., the ankle strategy, to stabilize walking (4). The ankle strategy is used more in the sagittal than in the frontal plane, and at slow speeds than at fast speeds (4). Yet, it is unclear whether individuals rely less on the ankle strategy at fast walking speeds because they rely more on stepping strategies, or because they require less active balance control to maintain stability because walking is more robust against sagittal plane perturbations at higher speeds (5, 6). In the first case, the metabolic cost for stabilizing walking at fast speeds would not necessarily be lower if anticipatory or reactive foot placement strategies are indeed important determinants of metabolic cost. Whereas in the second case, we would expect a lower cost for stabilizing walking at fast speeds. Here, we explored the metabolic cost of stabilizing walking in the sagittal plane across speeds and its relationship with control strategies.

The metabolic cost of stabilizing walking has been studied experimentally by either stabilizing walking or by perturbing walking. Typically, stabilizing walking does not only involve preventing a fall (balance control) but also maintaining the steady state (e.g., preventing net medio-lateral displacement or net changes in walking speed). When reducing the need for active control by springs that stabilize the pelvis in the mediolateral direction, energy consumption decreased by 5.7% compared to walking without stabilization (1). On the other hand, mediolateral virtual perturbations to the visual field increased walking energy consumption by 5.9%-12% compared to unperturbed walking (2, 3). Most studies focused on frontal plane stabilization because walking is thought to be less robust against perturbations in the frontal plane (1). In addition, the cost of walking on uneven terrain, which challenges stability across planes, has been studied. When walking on uneven surfaces, healthy adults increased energy consumption by 28% (7). Together, these studies suggest that there is a considerable energetic cost associated with stabilizing walking, but it remains unclear if, and to what extent, stabilizing walking in the sagittal plane contributes to this cost.

Humans use both anticipatory and reactive control strategies to stabilize walking. Healthy adults increase step width and decrease step length in the presence of continuous perturbations (2, 3, 7, 8) or when expecting discrete perturbations whose timing is unpredictable (9, 10). These adaptations have been interpreted as anticipatory strategies as they occur before perturbation onset (9). In addition, humans use feedback or reactive stepping and ankle strategies. Healthy adults adapt foot placement in response to disturbances such as uneven terrain (e.g., 7), mechanical (e.g., 11, 13), and visual perturbations (2, 3, 8). Healthy adults also modulate their ankle torque in response to perturbations by adapting ankle muscle activity to control the center of pressure. This ankle strategy is mainly used in the sagittal plane where the shape of the foot allows for larger displacements of the center of pressure (4, 13). In summary, healthy adults stabilize walking against disturbances by combining anticipatory adjustments, including increases in step width and reductions in step length, with reactive foot placement and ankle responses.

The energetic cost of stabilizing walking has been related to both anticipatory adjustments in mean step length and step width, and reactive foot placement strategies. When externally stabilizing the pelvis in the frontal plane, Donelan et al. (1) observed both a decrease in energetic cost and in step width variability and, therefore, suggested that the reduced need for using a reactive foot placement strategy in the frontal plane was the most likely explanation for the observed reductions in energy cost. O'Connor et al. (2) tested associations between increases in energy consumption due to visual field perturbation and changes in mean step width and step width variability. They found that energetic cost was most strongly associated with step width variability. However, Ahuja and Franz (3) observed simultaneous changes in energetic cost and mean step length and width. Healthy adults initially took shorter and wider steps and consumed more energy in response to mediolateral perturbations of the visual field, but alterations in step width and length, as well as energy consumption, decreased with prolonged exposure to perturbations. Hence, both alterations in anticipatory and reactive stepping strategies might have an important contribution to the energetic cost of stabilizing walking in the frontal plane. It is unclear whether this is also true for stabilizing walking in the sagittal plane, where the reliance on the ankle strategy is larger.

Healthy adults rely more on an ankle strategy at slower than at faster speeds to stabilize walking in the sagittal plane. Modulation of ankle moments in response to treadmill belt speed perturbations is larger during slow compared to fast walking (4). It is unclear whether the reliance on stepping strategies increases with increasing walking speed. In the frontal plane, mediolateral foot placement in unperturbed walking is better explained by center of mass kinematics at faster walking speeds, suggesting an increased reliance on foot placement strategies (6). It is possible that something similar happens in the sagittal plane. With increasing speed, step frequency increases, and thus, the time between consecutive foot contacts decreases, allowing for faster corrections through foot placement (4). At the same time, reductions in stance time decrease the available time for moving the center of pressure under the foot and, thus, the application of an ankle strategy. It has also been suggested that passive walking dynamics is more robust against perturbations in the sagittal plane at faster speeds, which would reduce the need for active control (5, 6). Whether stabilizing walking in the sagittal plane relies more on the intrinsic dynamics or requires more active foot placement adjustments at faster than

at slower speeds will have consequences for how the energetic cost of stabilizing walking in the sagittal plane varies with speed.

In this study, we assessed the metabolic cost of stabilizing walking in the sagittal plane across speeds and explored its relationship with control strategies. To this aim, we applied continuous treadmill belt speed perturbations to healthy individuals. Such perturbations mainly perturb walking in the sagittal plane and were previously found to elicit ankle responses, which were stronger at slower speeds (4). We hypothesized that these perturbations would increase the energy consumption of walking due to the need for active stabilization and that increases in energy consumption would be speed-dependent. In addition, we explored whether between-subject variation in energy consumption for stabilizing walking could be explained by variation in anticipatory and reactive control strategies. We assessed anticipatory alterations in the gait pattern based on changes in average step length and step width. We assessed reactive foot placement strategies based on changes in step length and step width variability, and reactive ankle strategies based on changes in variability of ankle muscle activity (measured through electromyography).

MATERIALS AND METHODS

Description subjects

We recruited subjects who understood English or Dutch. Individuals were excluded from participation when they suffered from conditions or took medication that influenced balance control or locomotor ability. We collected a representative dataset by including at least one female and one male participant in each age range (18-20, 21-25, 26-30, …, 56-60, and 61-65 years old). In total, 22 healthy adults between 19-65 years old (female: N=12, mean body mass: 71.9 kg, mean leg length: 0.93 m) enrolled in this study and provided written informed consent. The experimental protocol was approved by the Social and Societal Ethics Committee of the KU Leuven (G-2024-7626). The experimental protocol was pre-registered on Open Science Framework (14) after collecting data from the first three subjects.

Experimental procedure

Subjects walked on an instrumented treadmill (M-Gait, Motekforce link, Amsterdam, Netherlands) with the same type of shoes (i.e., sneakers with a low-damping sole) at three speeds, 0.8, 1.2, and 1.6 ms^{-1}, with and without treadmill belt speed perturbations. Walking conditions were randomized per block, i.e., first by speed and then by perturbation condition (Figure 1A). In each trial, subjects walked for about 6 minutes with at least 5 minutes of rest between walking trials. Subjects performed a 5-minute unperturbed walking trial for habituation at 1.2 ms^{-1} before the start of the experiment. During walking trials, subjects wore a safety harness, tethered to the ceiling, to prevent falls in the event of balance loss.

Data acquisition

We measured oxygen consumption and carbon dioxide production to estimate metabolic energy consumption using the K5 metabolic measurement unit (COSMED, Rome, Italy). Marker and ground reaction force (GRF) data were acquired through the Vicon Giganet system (Oxford Metrics, Oxford, England) in combination with a Vicon camera system to measure the position of the lateral malleoli using retroreflective markers at 200 Hz to estimate step length and width at heel strike. Gait events

were detected based on GRF, which were measured on the M-Gait instrumented treadmill at 1000 Hz. Ankle muscle activation patterns were measured using surface electromyography (EMG). EMG signals were sampled at 1000 Hz and measured using a bipolar electrode configuration (Cometa Wireless 32-channel EMG System, Cometa systems, Bareggio, Italy) or with the Delsys system (three last experiments - subjects 13, 21, and 22 – due to failure of the Cometa system; Trigno Wireless Biofeedback System with Avanti Sensors, Delsys, Natick, USA). EMG electrodes were placed over the tibialis anterior, gastrocnemius medialis, and soleus of the left leg after standard skin preparation (i.e. shaving and cleaning of the skin using alcohol).

Perturbation protocol

During perturbation trials, walking was perturbed by continuous random fore-aft treadmill belt speed variations. To construct the belt speed control signal for the perturbation trials, we adapted the procedure described in (15). The control signals for the velocity-controlled treadmill were generated in MATLAB and Simulink using software that was distributed with (15). First, we created pseudo-random belt accelerations using discrete Gaussian white noise with a standard deviation of 5 ms^{-2} and bounded between -15 and 15 ms^{-2}. Then, we integrated the accelerations to generate a velocity perturbation signal. The velocity perturbation signal was high-pass filtered at 1.7 Hz to eliminate drift, bounded between 0 and 3.6 ms^{-1}, and added to a constant speed of either 0.8, 1.2, or 1.6 ms^{-1}, depending on the condition. Because we were interested in the effect of speed on the energy required to stabilize walking, we kept the perturbation magnitude constant over walking speeds. In contrast to (15), the velocity perturbation signal for all walking speeds was generated using the same level of random noise, acceleration bounds, and high-pass filter cut-off frequency. We measured treadmill belt speed variation using the treadmill's belt speed sensors. The standard deviation of measured velocities was approximately 0.13 ms^{-1}. Perturbations did not affect the intended average belt speed and velocity bounds were never reached, i.e., 0.80 (min=0.37 – max=1.20) ms^{-1}, 1.20 (min=0.82 – max=1.63) ms^{-1}, and 1.60 (min=1.14 – max=2.03) ms^{-1} (Figure 1B). Measured treadmill belt velocity during perturbed walking at different speeds had similar frequency contents, indicating a similar perturbation magnitude across walking speeds.

Data pre-processing

GRF and marker data, and EMG signals were collected using Vicon's Nexus. We labeled marker data in Nexus and processed data in MATLAB (R2023, MathWorks, Natick, USA). EMG data was band-pass filtered between 20 and 200 Hz, normalized to the median muscle activation over all unperturbed walking trials, and rectified. Rectified EMG, GRF, and marker data were low-pass filtered using a third-order bidirectional Butterworth filter with a cut-off frequency of 20 Hz. Periods of missing marker data shorter than 0.1 seconds were linearly interpolated to account for marker occlusion.

In some EMG channels in subjects 3, 6, and 19 we registered large peaks, likely due to tension on the wires to the electrodes. We removed and linearly interpolated peaks with a prominence (amplitude from base to peak) larger than 20 standard deviations of the EMG over the entire trial by using MATLAB's findpeaks algorithm. However, in some instances, removing peaks did not suffice to remove movement artifacts from the EMG data. Therefore, we excluded EMG data from these instances from

192 further analysis. This was data in the perturbed trial from the gastrocnemius at speeds 1.2, and 1.6 ms^{-1}
193 for subject 3, and from the tibialis anterior at speeds 0.8, 1.2, and 1.6 ms^{-1} for subject 6.

194 Heel strike and toe-off were defined as the moment at which the vertical component of the GRF under
195 the respective foot increased above or decreased below 5% of body weight, respectively. Since subjects
196 did not always walk with one foot on each belt, we had to introduce additional criteria to only detect
197 correct steps, i.e. steps in which only one foot contacted the belt, allowing for correct evaluation of step
198 length and width outcomes. Gait events were included in further analysis if before heel strike and after
199 toe-off 1) the vertical GRF was below 1% of the maximal GRF for 12% of the median stance duration; this
200 was not the case when the contralateral foot was partially on the belt, 2) the slope of the GRF after heel
201 strike and before toe-off was sufficiently steep (i.e. average slope of the vertical GRF over 12% of
202 median stance duration was at least 75% of the maximum force over the entire walking trial divided by
203 the median stance phase duration); this was also not the case when the contralateral foot was partially
204 on the belt, and 3) the vertical GRF reached at least half of the maximum value over the entire walking
205 trial within the stance phase; this was not the case when the ipsilateral foot was only partially on the
206 belt. Visual inspection confirmed that these criteria led to correct determination of heel strike and toe-
207 off. These criteria resulted in a median removal rate of 2.2% (min=0.0%, max=25.7%) of events over all
208 walking trials and subjects (see Figure S2 for a visual representation of the event inclusion criteria, and
209 Figure S3 for exclusion rates per subject and condition).

210 **Measurement equipment and outcomes**

211 *Metabolic energy consumption*

212 We estimated metabolic rate from oxygen consumption and carbon dioxide production, measured by
213 the K5, using the standard formula of Brockway (17). The energy consumption of the walking trials was
214 represented by the average metabolic rate over the last two minutes of the trial. Subjects were asked
215 not to consume caffeine (12 hours), nicotine (12 hours), or food (3 hours) before the experiment to
216 prevent variability in measured signals unrelated to physical effort.

217 *Step width and length*

218 Step width and length were estimated by calculating the mediolateral and anteroposterior distance
219 between markers on the lateral malleoli at heel strike of the leading leg for the last two minutes of the
220 walking trial. We used the difference in mean step width and length between perturbed and
221 unperturbed walking at each speed to evaluate anticipatory changes to the gait pattern. We used the
222 difference in standard deviation of step width and length between perturbed and unperturbed walking
223 at each speed to evaluate changes in reactive foot placement.

224 *Variability in ankle muscle activation*

225 We expressed muscle activation (processed EMG signals) as a percentage of the single support phase of
226 the left leg, i.e. the period from right toe-off to right heel strike when the right leg was in swing. We only
227 included single support phases in the last two minutes of the walking trial.For each subject, condition,
228 and muscle, we first computed the standard deviation in muscle activation over steps at each
229 percentage of the single support phase to assess between-step variability. Then, we combined the
230 standard deviations by consecutively computing the root mean square values over the single support

phase and across all muscles to obtain one representative value for variability of ankle muscle activity. We used the difference in variability between perturbed and unperturbed walking at each speed to evaluate changes in reactive ankle strategy.

We normalized metabolic rate to $g^{3/2}l^{1/2}m$, and step length and width to l to obtain all outcomes in dimensionless units (i.e. n.d.), where g is the gravity constant, l is leg length measured between the right lateral malleolus and posterior superior iliac spine, and m is the mass of the subject. Normalization factors for a subject with an average leg length and body weight were 2126 W for metabolic rate, and 0.93 m for step length and width.

Statistical analysis

Statistical analysis was performed using R statistical software (R Foundation for Statistical Computing, Vienna, Austria). For testing our hypotheses, we used the difference in outcome variables between perturbed and unperturbed conditions as dependent variables in statistical models. We evaluated the effect of walking speed by fitting separate linear mixed-effect models on the change in metabolic rate, anticipatory foot placement (mean step width and step length), reactive foot placement (standard deviation in step width and length), and ankle strategy (ankle muscle activation variability) outcomes to test for the effect of walking speed with a fixed slope for speed and random intercept for each subject. In addition, we assessed the effect of perturbations on outcomes describing the control strategy (mean step width/length, step width/length variability, variability of ankle muscle activity) and metabolic rate for each walking speed separately using t-tests. When assumptions underlying parametric tests were violated, we used the Wilcoxon signed-rank test. We used R^2 of the fixed and random effects, Cohen's d for t-tests, and r scores for the Wilcoxon signed-rank test to quantify the effect sizes. We corrected for multiple comparisons to prevent Type-I error inflation due to the number of statistical tests that we performed using the Benjamini-Hochberg procedure. We considered probability values lower than 0.05 as statistically significant.

We explored the contribution of anticipatory, reactive foot placement, and ankle strategies to energy consumption using linear mixed-effect models by predicting between-subject variability in changes in average metabolic rate due to perturbations from changes in outcomes describing the control strategy due to perturbations. Here, we included speed as a categorical variable, outcomes describing the control strategy as fixed effects, and a random offset per subject. Speed was included as a categorical variable as this reduced multicollinearity between variables compared to including speed as a numerical variable. We accounted for the interaction between speed and outcomes describing the control strategy. Outcomes describing the control strategy that did not predict the metabolic rate of stabilizing walking were removed from the model through stepwise elimination based on F-tests for marginal effects while retaining random effects.

We tested whether age may have affected our results. As we aimed at collecting a dataset representative for the active population, the age range was large, i.e. 19 to 65 years old. Therefore, we explored whether adding age as a predictor in the linear mixed-effect models influenced the outcomes. Age did not affect the change in metabolic rate and control outcomes due to perturbations. However, we found some effects of age on the relationship between change in metabolic rate and changes in

outcomes describing the control strategy due to perturbations. Results from this analysis are described in the supplementary material (S2).

Missing data

Missing ankle muscle variability data due to exclusion of some EMG data (see above) were replaced by substitute values for the statistical analysis through data imputation using the mice package in R (16). Substitute values were generated based on a two-level normal model with subject as a random and speed as a fixed effect. Standard deviations of activation for individual muscles were first imputed 30 times, generating multiple datasets. For each dataset, the average standard deviations over the three muscles were computed, and differences between perturbed and unperturbed walking were calculated to obtain values for ankle muscle variability for each imputed dataset. We then fitted linear mixed-effect models on each imputed data set and combined the results. In addition, we evaluated the effect of perturbations per walking speed using t-tests. While residuals of fitted linear-mixed effect models were normally distributed, the change in ankle muscle variability was not normally distributed for every walking speed. Instead of performing t-tests on the imputed dataset, we first bootstrapped 500 times and then imputed the bootstrapped datasets 30 times. We performed t-tests on each separate dataset and pooled the results across datasets.

Departures from preregistration

Hypotheses and methods were preregistered before conducting this study (14). We decided to deviate from the preregistered document on different points. The most important departures from the pre-registration include the decision to apply data imputation for missing data, the use of pairwise testing to assess the effect of perturbations for each speed independently, and to accommodate for multiple comparisons using the Benjamini-Hochberg procedure. A more elaborate description of the changes with respect to the pre-registered document can be found in the supplement (S1).

RESULTS

Statistical tests were performed after scaling outcomes to dimensionless units. For the sake of interpretability, we report in the text the corresponding values for the average subject, i.e. a subject with a leg length of 0.93 m and a body mass of 71.8 kg.

Subjects increased metabolic rate in perturbed compared to unperturbed walking trials and increases in metabolic rate were larger for slower than for faster walking speeds (Figure 2A). The metabolic rate of an average subject increased with 43 W at 0.8 ms^{-1}, 30 W at 1.2 ms^{-1}, and 21 W at 1.6 ms^{-1} (see Table 1), i.e. the metabolic rate associated with control for stabilizing walking decreased by 28 W for every 1 ms^{-1} increase in walking speed (slope=-0.013 $m^{-1}s$, CI=-0.020 – -0.006 $m^{-1}s$, p=0.001, R2=0.62).

Subjects walked with shorter and wider steps in the perturbed compared to unperturbed condition, but step width increased less with increasing speed (see Figure 2B&D). Subjects decreased mean step length by 0.030 m at 0.8 ms^{-1}, 0.019 m at 1.2 ms^{-1}, and 0.024 m at 1.6 ms^{-1}, and increased mean step width by 0.023 m at 0.8 ms^{-1}, 0.017 m at 1.2 ms^{-1}, and 0.012 m at 1.6 ms^{-1} in perturbed versus unperturbed walking (see Table 1). Adaptations in step width, but not step length, depended on walking speed. The average subject reduced adaptations in step width by 0.014 m for every 1 ms^{-1} increase in walking speed (slope=-0.015 $m^{-1}s$, CI=-0.023 – -0.006 $m^{-1}s$, p=0.002, R^2=0.59).

Step length and width were more variable in perturbed than in unperturbed walking, but step length variability increased less with perturbations at faster speeds (see Figure 2C&E). For the average subject, step length variability increased in perturbed versus unperturbed walking by 0.032 m at 0.8 ms^{-1}, 0.020 m at 1.2 ms^{-1}, and 0.014 m at 1.6 ms^{-1} (see Table 1). Changes in step width variability were considerably smaller, the average subject increased step width variability by 0.004 m at 0.8 ms^{-1}, 0.004 m at 1.2 ms^{-1}, and 0.003 m at 1.6 ms^{-1} (see Table 1). Adaptations in step length variability decreased with walking speed by 0.022 m for every 1 ms^{-1} increase in speed for an average subject (slope = -0.024 $m^{-1}s$, CI= -0.028 – -0.020 $m^{-1}s$, $p<0.001$, $R^2=0.77$).

Ankle muscle activation was more variable in perturbed than unperturbed walking but the increase in variability in response to perturbations was smaller at faster walking speeds. Variability in ankle muscle activation increased by 1.05 (60.4 %) at 0.8 ms^{-1}, 0.73 (44.2 %) at 1.2 ms^{-1}, and 0.50 (27.6 %) at 1.6 ms^{-1} in perturbed compared to unperturbed walking (an increase of 1 corresponds to an increase equal to the median activity during unperturbed walking; see Table 1). Variability in ankle muscle activation increased less at fast than at slow walking speeds. Variability in ankle muscle activity decreased by 0.69 with every 1 ms^{-1} increase in speed (CI=-0.87 – -0.50 $m^{-1}s$, $p<0.001$, $R^2=0.77$).

Increases in energy consumption due to perturbations were variable across subjects, and this between-subject variability was partially explained by between-subject differences in changes in mean step length, the interaction between changes in mean step length and speed, and step width variability. Metabolic rate increased more if subjects reduced their step length more, and this was more pronounced at the slowest compared to the fastest walking speed. At 0.8 ms^{-1}, metabolic rate increased by 6.4 W per cm decrease in mean step length for the average subject (or by 640 Wm^{-1}, Table 2), while at 1.6 ms^{-1}, metabolic rate decreased by 0.66 W per cm decrease in step length (or by 66.1 Wm^{-1}). There were no differences between 0.8 and 1.2 ms^{-1}, see Table 2. Subjects who increased step width variability more increased metabolic rate more. Each centimeter increase in step width variability amounted to an increase in metabolic rate of 17.6 W (or 1761 Wm^{-1}, Table 2). Although the increase in metabolic rate per centimeter increase in step width variability is about three times larger than the increase in metabolic rate per centimeter decrease in mean step length, the inter-subject variability in decreases in step length is about 10 times larger than the inter-subject variability in increases in step width variability and therefore inter-subject differences in changes in mean step length explain most of the variability in changes in metabolic cost.

DISCUSSION

In this study, we evaluated the metabolic cost of stabilizing walking in the sagittal plane across speeds and explored its relationship with control strategies. Random treadmill belt speed perturbations (standard deviation of velocity of 0.13 ms^{-1}) elicited average increases in energy consumption of 16.7% at 0.8 ms^{-1}, 9.8% at 1.2 ms^{-1}, and 4.6% at 1.6 ms^{-1}, suggesting that there is a speed-dependent energetic cost associated with stabilizing walking in the sagittal plane. Subjects adopted shorter and wider steps, and more variable foot placement and ankle muscle activation patterns during perturbed as compared to unperturbed walking, suggesting that subjects relied on both anticipatory adjustments of gait kinematics, reactive foot placement, and ankle strategies for stabilizing walking. Similar to changes in metabolic cost, perturbation-induced alterations in kinematics and muscle activity were smaller at faster

than at slower speeds, suggesting that the energetic cost of stabilizing walking is smaller when walking faster because walking faster is inherently more robust against disturbances. In addition, our exploratory analysis showed that between-subject variability in the increase in metabolic cost due to perturbations can – in part – be explained by between-subject variability in control strategies to stabilize walking. There was large variability in the reduction in mean step length when walking was perturbed (interquartile range of approx. 3 cm) and energy consumption increased less in perturbed versus unperturbed walking for subjects who adjusted their step length less, especially at slower speeds. In addition, subjects who increased step width variability more in response to perturbations also increased energy consumption more. Unperturbed walking requires control as well, and it is therefore likely that also during unperturbed walking, the energy cost of stabilizing walking in the sagittal plane is higher at slow than at faster speeds. This is especially relevant for individuals with mobility impairments. Many neuromusculoskeletal disorders lead to both a decrease in preferred walking speed (17–20) and alterations in balance control (21–24), which together might have an important contribution to the increased energetic cost observed in many neuromusculoskeletal disorders (18–20, 25).

Although walking is thought to be inherently more robust against perturbations in the sagittal than in the frontal plane, we showed that there is also a considerable cost associated with stabilizing walking in the sagittal plane. Stabilizing walking is thought to require more active control – and thus energy – in the frontal than in the sagittal plane. Therefore, most previous studies focused on the relationship between frontal plane stabilization of walking and energy consumption (2, 3) but O'Connor et al. (2) evaluated the effect of both mediolateral and fore-aft visual field perturbations. They found that mediolateral but not fore-aft visual perturbations increased the metabolic cost of walking. In contrast, we observed that increases in energetic cost due to sagittal plane perturbations of walking can be considerable as well, i.e., energy consumption increased on average by 16.7% when walking at an average speed of 0.8 ms^{-1} with compared to without treadmill belt speed perturbations. It is likely that our perturbations challenged stability more than the visual perturbations applied by O'Connor et al. (2). In contrast to the belt-speed perturbations applied here, fore-aft visual perturbations did not elicit alterations in foot placement (2). One possible reason for these conflicting findings may be the difference in the nature of the perturbations. Subjects might compensate for visual perturbations by relying more on other sensory inputs, whereas such a strategy is not possible for mechanical perturbations. A challenge with mechanical perturbations is that they might inject or dissipate mechanical energy, and if so, this would complicate the interpretation of metabolic energy results. Here, we assume that no net work was performed by the perturbations on average because the speed was normally distributed around the mean speed. It is important to consider that the metabolic cost of stabilizing walking might be different overground and on a treadmill. Walking on a treadmill might require tighter control of walking speed, as subjects should stay on the treadmill and might avoid getting too close to the edges of the treadmill. When walking overground, people might not care about controlling their forward speed if the sole aim is to reach a destination. Yet, there might be situations, such as when walking with others, where controlling speed becomes an aim during overground walking as well.

Subjects who relied more on anticipatory reductions of step length increased energy consumption more when perturbed, especially at slower speeds. In line with the relationship between increases in

metabolic cost and anticipatory adaptations in the walking pattern observed here, Ahuja and Franz (2022) found that both metabolic cost and step width decreased during adaptation to visual perturbations in the frontal plane (3). Whereas studies investigating the energy cost associated with anticipatory balance control are scarce, the energy cost of voluntary alterations in step length and width has been extensively studied (26, 27). When people are asked to reduce step length and thus increase step frequency while maintaining the same walking speed, they consume more energy (28). The observation that energy consumption increases when subjects walk at higher or lower frequencies than their preferred frequency has contributed to the hypothesis that humans select gait patterns that minimize energy consumption (29). It is unclear whether humans select a control strategy that minimizes energy consumption or prioritize robustness against perturbations over energetic cost. In other words, anticipatory reductions in step length might reduce the need for reactive strategies, which also come at an energetic cost, and it is possible that humans select a combination of anticipatory and reactive strategies by optimizing the overall metabolic cost. Between-subject variability in this trade-off might then be explained by differences in the neuro-musculoskeletal system. Alternatively, anticipatory adjustments might increase robustness to perturbations, and some individuals might prefer this increased robustness even if it comes at an increased metabolic cost.

Sagittal plane perturbations elicited frontal plane foot placement strategies and subjects who relied more on frontal plane reactive foot placement strategies increased energy consumption more. While treadmill belt speed perturbations mainly challenged sagittal plane stability, subjects also made small adjustments in step width that were associated with changes in energy consumption when perturbed. This is in line with simultaneous decreases in energy consumption and step width variability when stabilizing walking in the frontal plane (1) and associations between increases energy consumption and step width variability when applying mediolateral perturbations to the visual field (2). In our study, between-subject variation in step width variability was low and therefore between-subject variability in step width variability explained little of the between-subject variability in energy consumption notwithstanding the high sensitivity of the metabolic cost to step width variability predicted by the statistical model.

It remains challenging to determine contributions of different control strategies to energy consumption based on the experiment performed here. Our results suggest that anticipatory adaptations to the gait pattern contributed most to the energetic cost of stabilizing walking. Yet, individuals who rely more on anticipatory strategies may rely less on reactive strategies. This potential interdependence between anticipatory and reactive control hinders the attribution of changes in energy consumption for stabilizing walking to either an increased reliance on anticipatory or a decreased reliance on reactive control strategies. We used a principal component analysis to explore the interdependence of our five measures of strategies to stabilize walking (mean step length and width, variability of step length, step width and muscle activity; Supplementary Figure S1). All five principal components contributed considerably to explaining between-subject differences in strategies to stabilize walking against perturbations (at least 10% at 0.8 m/s, 4% at 1.2 m/s, and 7% at 1.6 m/s), indicating that no outcomes were redundant. However, at the slowest walking speed, the component explaining most of the variability (31%) couples larger changes in anticipatory step length adjustments to smaller changes in reactive step length variability and ankle muscle variability. This suggests that

subjects who relied more on anticipatory control strategies relied less on reactive responses. This coupling might have prevented us from detecting relationships between the reliance on reactive responses and changes in metabolic energy consumption. To further explore relationships between metabolic cost and control strategies, a larger sample (since outcomes are independent to some degree) or experiments that manipulate the participants' ability to use specific control strategies are required.

The metabolic cost of stabilizing walking is higher at slower than at faster speeds, suggesting that more active control for stabilizing walking is required at slow speeds. At faster speeds, both changes in step width/length and muscle activity, and increases in metabolic cost in response to perturbations were smaller than at lower speeds. An additional analysis showed that not only the change in step length variability but also the absolute step length variability was smaller at high than at low speeds in both unperturbed and perturbed walking (Figure S5). This suggests that increasing speed improves the robustness against perturbations. Hak and colleagues (5) also concluded that walking at faster speeds is inherently more robust against perturbations by evaluating the margin of stability across a combination of speeds and step frequencies in unperturbed walking. Faster speed and higher step frequencies increased the stability margin in the backward direction, while higher step frequency also improved the mediolateral stability margin. The increased robustness at higher speeds might stem from the larger inertia. Furthermore, it is possible that passive dynamics contribute more to step length adjustments at higher speeds contributing to the reduced energetic cost but our approach could not distinguish active and passive adjustments in step length. As far as we know, this study is the first to show the importance of walking speed when studying the energetic cost of stabilizing walking.

The higher energetic cost of stabilizing walking at slow versus fast walking speeds has important implications for many mobility-impaired individuals who walk slower. Mobility impairments, e.g. due to aging or lower-limb amputation, are often associated with slower walking speeds (17, 19, 21). Our results suggest that the slower walking speed might amplify age- or disease-related balance control problems and increase metabolic cost. Here, we used perturbations to assess the energetic cost of walking but control to stabilize walking is also required during unperturbed walking. Sensorimotor processes that underlie human motor control are thought to be stochastic (30), meaning that stability during walking is continuously challenged by noisy sensory information and motor controls. In addition, the environment is uncertain as well, e.g. the floor is never exactly level, and this is especially true when walking outside. Our findings might, therefore, generalize to unperturbed walking. Indeed, when walking is externally stabilized using springs, metabolic cost and variability in foot placement decrease (1). When balance control is compromised by age or disease (31–33), control strategies and, thereby also the energetic cost of stabilizing walking might change. Given the potential effect of aging and the large age range (between 18 and 65 years) of our participants, we explored whether age had an effect on our outcomes. We did not find an effect of age on increases in metabolic rate with perturbations or on changes in mean step length/width, step length/width variability, or variability in ankle muscle activity. We also repeated or exploratory analysis of the relationship between increases in metabolic energy and control strategies with age as an additional variable (Supplementary Table S1). Including age changed the main effects, the main effect of mean step length disappeared and we found a main effect of step length variability, and we found interactions between step width and length variability and age. However, we did not find interaction effects of age when using separate linear mixed effect models for

each control outcome. Given the exploratory nature of this analysis and the different findings when using separate models, results should be interpreted with care. Additional research is needed to confirm associations between metabolic energy consumption and control strategies and to investigate whether and how they change with age.

We interpreted kinematic outcomes in terms of anticipatory versus reactive control strategies based on insights from previous research, but interpreting kinematic outcomes is not straightforward. Given that the perturbations in this experiment were (quasi-)random, participants could not anticipate the direction of the perturbation and make perturbation-specific anticipatory adaptations in step length or ankle muscle activity, which would contribute to increased variability. Foot placement and ankle torque variability were therefore interpreted as an increased use of reactive strategies. In contrast, we interpreted alterations in mean step width and step length as anticipatory strategies to the presence of perturbations. Yet, if reactive increases in step length would have been larger than reactive decreases in step length (or vice versa), this asymmetry would have been reflected in the mean step length. We therefore verified that step lengths were symmetrically distributed, suggesting that the mean step length indeed reflects anticipatory adaptations and not an asymmetry in reactive step length adjustments following forward or backward perturbations (see Figure S4). Additionally, learning over the course of the walking trial might have led to variability in step length, i.e. subjects might have used larger anticipatory adjustments of step length in the beginning versus at the end of the trial, which would contribute to step length variability and lead to a wrong interpretation of step length variability as a signature of reactive responses. By computing step length and width outcomes over the last two minutes of the trials, we limited the effect of learning on our outcomes.

In conclusion, continuous treadmill speed perturbations increased energy consumption associated with stabilizing walking more at slow than at fast walking speeds and this might be explained by walking being more robust at faster than at slower walking speeds. Our results suggest that the use of anticipatory strategies contributed most to the energetic cost of walking control to stabilize walking, especially at slow walking speeds. These findings are especially relevant to explain the increased metabolic cost of individuals with mobility impairments, who often walk slower and use different control strategies.

DATA AVAILABILITY

Data and software for the statistical analysis of this study are openly available at DOI: https://doi.org/10.48804/HQLZC9.

SUPPLEMENTAL MATERIAL

Supplementary text describing deviations from the preregistration document (S1), the analysis of interaction between balance outcomes and age (S2 and Table S1), the analysis of the interdependence between balance outcomes (S3 and Figure S1), details on gait event selection (S4 and Figure S2), a description of the distribution of stepping responses to perturbations (S5 and Figure S4), and a figure presenting step length variability for the unperturbed and the perturbed conditions at the different walking speeds (S6 and Figure S5) can be found at: https://doi.org/10.48804/HQLZC9.

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DISCLOSURES

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AUTHOR CONTRIBUTIONS

FDG, RR, MA, and WM developed the study concept and designed the experiments. After the design of the study, MA and WM preregistered the study. WM acquired and analyzed the data. FDG and WM wrote the manuscript. FDG, RR, MA, and WM interpreted the results and edited the manuscript. All authors contributed to the article and approved the submitted version.

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FIGURE LEGENDS

Figure 1 – Summary of the experimental protocol. ***A.*** *Schematic representation of the order of experimental conditions and the randomization procedure.* ***B.*** *The time series of the treadmill belt velocity in perturbation trials measured at 300Hz (top plot), the histogram (left bottom plot) depicts the number of discrete treadmill velocities with a velocity bin width of 0.01 ms^{-1} over the perturbation trials for each speed, and the power spectrum (right plot) shows the frequency content of the measured treadmill belt velocities for the three different speeds for a representative subject.* ***C.*** *Visualization of the relationship between experimental data and study outcomes (lat. mal. stands for lateral malleolus).*

Figure 2 - Effect of perturbations on metabolic rate and balance control outcomes as a function of walking speed. Graphs depict the difference between perturbed and unperturbed walking in study outcomes. ***A.*** *Effect of perturbations on metabolic rate.* ***B.*** *Anticipatory adaptations in mean step length.* ***C.*** *Changes in the standard deviation (s.d.) of step length characterizing changes in reactive foot placement strategy.* ***D.*** *Anticipatory adaptations in mean step width.* ***E.*** *Changes in the standard deviation (s.d.) of step width characterizing changes in reactive foot placement strategy.* ***F****. Changes in ankle muscle activation variability characterizing changes in ankle strategy. All outcomes are non-dimensional (n.d.).*

TABLES

Table 1 – Difference in study outcomes between perturbed and unperturbed walking conditions at each walking speed. We described the central tendency, relative change compared to the speed-matched unperturbed walking condition, spread, and effect size using the mean, standard deviation (s.d.), cohens-d (d) for parametric tests and median, interquartile range (iqr), test statistic over the square root of the observations (r) for non-parametric tests. We scaled non-dimensional central tendencies (Non. dim. centr. ten.) to an average subject (Scaled centr. ten.). Probability values were corrected for multiple comparisons using the Benjamini-Hochberg procedure.

Δmetabolic rate	*Scaled centr. ten. (median)*	*Relative change (%)*	*Non. dim. centr. ten. (median)*	*Spread (iqr)*	*P-value*	*Effect size (r)*
0.8 ms^{-1}	43 W	16.7	0.020	0.015	<0.001	0.87
1.2 ms^{-1}	30 W	9.8	0.014	0.013	<0.001	0.83
1.6 ms^{-1}	21 W	4.6	0.010	0.018	0.003	0.63
Δmean step length	*Scaled Centr. ten. (median)*	*Relative change (%)*	*Non. Dim. Centr. ten. (median)*	*Spread (iqr)*	*P-value*	*Effect size (r)*
0.8 ms^{-1}	-0.030 m	-4.8	-0.032	0.032	<0.001	0.81
1.2 ms^{-1}	-0.019 m	-2.7	-0.021	0.03	0.001	0.70
1.6 ms^{-1}	-0.024 m	-2.6	-0.026	0.029	<0.001	0.79
Δmean step width	*Scaled Centr. ten. (mean)*	*Relative change (%)*	*Non. Dim. Centr. ten. (mean)*	*Spread (s.d.)*	*P-value*	*Effect size (d)*
0.8 ms^{-1}	0.023 m	11.3	0.025	0.021	<0.001	1.17
1.2 ms^{-1}	0.017 m	8.1	0.018	0.017	<0.001	1.10

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1.6 ms^{-1}	0.012 m	5.9	0.013	0.013	<0.001	1.02
***Δ**var. step length*	*Scaled Centr. ten. (mean)*	*Relative change (%)*	*Non. Dim. Centr. ten. (mean)*	*Spread (s.d.)*	*P-value*	*Effect size (d)*
0.8 ms^{-1}	0.032 m	157.1	0.035	0.010	<0.001	3.41
1.2 ms^{-1}	0.020 m	134.0	0.022	0.006	<0.001	3.53
1.6 ms^{-1}	0.014 m	102.2	0.016	0.006	<0.001	2.67
***Δ**var. step width*	*Scaled Centr. ten. (mean)*	*Relative change (%)*	*Non. Dim. Centr. ten. (mean)*	*Spread (s.d.)*	*P-value*	*Effect size (d)*
0.8 ms^{-1}	0.004 m	24.6	0.004	0.003	<0.001	1.28
1.2 ms^{-1}	0.004 m	25.5	0.004	0.004	<0.001	1.16
1.6 ms^{-1}	0.003 m	18.3	0.004	0.005	0.002	0.75
***Δ**var. musc. act.*	*Non. Dim. Centr. ten. (mean)*	*Relative change (%)*	*Non. Dim. Centr. ten. (mean)*	*Spread (s.d.)*	*P-value*	*Effect size (d)*
0.8 ms^{-1}	1.05	60.4	1.05	0.58	<0.001	1.83
1.2 ms^{-1}	0.73	44.2	0.73	0.40	<0.001	1.83
1.6 ms^{-1}	0.50	27.6	0.50	0.42	<0.001	1.20

Table 2 – Changes in metabolic rate were partially explained by changes in mean step length. The linear mixed-effect model was fitted on dimensionless data. The model that predicted changes in metabolic rate after performing step reduction is described in the table below. Estimated effects can be found in the dimensionless effect column with corresponding confidence intervals (Non. dim. estimates). We scaled dimensionless estimates based on body weight and leg length of an average subject to aid the interpretation of the results (described in the Scaled estimates column). In case there is a speed interaction effect, estimates must be summed to the main effect to obtain the relationship between changes in mean step length and metabolic rate at higher walking speed (e.g., -640 Wm^{-1}(at 0.8 ms^{-1})+706 Wm^{-1}(at 1.6 ms^{-1}) = 66 Wm^{-1}).

Predictors	*Scaled estimates*	*Non. dim. estimates (CI)*	*p*
Intercept	20 W	0.009 (0.003 – 0.016)	0.009
Δstep width variability	1761 Wm^{-1}	0.768 (0.024 – 1.512)	0.043
Δmean step length at 0.8 ms^{-1}	-640 Wm^{-1}	-0.279 (-0.407 – -0.151)	<0.001
Interaction - Δmean step length at 1.2 ms^{-1}	265 Wm^{-1}	0.116 (-0.028 – 0.259)	0.112

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Interaction - Δmean step length at 1.6 ms^{-1}	706 Wm^{-1}	0.308 (0.182 – 0.434)	<0.001
Marginal R^2 / Conditional R^2	0.193 / 0.740		

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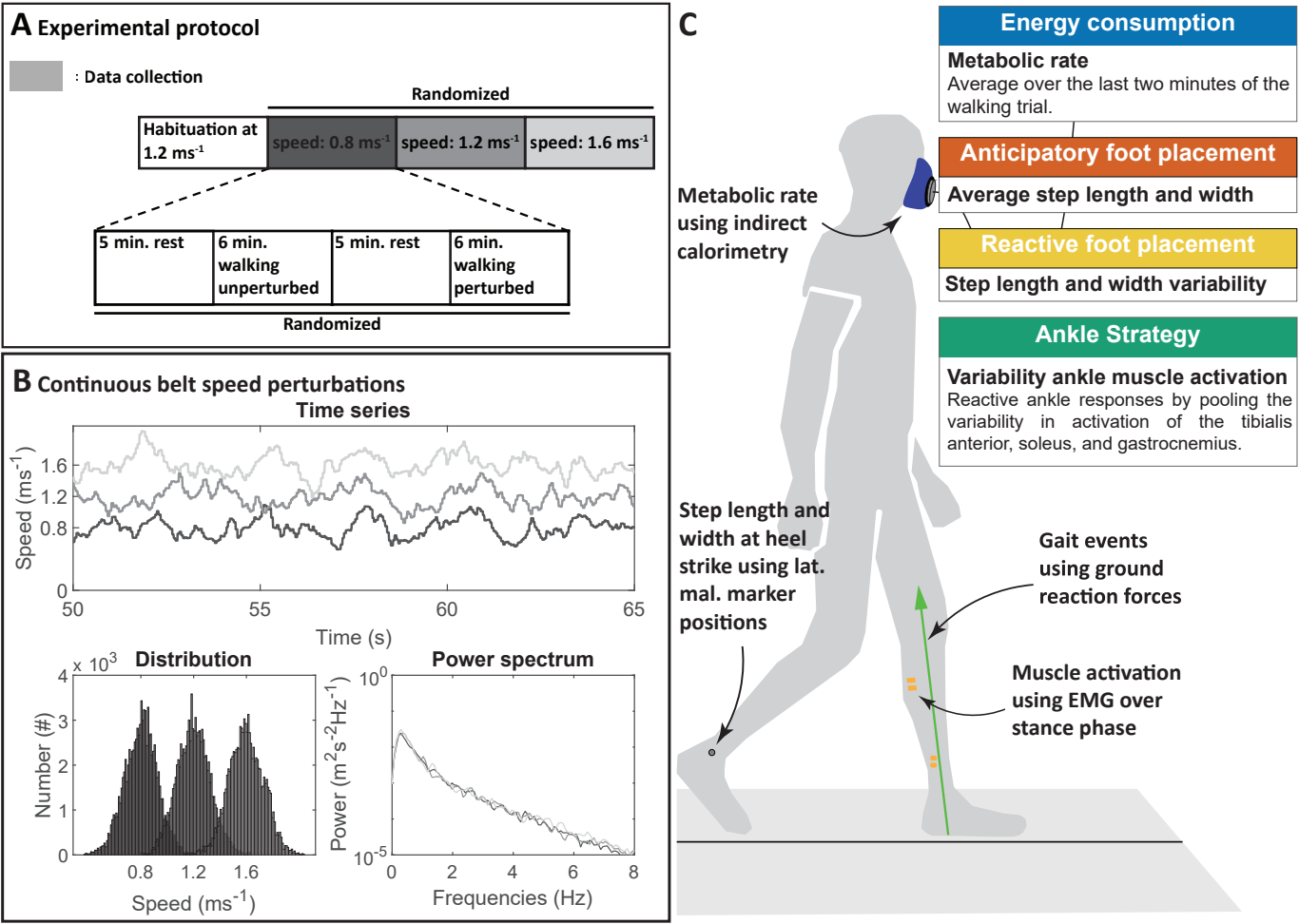
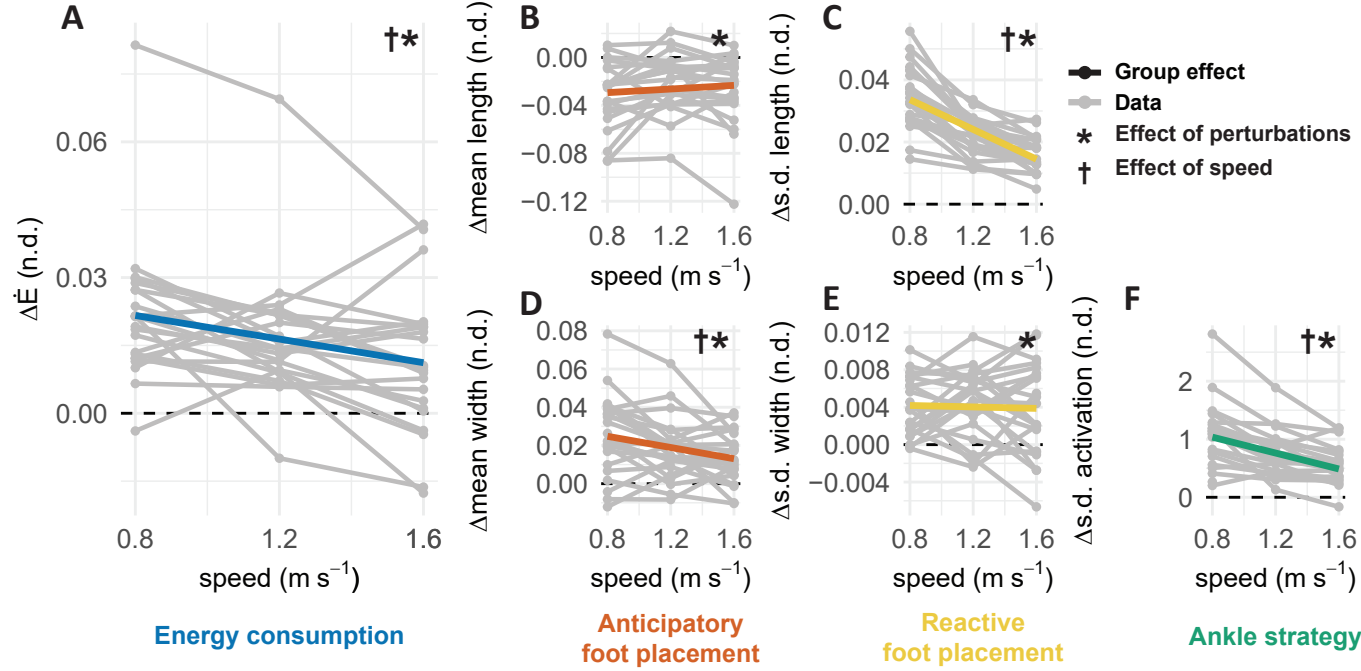

A Experimental protocol
: Data collection
Randomized
Habituation at 1.2 ms-1
speed: 0.8 ms-1
speed: 1.2 ms-1
speed: 1.6 ms-1
5 min. rest
6 min. walking unperturbed
5 min. rest
6 min. walking perturbed
Randomized
B Continuous belt speed perturbations
Time series
Speed (ms-1)
Time (s)
Distribution
Number (#)
Speed (ms-1)
Power spectrum
Power (m2s-2Hz-1)
Frequencies (Hz)
C
Energy consumption
Metabolic rate
Average over the last two minutes of the walking trial.
Anticipatory foot placement
Average step length and width
Reactive foot placement
Step length and width variability
Ankle Strategy
Variability ankle muscle activation
Reactive ankle responses by pooling the variability in activation of the tibialis anterior, soleus, and gastrocnemius.
Metabolic rate using indirect calorimetry
Step length and width at heel strike using lat. mal. marker positions
Gait events using ground reaction forces
Muscle activation using EMG over stance phase


A
†*
0.06
0.03
0.00
ΔĖ (n.d.)
0.8
1.2
1.6
speed (m s^{-1})
Energy consumption
B
*
0.00
−0.04
−0.08
−0.12
Δmean length (n.d.)
0.8
1.2
1.6
speed (m s^{-1})
C
†*
0.04
0.02
0.00
Δs.d. length (n.d.)
0.8
1.2
1.6
speed (m s^{-1})
Group effect
Data
* Effect of perturbations
† Effect of speed
D
†*
0.08
0.06
0.04
0.02
0.00
Δmean width (n.d.)
0.8
1.2
1.6
speed (m s^{-1})
Anticipatory
foot placement
E
*
0.012
0.008
0.004
0.000
−0.004
Δs.d. width (n.d.)
0.8
1.2
1.6
speed (m s^{-1})
Reactive
foot placement
F
†*
2
1
0
Δs.d. activation (n.d.)
0.8
1.2
1.6
speed (m s^{-1})
Ankle strategy

Walking faster decreases energy needed for stabilizing walking in the sagittal plane

Energy consumption

Metabolic rate ($\dot{E}$)

Anticipatory foot placement

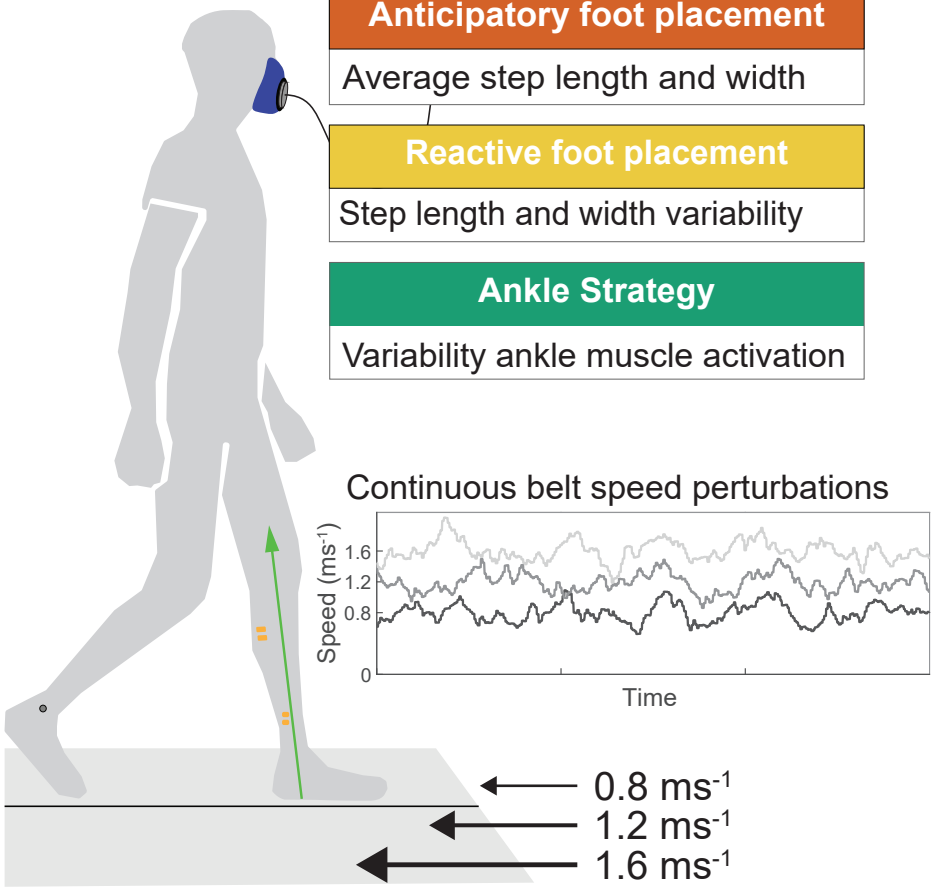
Average step length and width

Reactive foot placement

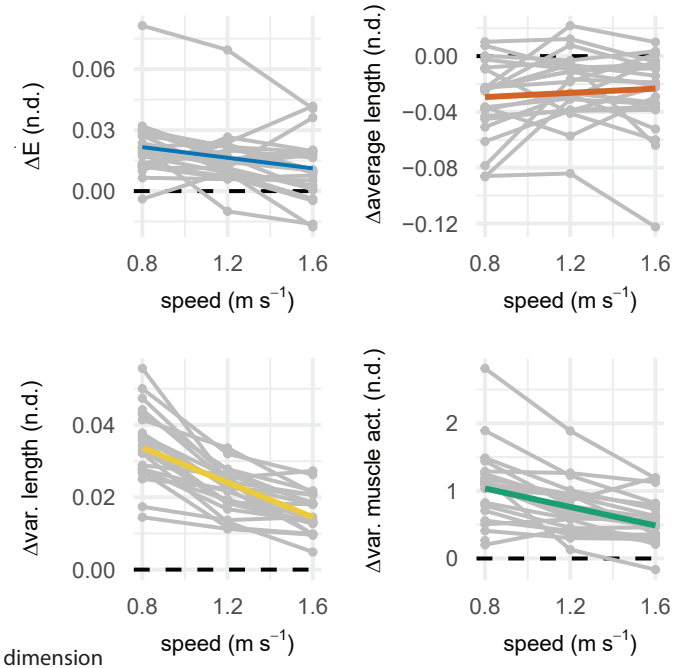
Step length and width variability

Ankle Strategy

Variability ankle muscle activation



Sagittal plane balance perturbations increase the metabolic cost of walking. Perturbations lead to larger changes (Δ) in metabolic rate and control strategies at lower than at higher speeds.



n.d. = no dimension

Conclusion

There is a considerable energetic cost associated with sagittal plane control strategies, especially at slow walking speeds. This may explain increased metabolic cost in mobility impaired individuals who walk slower.