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RESEARCH ARTICLE

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Changes in gait asymmetry may be caused by adaptation of spinal reflexes

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Abstract

In a recent human study, we found that adaptive changes in step length asymmetry (SLA) are correlated with similar changes in the H-reflex gains of the leg muscles during split-belt treadmill locomotion. Although this observation indicated a closer link between gait asymmetry and spinal reflex adaptation, it did not reveal their causal relationship. To better understand this relationship, here we use a neuromuscular model of human walking whose control relies primarily on spinal reflexes. Subjecting the model to split-belt treadmill locomotion with different combinations of belt speed and reflex gain patterns, we find that belt speed changes increase the variability in SLA but do not result in consistent SLA patterns as observed in human experiments, whereas reflex gain changes do. Furthermore, we find that the model produces SLA patterns similar to healthy adults when its reflex gains are adapted in a way similar to the H-reflex changes we observed in our previous study. The model also predicts SLA patterns similar to the ones observed for cerebellar degeneration patients when the reflexes do not adapt beyond a sudden dip at the time the ipsilateral belt speed is lowered. Our results suggest that SLA does not arise from imposing belt speed changes but requires the change of the reflex gains and that the dynamic adjustment of these gains may be an essential part of human gait control when encountering unexpected environmental changes such as uneven speed changes in split-belt treadmill locomotion.

NEW & NOTEWORTHY This work uses computational modeling to investigate the role of spinal reflex tuning during locomotor adaptation. We show, in simulation, that tuning spinal reflex gains leads to gait asymmetry adaptation, not vice versa, and that patterns of gait adaptation on a split-belt treadmill are mostly driven by tuning of spinal reflexes, and not by biomechanical disturbances triggered by belt changes. The model further hints at the cerebellum as the source of spinal reflex modulation.

computational modeling; gait adaptation; neuromuscular model; spinal reflex; split-belt

INTRODUCTION

The human motor system can adapt gait both permanently and transiently in response to neurological diseases, injuries, and gait disturbances (1–3). Understanding the neural mechanisms behind this adaptation can not only provide scientists with a deeper knowledge of human gait physiology but also serve clinicians as a guide for correcting gait pathologies when necessary.

Locomotion on a split-belt treadmill with asymmetric belt speeds has become a well-established paradigm for studying human gait adaptation in the laboratory environment (4, 5). Many of the original works using this paradigm identified key observations about split-belt adaptation, such as that it involves sensorimotor calibration (6) and that supraspinal centers are essential for the adaptation (7, 8). In more recent years, a number of studies have started to focus on the neural origins of these observations. For instance, Iturralde and Torres-Oviedo (9) measured electromyography (EMG) from major leg muscle groups during split-belt adaptation and found that, for some muscles, EMG activity is distinctly different in early versus late adaptation. For another example,

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using electroencephalography (EEG), Jacobsen and Ferris (10) have found that multiple brain areas adapt significantly during split-belt locomotor adaptation. Although these and other examples provide evidence that supraspinal centers are involved in gait adaptation, it remains unclear whether and how this involvement affects locomotor control circuits in the spinal cord.

In a recent study, we combined split-belt locomotion with H-reflex measurements to study spinal reflexes during gait adaptation in able-bodied humans (11). H-reflexes provide a compound measurement of the overall gain of the spinal afferent-motoneuron connections (12, 13). We found that changes in step length asymmetry (SLA) are correlated with similar changes in H-reflex gains of the leg extensor muscles (Fig. 1), suggesting that SLA is accompanied by adaptation of reflex gains, which is defined as dynamic change that accompanies gait adaptation. However, these results did not reveal whether reflex gain adaptation is caused by SLA adaptation or the other way around. Some support for the latter case comes from the work of Thompson et al. (14, 15), who showed that conditioning of reflexes over months can correct gait asymmetry in people with paraplegia due to spinal cord injury. Yet the reflex gain adaptation we observed occurred over the course of mere minutes and in near lockstep with the SLA changes (Fig. 1).

Here, we use a neuromuscular model of human walking on a split-belt treadmill to investigate the causal relationship between SLA and spinal reflex gain adaptation. The model's control primarily consists of muscle reflexes (encoding muscle lengths and forces). Using computer simulations, we adapt the gains of these reflexes in different combinations along with the belt speeds of the simulated treadmill. We find that in the model, reflex gain adaptation causes gait asymmetry rather than being a result of it. We also find that tuning reflex gains closer to baseline accounts for the reductions in gait asymmetry that occur during adaptation to the split-belt condition, a feature absent in cerebellar degeneration patients (8). Finally, we discuss the implications of these findings for the physiological processes underlying gait adaptation in the human motor system.

METHODS

Neuromuscular Model

We used a two-dimensional (2-D) version of a previously published neuromuscular model of human walking (16, 17) (Fig. 2*A*). The model can walk at different speeds and stabilize against gait disturbances such as sudden speed changes of a simulated treadmill (18). The model includes the major leg muscle groups involved in sagittal plane walking (see Fig. 2*A* for a diagram of the muscles included). All muscles are represented as Hill-type muscles that include series and parallel elasticity with static moment arms. The model is simulated in MATLAB Simulink (SimMechanics, MathWorks). The model neural control distinguishes between stance and swing. During swing, the control drives the leg into a target pose that is adjusted based on stance width (Δx) and center of mass speed (v_{CM}) to stabilize gait. During stance, the control relies on reflexes based on proprioceptive (muscle lengths and forces), vestibular (trunk lean), and mechano-receptor (perceived leg loading and foot contact) inputs to generate locomotion behavior. The strength of an individual reflex is characterized by a gain G^i. For instance, the soleus muscle stimulation $s(t)$ in the model is controlled by a time-delayed, positive force feedback, $s_{SOL}(t) = s_0 + G^{SOL}F^{SOL}(t - \Delta t)$, whose strength is determined by the reflex gain G^{SOL}. Note that although the gains in the model are most closely associated with the synaptic strength of the reflex connections, these net gains could also change due to changes in presynaptic inhibition or motoneuronal excitability. The model cannot distinguish between these alternatives.

The model control consists of two components: one for stance and one for swing phase. The stance phase controller consists of positive force feedback to the ankle and knee extensors (soleus, gastrocnemius, and vastus): the main muscles driving leg rebound behavior during stance. The hip flexor and extensor are driven by a proportional-derivative controller to stabilize the trunk at a certain angle during stance (set to 5° for all simulations in this work). The hamstrings are also driven by trunk angle feedback to prevent

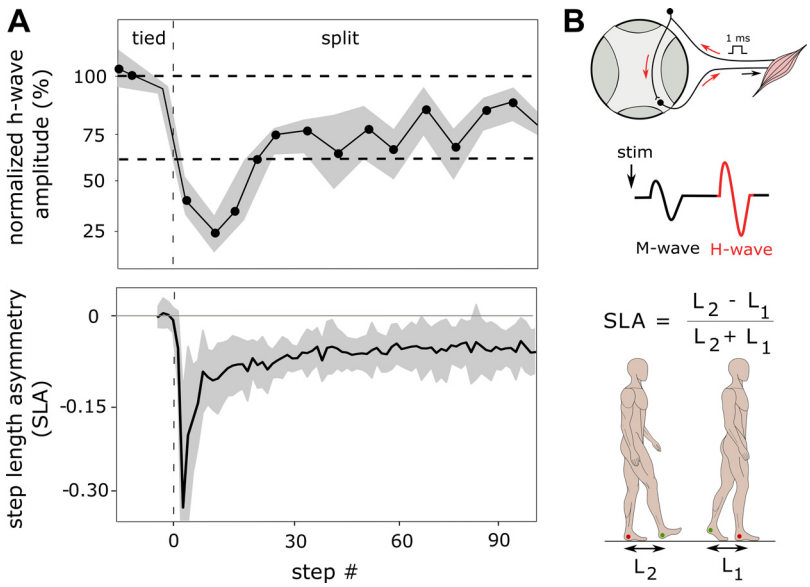

Figure 1. Step length asymmetry (SLA) and H-reflex gains of the leg extensor muscles during split-belt locomotion. *A*: H-reflexes and SLA adapt similarly when unilateral speed changes are introduced during split-belt locomotion (adapted from Ref. 11). *B*: schematic of H-reflex measurement with M- and H-wave responses highlighted (*top*), and definition of SLA (*bottom*). H-reflex amplitudes were reduced to average values obtained at baseline walking trials at matching tied-belt speeds.

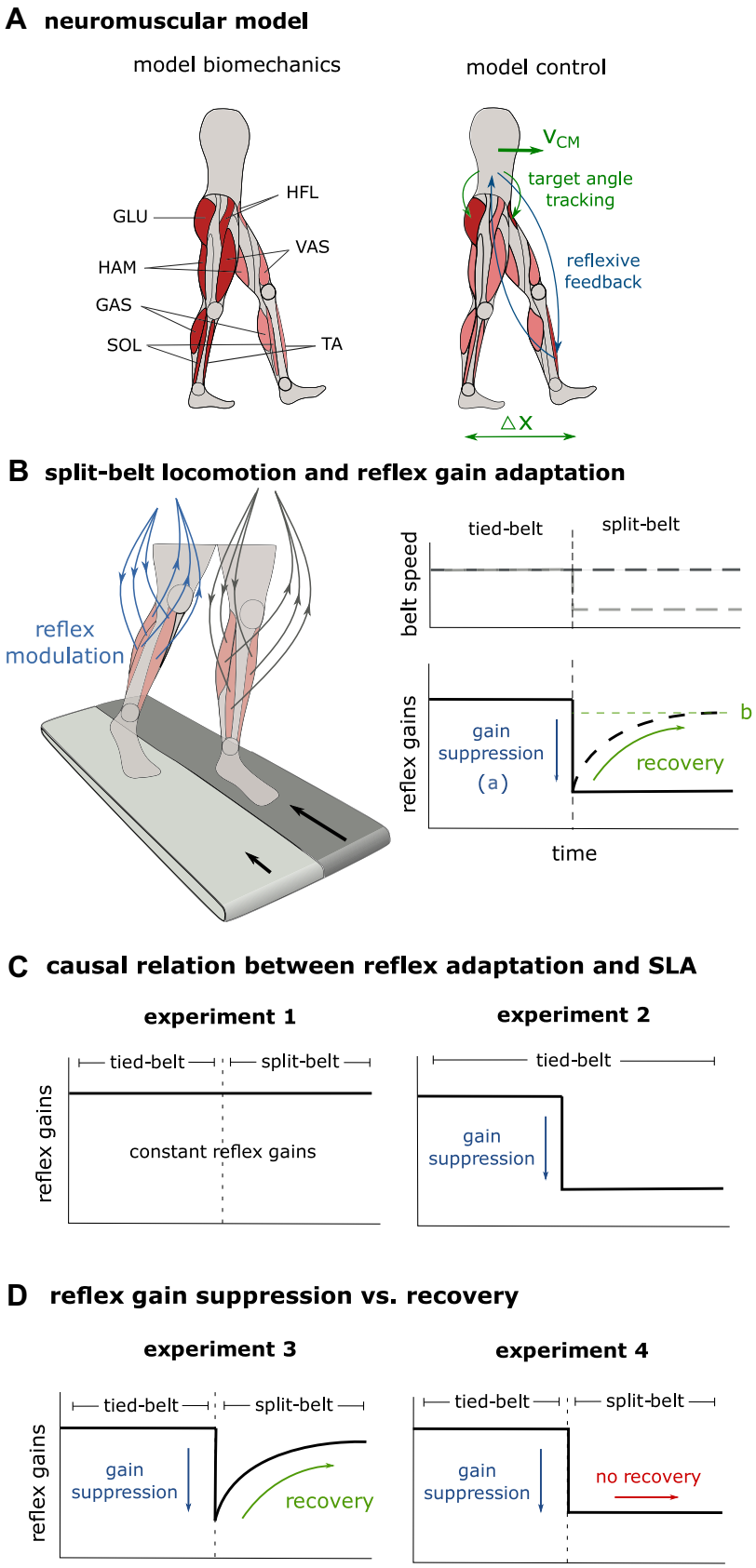

Figure 2. Overview of neuromuscular model and simulation experiments with four combinations of belt speed changes and reflex gain adaptations. *A*: two-dimensional (2-D) neuromuscular model with leg muscles (GAS, gastrocnemius; SOL, soleus; TA, tibialis anterior; BFsH, biceps femoris short head; HAM, hamstrings; GLU, gluteus maximus; HFL, hip flexors) and main control elements indicated. *B*: reflex gain adaptation function (*Eq. 1*). *C*: *simulation experiments 1* and *2* for studying causal relationship between gait asymmetry and reflex gain changes. *D*: *simulation experiments 3* and *4* for studying the roles of reflex gain suppression and recovery during split-belt locomotor adaptation.

knee overextension resulting from hip extension moments. The tibialis anterior is controlled by positive length feedback from itself and a negative force feedback from the soleus to clear the ankle and prevent ankle overextension in the late stance. The stance control gains are the only gains that were modulated in the simulations presented here. The swing control consists of force and length feedbacks to hip muscles, hamstrings, and tibialis anterior, plus an additional controller to drive the hip angle toward a target angle. The target angle is determined by step width and center of mass speed to ensure leg placement at a stable location (see Refs. 16 and 17 for more details).

Reflex Gain Modulation

To explore the causal relationship between reflex gain adaptation and SLA, we introduced in the model an overall gain adaptation $G^i(t) = \alpha(t)G_0^i$ with

$$\alpha(t) = \begin{cases} 1 & t \le t_0 \\ b + (1 - a - b)e^{-(t-t_0)/\tau} & t > t_0 \end{cases} \quad (1)$$

to all the reflexes of the leg on the belt undergoing speed changes (ipsilateral leg). The adaptation is parameterized by an initial gain suppression a at the time t_0 of the belt speed change, a gain recovery time constant τ ($\tau = 10$ s throughout the paper), and a steady-state level b at which the reflex gains settle after adaptation (Fig. 2*B*). Although motivated by the time course of the H-reflex changes seen in the experiments (compare Fig. 1), $\alpha(t)$ can also represent persistent suppression without recovery in the reflex gains ($b = 1 - a$) and no gain changes at all ($a = 0$, $b = 1$). Note that although we modulate all stance phase gains indiscriminately, this mainly affects the leg extensor muscles, since the hamstrings and hip muscles mainly contribute to trunk stabilization and prevention of knee overextension, and the tibialis anterior contributes to foot clearance.

Spinal Gain Optimization

We used optimization of the initial gains G_0^i to identify walking solutions of the neuromuscular model when it is subjected to different experimental conditions (*simulation experiments 1* through *4* indicated in Fig. 2, *C* and *D* and detailed in *SLA Effect of Reflex Gain Change Rather than Belt Speed*; *Imposing Gain Suppression Followed by Recovery Leads to SLA Pattern Observed in Healthy Adults*; *Imposing Gain Suppression without Recovery Leads to SLA Pattern Observed in Cerebellar Degeneration Patients*). Specifically, we used simple Bayesian optimization with Gaussian process prior (19) to minimize the cost function

$$J = \frac{1}{2}(N_{\text{steps}}^{\text{des}} - N_{\text{steps}})^2, \quad (2)$$

where $N_{\text{steps}}^{\text{des}}$ is a desired number of steps (set to 32 steps for a maximum simulated time of 40 s throughout this work), and N_{steps} denotes the actual steps taken by the model. The model showed stability over greater numbers of strides. Simulation times were used to roughly match typical cadence observed in human experiments, and to allow enough time for the model to adjust gait over several strides. At the start of each optimization, we set the gain parameters G_0^i to baseline values reported in earlier work (16, 17) and initialized the standard deviation of the Gaussian noise in the parameter space to 50% of these values. The optimization then repeatedly generated five new parameter sets by adding Gaussian noise to the baseline values, simulated the model for each parameter set, and chose the best-performing set as the new baseline. With each iteration, we halved the standard deviation. In addition, we limited the range of the reflex gain values to between 0% and 250% of their initial values. The 0 to 250% range was determined based on suppression (up to 70%) and facilitation (up to 60%) levels reported in our prior work (11), and typical ranges of H-reflex modulation reported in other studies (20–22). For each experiment, we repeated the optimization five times to account for its stochastic nature and performed statistical analysis (see *Statistical Analysis*) of this group of optimization runs when investigating SLA changes in different experimental conditions.

Definition of Step Length Asymmetry

We measured SLA in the model according to the conventions used in the literature (5, 8). For two subsequent steps in the model, we used the distance between the left and right ankles at their respective heel strikes to define the step length L_{step}. We then measured SLA as the difference between the left and right (or vice versa) step lengths, using

$$\text{SLA} = \frac{L_{\text{step}}^{\text{r/l}} - L_{\text{step}}^{\text{l/r}}}{L_{\text{step}}^{\text{r}} + L_{\text{step}}^{\text{l}}} \quad (3)$$

in simulations with tied belts of the treadmill (*experiment 1*) and

$$\text{SLA} = \frac{L_{\text{step}}^{\text{fast}} - L_{\text{step}}^{\text{slow}}}{L_{\text{step}}^{\text{fast}} + L_{\text{step}}^{\text{slow}}} \quad (4)$$

in simulations with split belts (*experiments 2–4*), where "fast" and "slow" refer to the leg moving on the faster and slower belt, respectively (Fig. 1).

Statistical Analysis

To compare the SLA adaptation observed in the four simulation experiments, we used several statistical tests. Specifically, we used two-way ANOVA (23) to compare the same time intervals across the simulated experiments (see, *SLA Effect of Reflex Gain Change Rather than Belt Speed*) and one-way ANOVA to compare between different time intervals of each simulated experiment (see, *Imposing Gain Suppression Followed by Recovery Leads to SLA Pattern Observed in Healthy Adults*). If ANOVA yielded a statistically significant difference in SLA, we performed post hoc *t* tests to identify statistically significant differences among different time intervals. In addition, we corrected the *P* values of t-post hoc *t* tests for false discovery rates using the Benjamini-Hochberg method (24) and used one-sample *t* tests to check whether the SLA is different from zero during tied-belt walking. Throughout this paper, we used 0.05 as the cutoff *P* value for statistical significance.

RESULTS

SLA Effect of Reflex Gain Change Rather than Belt Speed

To better understand what causes SLA, we performed two simulation experiments. In the first experiment (*experiment 1*),

we lowered the speed of the ipsilateral belt 10 s into the simulation by 30% ($t_0 = 10$ s) but left the reflex gains of the model constant throughout ($a = 0$, $b = 1$). In the second experiment (*experiment 2*), we did not change the belt speed but instead lowered the reflex gains at t_0 by 20% on the ipsilateral leg of the model ($a = 0.2$, $b = 0.8$). We expected either asymmetry to induce SLA in the model. However, the walking solutions that the neuromuscular model converged to express asymmetric gait only in response to reflex gain changes.

Figure 3 summarizes the SLA changes observed for the optimized neuromuscular model (mean and SD from five optimization runs per experiment) in the two experiments. Analyzing the SLA among four time intervals (right before and right after t_0, midway through, and at the end of the recovery phase), we find a statistically significant interaction between the two groups of solutions (two-way ANOVA, $F = 2.81$, $P = 0.002$). We further observe that the model solutions in *experiment 1* show increased SLA variability when introducing asymmetric belt speeds but no statistically significant trend ($P > 0.05$ between all intervals) (Fig. 3*A*). In contrast, model solutions in *experiment 2* consistently show significant changes in SLA when the reflex gains drop at t_0 (post hoc t tests: $P = 0.00016$, $P = 0.0018$, and $P = 0.0005$ between the first and all subsequent intervals, respectively) but not thereafter ($P > 0.25$ between *intervals 2–4*) (Fig. 3*B*). Thus, consistent trends in SLA are caused by changes in the reflex gains rather than by the mechanical asymmetry introduced with belt speed changes.

Imposing Gain Suppression Followed by Recovery Leads to SLA Pattern Observed in Healthy Adults

To further test whether reflex adaptation could affect the SLA pattern observed in our previous human study (11) (Fig. 1), we optimized the neuromuscular model for walking with changes in belt speed and reflex gains similar to the ones observed in that study (*experiment 3*). Specifically, we optimized the model to walk on a simulated treadmill with an initially tied belt speed of 1.0 m·s^{-1} that dropped by 30% on the ipsilateral side after 10 s. At the same time, the reflex gains of the model's ipsilateral leg adapted according to *Eq. 1* with $t_0 = 10$ s, $a = 0.30$, and $b = 0.90$, mimicking the changes observed for the H-reflex in our human study (sudden gain drop by 80% at speed change followed by recovery to 90% of initial gain within ~45 s, compare Fig. 1).

The mean and standard deviation of the SLA patterns obtained from five optimization runs is summarized in Fig. 4*A* (see Supplemental Video S1 for an example of resulting model gait in *simulation 3*). There are statistically significant differences among the four time intervals introduced in the last section (one-way ANOVA, $F = 57.9$; $P = 1.4 \times 10^{-19}$). These differences occur not only between before (*interval 1*) and after the speed change (*intervals 2–4*, post hoc t-tests, $P = 6.8 \times 10^{-8}$, $P = 3.2 \times 10^{-9}$, and $P = 1.5 \times 10^{-5}$, respectively) but also between right after the speed change (*interval 2*) and later (*intervals 3* and *4*, $p = 0.002$ and $P = 2.1 \times 10^{-5}$, respectively), and between midway through the recovery (*interval 3*) and at its end (*interval 4*, $P = 2.6 \times 10^{-4}$). Overall, the SLA patterns that the model solutions produced in repeated optimization runs are consistent and followed closely the timescale of reflex gain changes. They also resemble the SLA pattern observed in our human study (11) (compare Fig. 1) and reported in the literature (4, 5, 8, 25).

Imposing Gain Suppression without Recovery Leads to SLA Pattern Observed in Cerebellar Degeneration Patients

Cerebellar degeneration patients show a different pattern of SLA when they are exposed to ipsilateral belt speed reductions in split-belt treadmill locomotion experiments. Although their SLA rapidly changes when the speed of one leg reduces, it does not recover to baseline over time (8). We found that we can reproduce this behavior in the model when we optimize it with the reflex gains unchanged after their initial drop ($a = 0.25$ and $b = 0.75$ in *Eq. 1*) (*experiment 4*, Fig. 4). In this case, the repeated optimization led to solutions whose SLA is significantly different before and after the split-belt speed transition (one-way ANOVA: $F = 50.6$; $P = 1.9 \times 10^{-16}$; post hoc t tests: $P = 4.2 \times 10^{-6}$, $P = 2.7 \times 10^{-6}$, and $P = 4.9 \times 10^{-6}$, between *intervals 1* and *2–4*, respectively) but showed no significant difference between the intervals following the transition ($P > 0.2$ for all tests among *intervals 2–4*). Hence, as for cerebellar degeneration patients, the SLA did not recover to baseline.

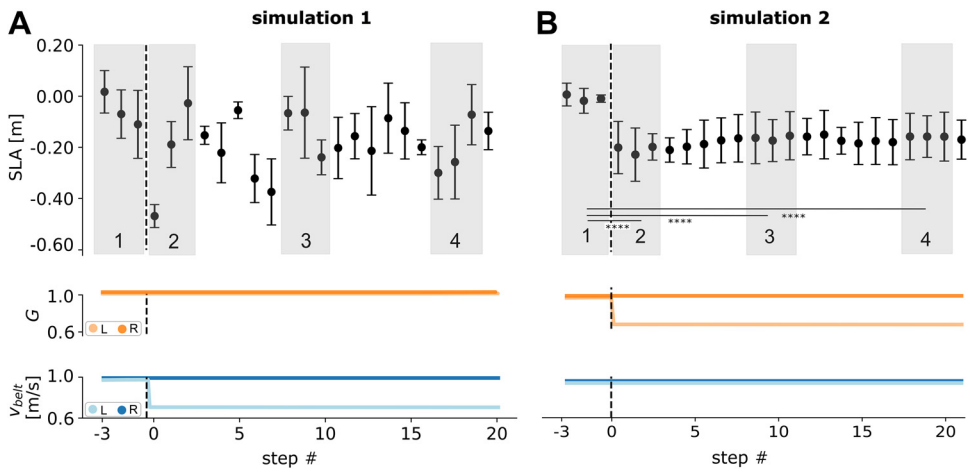

Figure 3. Cause of step length asymmetry (SLA) changes. *A*: when imposing belt speed changes without reflex gain changes, model solutions show increased variability in SLA but no trend. *B*: when imposing reflex gain changes without belt speed changes, model solutions show clear change in SLA. Error bars represent one SD; ****$P < 0.0001$.

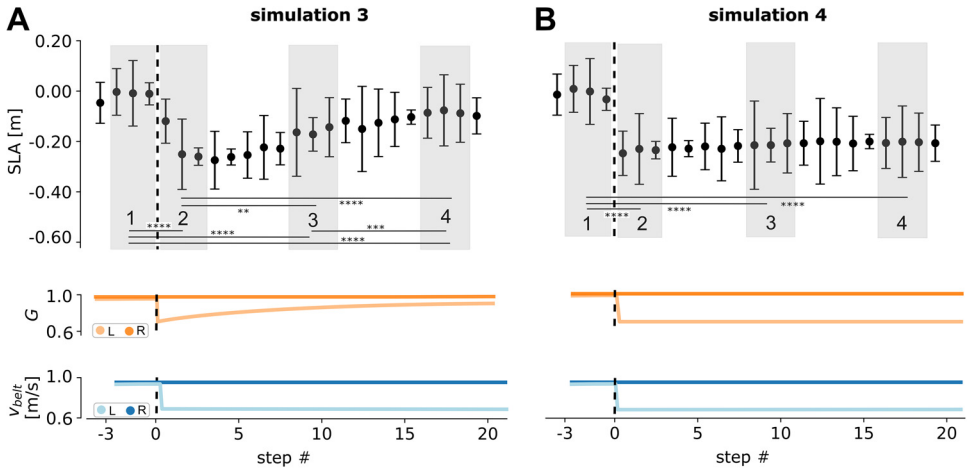

Figure 4. Reproducing step length asymmetry (SLA) patterns observed in healthy young adults and cerebellar degeneration patients. *A*: with reflex gain adaptation similar to observed H-reflex changes, model solutions converge to SLA pattern of healthy young adults (compare Fig. 1). *B*: without gain recovery after belt speed transition, model solutions show no long-term adaptation of SLA, a pattern observed in cerebellar degeneration patients. Error bars represent one SD; $^{**}P < 0.01$, $^{***}P < 0.001$, $^{****}P < 0.0001$.

DISCUSSION

We used a neuromuscular model of human gait to study the causal relationship between spinal reflex adaptation and gait asymmetry in split-belt treadmill locomotion. Optimizing the model to walk in simulated treadmill experiments that isolate the effects of reflex adaptation and belt speed changes, we found that consistent trends in gait asymmetry emerge from the former and not the latter (Fig. 3). Moreover, we found that the model produces SLA patterns similar to healthy adults (4, 5, 11, 25) when the reflex gains adapt in a way similar to the H-reflex changes we observed in a previous human study (11) (Fig. 4*A*). This adaptation is characterized by a gain suppression immediately after the ipsilateral belt speed is reduced followed by a longer recovery toward the gain before the speed change. Finally, we demonstrated that if the recovery part of the adaptation is not taking place in the model, it produces SLA patterns similar to those observed for cerebellar degeneration patients (8) (Fig. 4*B*).

Our results support the hypothesis that spinal reflex changes directly cause gait asymmetry in human locomotion. Previous studies have reported an indirect causal link; more specifically, Thompson et al. (14) found that down-conditioning the soleus H-reflex in spinal cord injured people can reduce gait asymmetry, corroborating earlier observations made by Chen et al. (26) in spinal cord injured rats. However, these changes in gait asymmetry occurred over the course of months and were likely the outcome of reduced hyper-reflexia due to the down-conditioning (15, 27). Our recent observation of near synchronous changes in the soleus H-reflex and the SLA during split-belt locomotion indicated a closer link between spinal reflex adaptation and gait asymmetry (11) (Fig. 1), and the modeling results obtained here provide evidence for changes in gait asymmetry to be directly caused by reflex gain changes.

The functional purpose of reflex gain adaptation and what mechanisms regulate it remain unclear. In the neuromuscular model, the gain changes were imposed by us (*Eq. 1*), and without them, the model optimization produced walking solutions that were more variable. More exactly, these solutions had walking patterns with somewhat erratic large and small steps due to repeated near stumbles, which accounts for the enlarged SLA variability observed in the first simulation experiment (Fig. 3*A*). Thus, in the model, reflex adaptation is needed to maintain stable walking, especially, right after the belt speed change. Whether humans adapt their reflexes for the same reason remains unclear. Although the predictive capabilities of the neuromuscular model (18, 28) and similar other ones (29, 30) have been demonstrated to some extent, these models clearly simplify the human motor control system. The human system could include many other circuits, such as oligosynaptic spinal circuits based on the stretch velocity of the hip muscles that modulate the gains of simpler reflex circuits in proportion to the current walking speed (31, 32). Such a layered network would necessarily change the gains of the simpler reflexes at the belt speed change, and the observed gain recovery thereafter could reflect a drift toward a new gain equilibrium as the gait biomechanics adjust to the new situation. In this scenario, none of the gain changes would be in response to a less stable walk.

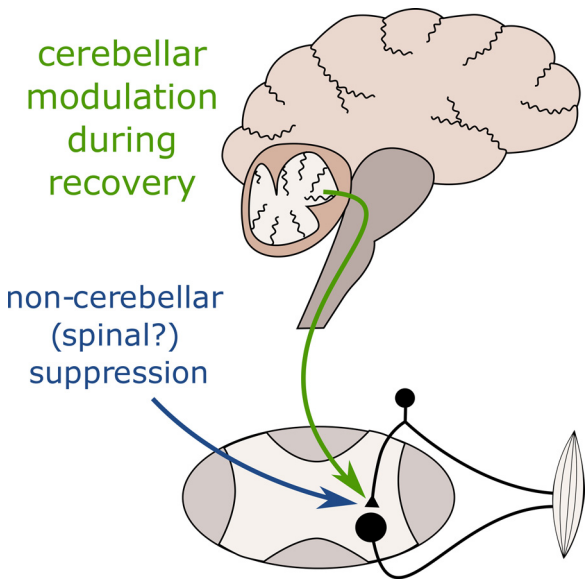

Figure 5. Overall picture of spinal gain adaptation during split-belt locomotion. Based on our results, we speculate that the cerebellum modulates spinal reflex gains during the SLA recovery phase, while the initial gain suppression is of noncerebellar origin.

Some indirect evidence that the immediate gain change at the belt speed transition may indeed be of spinal origin comes from animal studies. Cats respond to belt speed changes in split-belt locomotion with an asymmetric gait, although they lack the longer-term recovery toward gait symmetry seen in humans. This pattern is likely of spinal origin, as it persists in spinalized cats (33). Furthermore, the pattern also shows the modulation of cutaneous reflexes, which are reduced in correlation with the belt speed asymmetry in both the intact and spinalized cat (34). Together, these observations are consistent with the idea that the change of the reflex gains, and hence the gait asymmetry, at the belt speed transition is an immediate reaction of the spinal circuitry.

On the other hand, our observation that the model's SLA pattern matches those of cerebellar degeneration patients (8) when its gain recovery is removed (Fig. 4*B*) allows for speculation that the gain recovery is regulated by supraspinal rather than spinal inputs. The cerebellum plays a critical role in modulating reflexes in other scenarios; for instance, it has been shown that cerebellar lesions can prevent the ability of rats to down-condition their lower limb H-reflexes (35) and that such lesions eliminate conditioned eye blink reflexes in rabbits and saccadic eye movements in monkeys (36, 37). Closer to our current results, MacKay and Murphy (38) review substantial evidence that the cerebellum continuously modulates the gains of proprioceptive reflexes involved in limb movement. Given our model observations, we speculate that the cerebellum also regulates the reflex gain recovery in human split-belt walking and that the H-reflexes in patients with cerebellar degeneration will suddenly change at the belt speed change but not recover thereafter.

In summary, the overall picture that emerges is that the dynamic adaptation of spinal reflex gains may be an essential part of human gait control when encountering unexpected external changes such as the speed changes during split-belt locomotion, and that this adaptation has two components, an immediate response perhaps originating from the spinal circuitry itself and a longer-term recovery governed by the cerebellum (Fig. 5).

DATA AVAILABILITY

The data underlying the results of this work is included in the article. Raw data and other supplementary data may be shared upon request to the corresponding authors. Source data used for the paper figures are available online and can be accessed through https://www.doi.org/10.5281/zenodo.13147873.

SUPPLEMENTAL MATERIAL

Supplemental Video S1: https://doi.org/10.5281/zenodo.13147873.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

O.R., D.J.W., and H.G. conceived and designed research; O.R. and O.M. performed experiments; O.R. and O.M. analyzed data; O.R., D.J.W., and H.G. interpreted results of experiments; O.R. prepared figures; O.R. and H.G. drafted manuscript; O.R., D.J.W., and H.G. edited and revised manuscript; O.R., D.J.W., and H.G. approved final version of manuscript.

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