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Muscle function may depend on model selection in forward simulation of normal walking

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Abstract

The purpose of this study was to quantify how the predicted muscle function would change in a muscle-driven forward simulation of normal walking when changing the number of degrees of freedom in the model. Muscle function was described by individual muscle contributions to the vertical acceleration of the center of mass (COM). We built a two-dimensional (2D) sagittal plane model and a three-dimensional (3D) model in OpenSim and used both models to reproduce the same normal walking data. Perturbation analysis was applied to deduce muscle function in each model. Muscle excitations and contributions to COM support were compared between the 2D and 3D models. We found that the 2D model was able to reproduce similar joint kinematics and kinetics patterns as the 3D model. Individual muscle excitations were different for most of the hip muscles but ankle and knee muscles were able to attain similar excitations. Total induced vertical COM acceleration by muscles and gravity was the same for both models. However, individual muscle contributions to COM support varied, especially for hip muscles. Although there is currently no standard way to validate muscle function predictions, a 3D model seems to be more appropriate for estimating individual hip muscle function.

Keywords

Normal walking; Muscle function; Forward simulation; OpenSim; Modeling; 2D; 3D

1. Introduction

Human walking requires the coordination of the neuromuscular system to support body weight. However, muscle forces are not directly measurable and each muscle can accelerate multiple segments and joints it does not span (Zajac et al., 2002, 2003). Therefore, the relationships among neural control, muscle coordination and walking patterns are not readily accessible from experimental measurements.

Recently, computer simulations actuated by muscles have risen rapidly in order to understand the causal relationship between individual muscle function and walking patterns (Anderson and Pandey, 2001; Neptune, 2000; Pandey, 2001). But the complexity of the models was quite different depending on the objective of the study or the feasibility of using complex models. Some studies (Davy and Audu, 1987; Piazza and Delp, 1996; Neptune et al., 2001; Higginson et al., 2006) have used two-dimensional (2D) models to simulate the sagittal plane movement only. Other studies (Anderson and Pandey, 2001; Goldberg et al., 2004; Liu et al., 2006)

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Conflict of interest statement

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generated three-dimensional (3D) simulations over half of a gait cycle. Both 2D and 3D simulations can reasonably reproduce experimental data but there are some differences in the results of predicted muscle function. For example, Liu et al. (2006) using a 3D model found that hamstrings did not substantially contribute to either progression or support, while Neptune et al. (2004), with a 2D model, reported that hamstrings accelerated the body forward and provided some support during early stance. Moreover, Neptune et al. (2004) found that ankle plantarflexors provided center of mass (COM) support from early stance phase (30% gait cycle), while Anderson and Pandy (2003) using a 3D model, reported that ankle plantarflexors only provided COM support from late stance phase (50% gait cycle).

It is possible that the variation in muscle function reported in the literature (Anderson and Pandy, 2003; Liu et al., 2006; Neptune et al., 2001, 2004) was due to different number of degrees of freedom (DOFs) included in their models. Chen (2006) found that individual joint moments to ground reaction forces and segmental powers were different between models, even when net contributions were identical across different models. Moreover, increasing the DOFs of the model resulted in greater power redistribution attributed to the joint moments. Patel et al. (2007) reported that modeling the pelvis and trunk as separate segments impacts the interpretation of the role of the joint moment during normal walking. However, both studies used torque-driven models and only analyzed the roles of joint moments. To our knowledge, there exists no literature that documents how predicted muscle function would vary when changing the number of DOFs of the model.

In this study, we built 2D and 3D muscle-driven forward simulations in OpenSim (Delp et al., 2007) to reproduce the same normal walking data using different DOFs. Muscle excitations and contributions to COM support were computed and compared. The aims of this study were to (1) determine whether the 2D model can track sagittal plane movement similar with the 3D model, (2) identify any differences between predicted muscle excitations in 2D and 3D models and (3) compare calculated muscle contributions to COM support in 2D and 3D models. We hope to provide a guideline for deciding whether a 2D or 3D approach should be considered for normal walking simulation.

2. Methods

2.1. Experimental data

Three-dimensional kinematic and kinetic data were collected from nine healthy young subjects (height: 178.5 ± 5.1 cm; weight: 75.1 ± 6.6 kg; age: 23.1 ± 6.0 years) walking on a split-belt, motorized treadmill (Bertec Corp.) at their self-selected speeds. Subjects signed an informed consent approved by the human subjects review board. A 6-camera motion analysis system was used to record the 3D locations of 27 markers in the static trial and 23 markers in the walking trial at 60 Hz. Ground reaction forces and torques were recorded from the treadmill at 600 Hz. Experimental data were post-processed in Evart 4.4.3 (Motion Analysis Corp.) and Matlab (MathWorks Inc.).

2.2. Musculoskeletal models

In order to compare muscle function differences between models, we built two musculoskeletal models (Table 1) to generate simulations of the walking trials. The first model (Delp et al., 2007) was a 3D model with 13 segments and 12 joints which enabled movements in the sagittal, frontal and coronal planes. It had 23 DOFs and was actuated by 54 muscle-tendon units. The head, arms and torso (HAT) was modeled as a rigid segment with three rotational DOFs relative to the pelvis. The pelvis could rotate and translate in all three dimensions with respect to the ground. The hip joint was modeled as a ball-and-socket joint with three rotational DOFs. The knee was represented as a one DOF joint, in which the tibiofemoral translations and nonsagittal

rotations were constrained functions of knee flexion angle. The ankle-subtalar complex was represented by two revolute joints aligned with anatomical axes. The metatarsophalangeal joint was modeled as a one DOF hinge joint to allow toe flexion and extension.

The second model was a 2D model which enabled movement in the sagittal plane only. It had the same numbers of segments, joints and muscles as the 3D model. However, it only had 10 DOFs of freedom, all of which were in the sagittal plane. Specifically, the HAT only had one rotational angle with respect to pelvis. The pelvis had two translations and one rotation. The hip and ankle joints were both one DOF hinge joints. The knee joint was modeled the same as in the 3D model. No toe joint was included in the 2D model.

Both models had 54 muscles including soleus (SOL), medial gastrocnemius (GAS), tibialis anterior (TA), tibialis posterior (TP) vastus medius (VAS), rectus femoris (RF), biceps femoris short head (BFSH), biceps femoris long head (BFLH), iliacus (IL), adductor magnus (ADD MAG), gluteus maximus (GMAX), gluteus medius (GMED) and other muscles. The equations of motion for each musculoskeletal model were derived using Simbody (open-source order-n dynamics engine under development at Simtk.org).

2.3. Simulation development

OpenSim was used to generate forward simulations using both models (Delp et al., 2007). First the models were scaled to subject-specific geometry based on the marker positions during the static trial. Inverse kinematics and residual reduction algorithms were applied in order to calculate the joint kinematics during a walking trial. Computed muscle control (Thelen and Anderson, 2006) was then used to find the optimal muscle excitation patterns that would drive the models along the desired trajectory (i.e. the experimental measurements). Both models had the same cost function to calculate muscle excitations, which was the minimization of weighted squared sum of muscle forces. Finally, forward simulations were generated based on the optimal excitation patterns (Delp et al., 2007; Thelen and Anderson, 2006).

For each subject, we generated one walking trial with both 2D and 3D models. Simulation results for all nine subjects were normalized and averaged to one complete gait cycle, starting from right heel strike.

2.4. Analysis methods

To analyze individual muscle function, we computed vertical COM accelerations induced by each muscle. The perturbation tool (Liu et al., 2006) adjusted one muscle's force (± 1 N) and simulated forward over a short time interval (0.03 s) to observe the resulting change in position of the model's COM. A foot-ground contact model with linear and torsional springs (Liu et al., 2006) was applied to account for ground reaction forces change during perturbation. A second-order differential equation (Liu et al., 2006) was then used to compute the COM accelerations induced by that particular muscle. The process was repeated for every muscle over the whole gait cycle. We determined that a muscle contributed to COM support when it provided positive vertical COM acceleration.

We chose a subset of three subjects (one 2D and 3D simulation per subject) for perturbation analysis and results were averaged over the gait cycle.

3. Results

3.1. Kinematics and kinetics

Our simulation results showed that the 2D model was able to reproduce almost the same joint kinematics (Fig. 1a) and kinetics (Fig. 1b) as the 3D model. Similar kinematics and kinetics

tracking results ensured that the comparison between 2D and 3D results was due to model DOFs only.

3.2. Muscle excitation patterns

Predicted muscle excitations in both models (Fig. 2) were generally consistent with EMG patterns in the literature (Perry, 1992; Winter, 2004) although there were some differences between the two models. The ankle muscles generally had similar excitation patterns in both 2D and 3D models (Fig. 2, SOL, GAS, TP and TA). The constraint of inversion movement decreased the activity of TP in the 2D model but TA had similar muscle excitations with slightly increased magnitude.

Only minor differences were observed in the activity of muscles which cross the knee joint. VAS and RF had almost identical muscle excitation patterns in 2D and 3D models while BFLH slightly changed the magnitude (Fig. 2).

For those muscles spanning the hip joint, we observed three types of altered muscle excitation patterns. First, GMED had large muscle excitation in the 3D model but was completely silent in the 2D model (Fig. 2, GMED). Second, some muscles (Fig. 2, GMAX and IL) had similar muscle excitations in both models. Finally, other muscles (e.g. Fig. 2, BFLH and ADD MAG) had different muscle excitation patterns between models, with excess mid-stance activity in the 2D model.

3.3. Muscle function analysis

In both 2D and 3D models, total vertical COM acceleration was the same over the whole gait cycle (Fig. 3, line c compared with d). However, with the 3D model, larger downward vertical acceleration induced by gravity was predicted than with the 2D model (Fig. 3, line f was lower than line e). Total muscle contribution to COM support in 3D was larger than that in 2D (Fig. 3, line a was higher than line b).

Both models predicted that COM support was provided by a number of major muscles. Immediately after heel strike (0–10% gait cycle), ipsilateral TA (Fig. 4, ADF) and contralateral ankle plantarflexors (not shown in Fig. 4) supported the COM upwards. During loading response (10–20% gait cycle), knee extensors and hip extensors (mostly VAS, GMAX and GMED in 3D, Fig. 4) provided the most support to COM. For the rest of stance phase (until around 65% gait cycle), ankle plantarflexors (Fig. 4, APF) were the main resource for COM support.

Finally, individual muscle contributions to COM support was different for some muscles. With the 2D model, we predicted less vertical acceleration of the COM from APF than in the 3D model (Fig. 5, SOL, GAS and TP) despite similar muscle excitations (Fig. 2, SOL, GAS and TP). VAS had the same calculated contribution to COM support in both models (Fig. 5, VAS). Muscles that cross the hip joint generally had different predicted function (Fig. 5, ADD MAG, GMED, GMAX, IL and BFLH). Specifically GMED was silent in 2D model but actively supported the COM in the 3D model prediction (Fig. 5, GMED). ADD MAG and BFLH induced opposite vertical COM accelerations in 2D and 3D models (Fig. 5, ADD MAG and BFLH).

4. Discussion

The goal of this study was to compare muscle excitations and contributions to COM support in 2D and 3D simulations of normal walking. We built 2D and 3D models in OpenSim and used both models to reproduce the same healthy normal walking data. We compared individual muscle excitation patterns and induced vertical acceleration of the COM by each muscle. Our

simulation results indicated that both models were able to reproduce the same joint kinematics and kinetics in the sagittal plane. Vertical acceleration of COM was almost the same in both models but individual muscle contributions differed.

Although human walking is a 3D activity (Saunders et al., 1953), there have been quite a number of simulation studies in the literature using models limited to the sagittal plane (e.g. Higginson et al., 2006; Neptune et al., 2001). Our results confirmed that a 2D model is capable of simulating the same kinematics and kinetics (Fig. 1) for normal walking at self-selected speed. With less DOFs included, the 2D model selects a different combination of muscle activities (or different muscle coordination patterns) to achieve the same walking trajectory. Our results indicated that although there was some variation in hip muscle excitations, the predicted excitation patterns for ankle and knee muscles were similar between 2D and 3D models (Fig. 2). Because the ankle and knee joints were modeled almost the same in both models, muscles that cross the ankle and knee joints do not have to change their excitations (and therefore muscle forces) to achieve the same joint moments. The hip joint, which had one DOF in 2D but three DOFs in 3D, had to adjust its muscle excitations accordingly (Fig. 2). GMED, for example, was turned off by the 2D optimization because (1) it did not generate any hip flexion/extension torque (despite contributions to sagittal movement which we will discuss later) and (2) the cost function was trying to minimize total muscle forces and would turn off any unnecessary muscles. GMAX and IL, which have moment arms mainly in the sagittal plane, could achieve similar muscle excitation patterns in the 2D model. Some other muscles (BFLH and ADD MAG) obtained very different excitations because their moment arms crossed into both sagittal and frontal planes.

Vertical COM acceleration was mostly provided by muscle forces and resistance due to gravity (Anderson and Pandy, 2003; Liu et al., 2006). Our results (Fig. 3) showed that muscle forces and gravity induced larger vertical COM acceleration in the 3D model but the effect was balancing such that the total COM acceleration was the same as the 2D model. Similar results were reported by Chen (2006) with greater power redistribution attributed to the joint moments in a model with more DOFs. As a result, calculated individual muscle contributions in the 3D model were generally larger than those in the 2D model (Fig. 4). However, the distribution of muscle contributions was similar. For example, both models suggested that knee extensors and hip extensors provided the majority of the COM support in loading response, while APF were the primary contributors to vertical COM acceleration in stance phase (Fig. 5).

Finally, we inspected induced vertical acceleration of the COM by individual muscles in both models. Although ankle muscles had very similar excitation patterns in both models, their contributions varied in magnitude. APF (GAS, SOL and TP) in the 3D model generated more vertical COM acceleration than the 2D model (Fig. 5) while contralateral TA in the 2D model provided larger contribution to COM support to compensate for the reduced action of APF. GMED only generated frontal plane joint torque but it contributed to COM support during early stance phase in our 3D prediction (Fig. 5, GMED), which has been reported in the literature (Anderson and Pandy, 2003; Liu et al., 2006). The loss of GMED might need compensation in the 2D model but from our results it was not clear which muscles were used. Most of the hip muscles contributed little support to COM and only IL slightly increased their COM support in the 2D model when GMED was supposed to be active (Fig. 5). Moreover, most of the hip muscles (e.g. ADD MAG, IL and BFLH) had quite different calculated induced acceleration in both models, indicating that the 2D model might not be able to adequately predict hip muscle function.

There were several limitations in our study. Firstly, we generated all our simulations in OpenSim. When comparing our kinematics and kinetics simulations with inverse dynamics results from Orthotrak (Motion Analysis Corp.), we did notice there were some discrepancies.

However, for this study, we believe it was more important to have both models converge to the same trajectory in order to compare muscle function difference due to model DOFs only. Secondly, the predicted muscle excitations might change if we used a different cost function. The cost function of minimization of squared total muscle forces required GMED in the 2D model to be zero. If another cost function was applied (e.g. minimization of error between muscle excitation and EMG), GMED might be turned on again even in a 2D model. However, the default cost function has been previously used with success for determination of muscle forces in normal gait (Crowninshield and Brand, 1981; Anderson and Pandy, 2001). Moreover, the silence of GMED (and other frontal plane muscles) was actually in agreement with previous 2D muscle-driven models (Neptune et al., 2001; Higginson et al., 2006) that did not have GMED included. Third, since the experimental data were recorded from a motorized treadmill, our interpretation of muscle function might be slightly different from overground walking. We believed the difference should be subtle (Riley et al., 2007) and we tried to focus on the impact of model DOFs.

A number of investigators have been using modeling and simulation methods to understand how individual muscles or joint moments contribute to the motion of body segments and joints during dynamic activities. While the complexity of their models varied, different results were reported and it was not clear how the model assumptions affect the interpretation of muscle function. In this study, we looked at the model-predicted muscle activities (i.e. muscle excitation and function) when changing the number of DOFs. We showed that 2D and 3D models could converge to the same sagittal plane joint kinematics and kinetics patterns. However, changing the model DOFs would induce a redistribution of some muscles' excitation patterns and function. We noticed that ankle and knee muscles could have similar predicted excitations and individual contributions to vertical COM acceleration, with slightly different magnitude. Muscles that cross the hip joint, which was modeled quite differently in the 2D and 3D models had the largest variation in predicted excitations and function. While previous studies showed that changing DOFs would impact the interpretation of the role of joint moments during walking, this study reports how predicted individual muscle function would change due to model DOFs changes. Future studies will address changes in muscle contributions to joint motion and muscle function in pathological gait.

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Fig. 1. (a) Kinematics (joint flexion angles) and (b) kinetics (joint moments) tracking results of both 2D (dashed line) and 3D (solid line) models. Gait cycle was normalized to 100% starting from heel strike. Data was averaged over nine subjects with one gait cycle each.

Fig. 2.

Major muscles' excitation patterns in 2D (dashed line) and 3D (solid line) models. Muscle excitations were generally consistent with EMG signals. Ankle muscles (SOL, GAS, and TA) and knee muscles (VAS, BFSH, and RF) had similar excitations in both 2D and 3D models. Muscles that span the hip joint (GMED, ADD MAG and BFLH) changed their excitation patterns between both models. Data was averaged over nine subjects with one gait cycle each.

Fig. 3. Vertical COM acceleration comparison between 2D (dashed lines) and 3D (solid lines) model. Data was averaged over three subjects with one gait cycle each. Total vertical COM acceleration (lines c and d) was decomposed into muscle contribution (lines a and b) and gravity (lines e and f) in each model.

Fig. 4. Vertical COM acceleration induced by major muscle groups in 2D (a, dashed lines) and 3D (b, solid lines) models. Data was averaged over three subjects with one gait cycle each. ADF: ankle dorsiflexor TA. APF: ankle plantarflexors (TP, SOL and GAS). Knee and hip extensors included VAS, RF, GMED and GMAX. Bold lines indicate vertical COM acceleration induced by all muscles. Toe off at 65% gait cycle.

Fig. 5. Individual muscle contributions to COM support predicted for 2D (dashed lines) and 3D (solid lines) models. Data was averaged over three subjects with one gait cycle each. Notice that vertical axis is scaled differently for each muscle. See text for details.

Table 1
Model configuration comparison between 2D and 3D models

	No. of segments	No. of joints	No. of muscles	Total DOF	Torso DOF	Pelvis DOF	Hip DOF	Knee DOF	Ankle DOF	Toe DOF
3D	13	12	54	23	3	6	3	1	2	1
2D	13	12	54	10	1	3	1	1	1	0

Hip, knee, ankle and toe DOFs represent only one leg.