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How robust is human gait to muscle weakness?☆

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ABSTRACT

Humans have a remarkable capacity to perform complex movements requiring agility, timing, and strength. Disuse, aging, and disease can lead to a loss of muscle strength, which frequently limits the performance of motor tasks. It is unknown, however, how much weakness can be tolerated before normal daily activities become impaired. This study examines the extent to which lower limb muscles can be weakened before normal walking is affected. We developed muscle-driven simulations of normal walking and then progressively weakened all major muscle groups, one at the time and simultaneously, to evaluate how much weakness could be tolerated before execution of normal gait became impossible. We further examined the compensations that arose as a result of weakening muscles. Our simulations revealed that normal walking is remarkably robust to weakness of some muscles but sensitive to weakness of others. Gait appears most robust to weakness of hip and knee extensors, which can tolerate weakness well and without a substantial increase in muscle stress. In contrast, gait is most sensitive to weakness of plantarflexors, hip abductors, and hip flexors. Weakness of individual muscles results in increased activation of the weak muscle, and in compensatory activation of other muscles. These compensations are generally inefficient, and generate unbalanced joint moments that require compensatory activation in yet other muscles. As a result, total muscle activation increases with weakness as does the cost of walking. By clarifying which muscles are critical to maintaining normal gait, our results provide important insights for developing therapies to prevent or improve gait pathology.

1. Introduction

Muscle strength is important in many daily-life activities. Our muscles provide forces that allow us to walk, climb stairs, and perform athletic activities. With training, humans can even increase their performance and run faster, jump higher, or lift more weight. Disuse, aging, and disease can lead to a loss of muscle strength, which may limit the performance of activities. When muscles become excessively weak, daily-life activities such as walking may become impaired. Indeed, muscle strength measures have been shown to be strongly correlated to functional gait measures, for example in cerebral palsy [1,2] and stroke [3]. This suggests that muscle strength is an important prerequisite for walking performance.

Just how much weakness can be tolerated before normal daily activities such as walking become impaired is unknown. This capacity to tolerate weakness may differ between muscles: gait performance may be sensitive to weakness of some muscles and robust to weakness of others. Determining how much weakness can be tolerated and which weak muscles are most likely to limit gait may improve the ability to design successful strength training programs. Furthermore, knowing which muscles can tolerate weakness is important when considering treatments that may weaken muscles, such as muscle-tendon surgery or botulinum toxin treatment.

The purpose of this study was to investigate to what extent individual muscles can tolerate weakness before gait is impaired. We sought to answer the following questions:

- To what extent can generalized muscle weakness be tolerated before gait is impaired?
- Does the amount of weakness that can be tolerated differ between muscles?
- What compensation strategies are used when individual muscles get weaker?
- What is the additional cost for these compensation strategies?

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To answer these questions, we simulated the normal gait pattern of typical, able-bodied subjects, using a generic musculoskeletal model with average muscle strength. We then progressively weakened each of the major leg muscles and muscle groups in the model, and repeated the simulation. We evaluated how much weakness could be tolerated while maintaining normal gait, and what compensations occurred.

2. Methods

Gait analysis data of six healthy adolescent subjects (three male, three female) were selected from a normal pediatric dataset [4]. Ethical approval, parental consent, and subject assent had been obtained prior to data collection. We selected older and bigger subjects to limit the amount of model scaling necessary. All subjects were 15 years of age or older (mean ± *SD*: 16 ± 1 y), weighed 60 kg or more (68 ± 5 kg), and were 1.60 m or taller (175 ± 9 cm).

All subjects underwent standard three-dimensional gait analysis at free speed (1.08 ± 0.16 m/s). Details of the data collection and analysis procedures are described by Schwartz et al. [4]. In short, motion data was collected using a 12-camera Vicon MX system (Vicon, Oxford, UK) operating at 120 Hz. The standard Vicon Plug-in-Gait marker set was used, with some additional technical markers on the thighs and shanks. In addition, a trunk model was used, consisting of a marker over the 7th cervical spinous process (C7), and symmetric markers approximately 2.5 cm to the left and the right of the sternal notch. Functional hip joint centers and knee axes of rotations were determined using methods described by Schwartz and Rozumalski [5]. Ground reaction forces were recorded using four force plates (AMTI, Watertown, MA) sampled at 1080 Hz. Consecutive force plate strikes on all four force plates were acquired.

We generated simulations of one representative stride for each subject using OpenSim software [6]. We used a generic musculoskeletal model (Fig. 1A) [7,8] with 23 degrees of freedom and 92 muscle-tendon actuators. The generic model was scaled to the individual subject sizes, using the anatomical landmarks and functional joint centers as a reference (Fig. 1C, Step 1). The subjects' gait patterns were reproduced by the scaled model, using an inverse kinematic analysis tracking individual marker trajectories (Step 2). The full stride plus 100 ms before and 100 ms after was simulated to guarantee continuity in muscle activations. A residual reduction algorithm [6] was used to resolve dynamic inconsistency between the measured ground reaction forces and the model's kinematics (Step 3). Computed muscle control (CMC) [9] was used to calculate the optimal muscle activation pattern that generated the necessary joint moments to produce the measured kinematics (Step 4). No constraints on muscle activations were applied, so the model was free to adopt any activation pattern in response to the imposed muscle weakness (described below). Calculated muscle activations were generally consistent with experimental EMG patterns (Fig. 2) and in line with literature.

After the full-strength simulations were complete, we progressively weakened all major leg muscle groups and generated new simulations with the resulting weak models for each of the six subjects (Step 5). The weakening was imposed in two ways. First, we weakened all muscles in the model simultaneously to study the effect of generalized weakness. Second, we evaluated the effect of weakness of the following individual muscles and muscle groups,

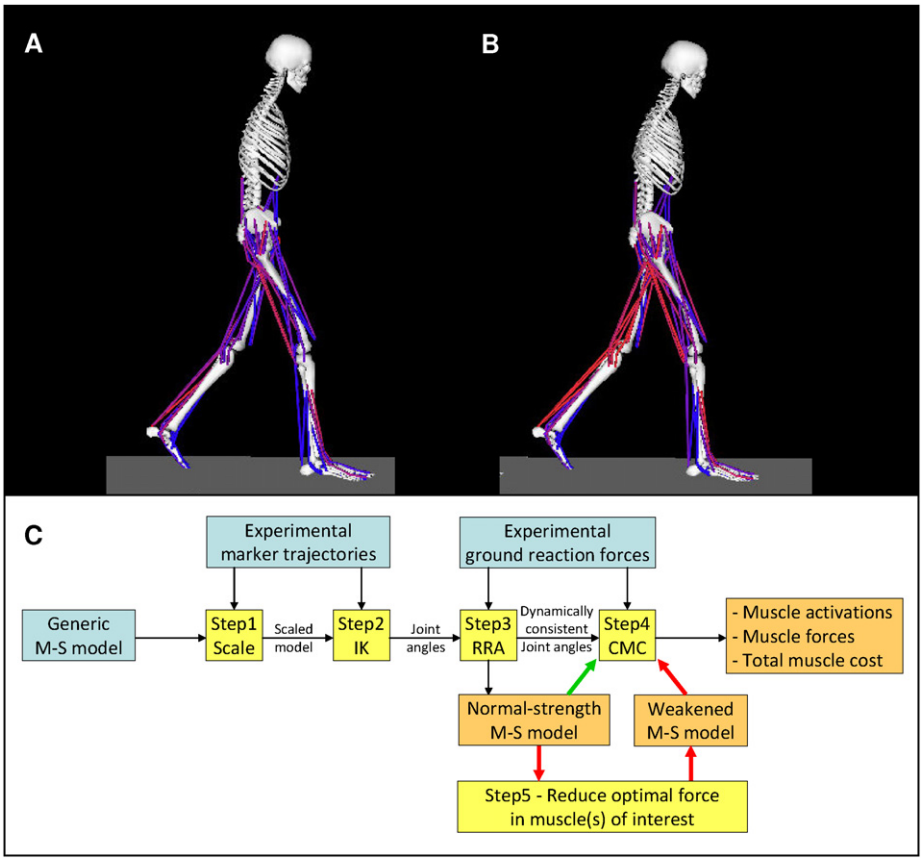

Fig. 1. (A) Graphical illustration of muscle activation during a step in the generic model with normal muscle strength and (B) in the same model but with 40% weakened muscles. The muscle colors represent the level of activation on a scale from dark blue (no activation) to bright red (full activation). (C) Steps for generating simulations using normal-strength (➡) and weak (➡) models. In blue: inputs to the simulation; in yellow: analysis steps; in orange: outcomes of the simulation. M-S: musculoskeletal, IK: inverse kinematics, RRA: residual reduction algorithm, CMC: computed muscle control. See text for details. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

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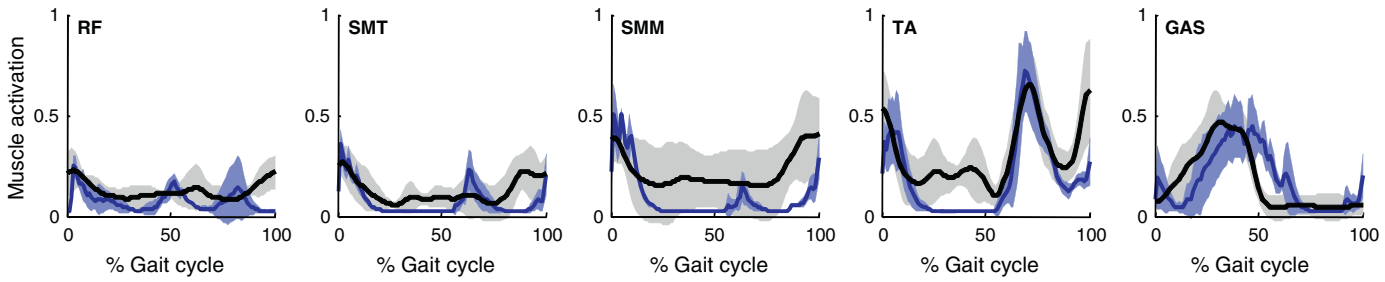

Fig. 2. Simulated muscle activations (blue lines) and experimental EMG (black lines), for all five muscles of which EMG was collected. Shaded areas show standard deviations. Experimental EMG data are rectified and bi-directionally low-pass filtered at 6 Hz, and their peak value is normalized to the peak value of the simulated muscle activation per subject. All data are averages over 5 subjects, one subject had to be excluded because of artifacts in the EMG. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

by weakening them one at the time: gluteus maximus; gluteus medius; iliopsoas (iliacus and psoas); hamstrings (semitendinosus, semimembranosus, and biceps femoris long head); rectus femoris; vasti (vastus medialis, lateralis, and intermedius); tibialis anterior; plantarflexors (gastrocnemius medialis, lateralis, and soleus combined); gastrocnemius (medialis and lateralis combined); and soleus. These muscles were chosen as they are the main leg muscle groups or prone to weakness. For all simulations, we decreased maximum force of the muscles in 20% decrements, with 100% weakness indicating no remaining muscle force. This was done by altering the maximum isometric force parameter in the muscle model, while leaving all other parameters constant. Since tendon stiffness and passive muscle stiffness scale to maximum muscle force, these parameters also decreased with weakness. This was considered to be realistic, since tendon stiffness and passive muscle stiffness generally adapt to muscle strength changes, e.g. due to training or age [10,11]. We then re-ran the CMC algorithm, computing optimal muscle control required to track normal gait kinematics in the presence of weakened muscle properties.

For all simulations with weakened models, we checked whether normal gait kinematics could still be tracked. Unsuccessful tracking was defined as either (1) no solution found; or (2) a deviation in any joint angle from the baseline simulation (normal muscle strength) of more than one degree; or (3) the use of reserve

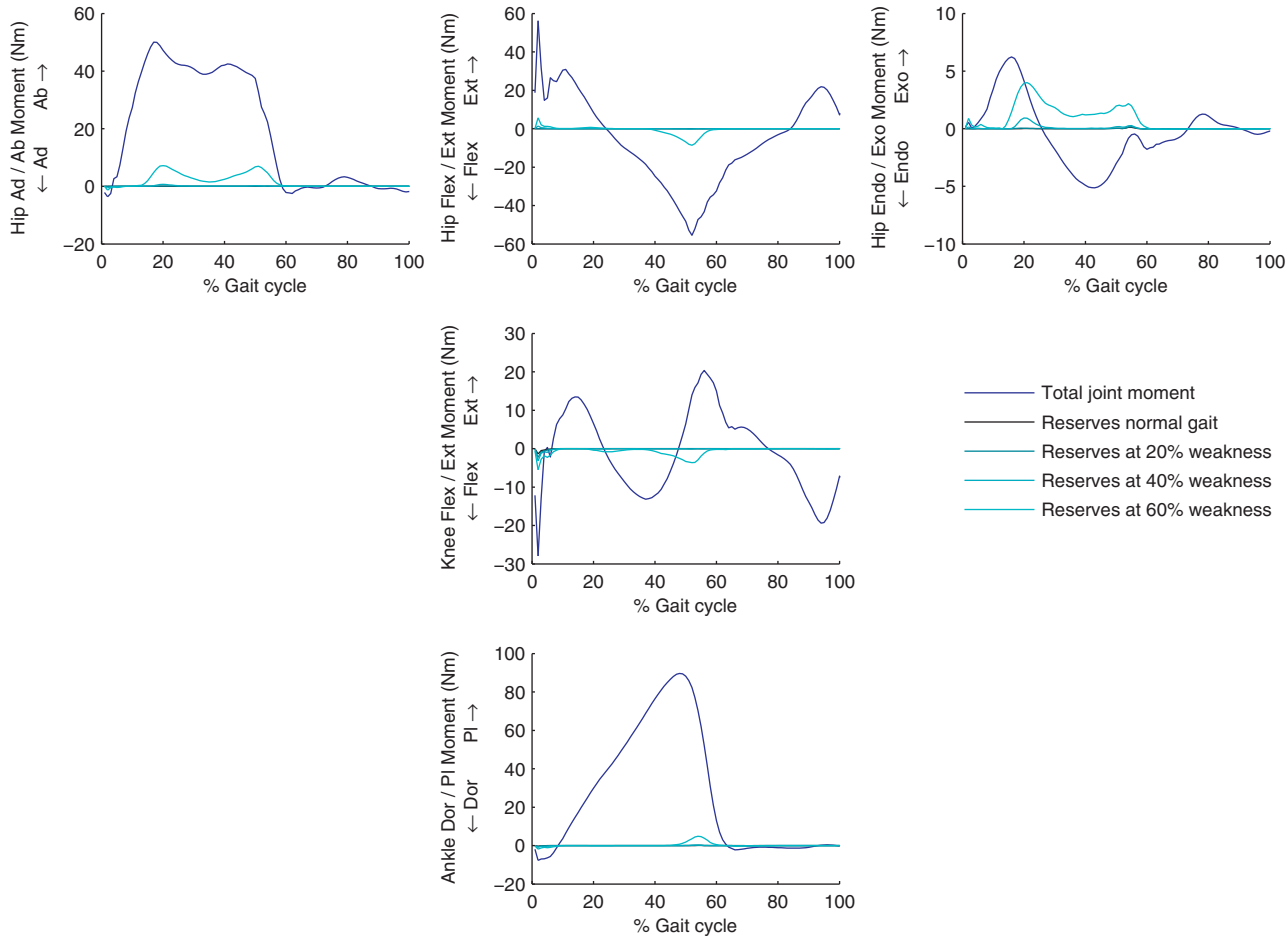

Fig. 3. Total joint moments and reserve joint actuator moments as a function of the gait cycle, at varying levels of generalized muscle weakness. Each line represents the average over 6 subjects. The presence of reserve actuators indicates the inability of muscles to deliver enough joint moment to produce normal gait. Reserves during normal gait and at lower % weakness are underlying at the zero line.

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actuators on any joint of more than 5% of peak joint moment, with a minimum of 1 N m. These reserve actuators generate small joint moments in normal simulations only to account for large joint angular accelerations. Since they are highly penalized by the cost function, their contribution normally is low and negligible. A contribution of >5% of overall joint moments indicates a failure of muscles to generate sufficient joint moments.

If normal gait kinematics could be successfully tracked, we then evaluated the resulting muscle forces, muscle activations, and muscle cost. Muscle cost was quantified for each muscle for each time step, as:

$$\text{Muscle cost} = \left(\frac{F_{\text{mus}}}{F_{\max}(l, v)}\right)^2$$

where F_{mus} is the muscle force, and $F_{\max}(l, v)$ is the instantaneous maximum muscle force taking into account the instantaneous length and velocity of the muscle fibers. Total muscle cost was calculated as the sum of all individual muscle costs integrated over time. If normal kinematics could not be tracked successfully, then the resulting reserve actuators were analyzed to see in what manner the gait pattern was first impaired.

3. Results

Generalized muscle weakness could be tolerated up to 40% decrease in strength. That is, gait was impaired (i.e. normal gait could not be simulated) when greater than 40% generalized muscle weakness was imposed (Fig. 3). With 40% weakness, most subjects could walk without problems, but in two out of six subjects the hip exorotation moment started to be insufficient at some instant during the gait cycle. With 60% weakness, both hip exorotation and hip abduction moments were insufficient during almost the entire stance phase, and hip flexion, knee flexion, and ankle plantar flexion moments were insufficient during pre-swing (Fig. 3).

The amount of weakness that could be tolerated for individual muscles differed between muscles/muscle groups. Gait was more sensitive to weakness of the hip abductors and ankle plantarflexors than to weakness in other muscle groups. For most muscles, the muscle could be removed and normal gait was still possible, albeit at a higher cost. However, normal gait was not possible without gluteus medius or without plantarflexors in any of the subjects. In some subjects, gait was impaired already at 60% weakness (one subject for gluteus medius) or at 80% weakness (two subjects for gluteus medius and three subjects for plantarflexors). Gait was also

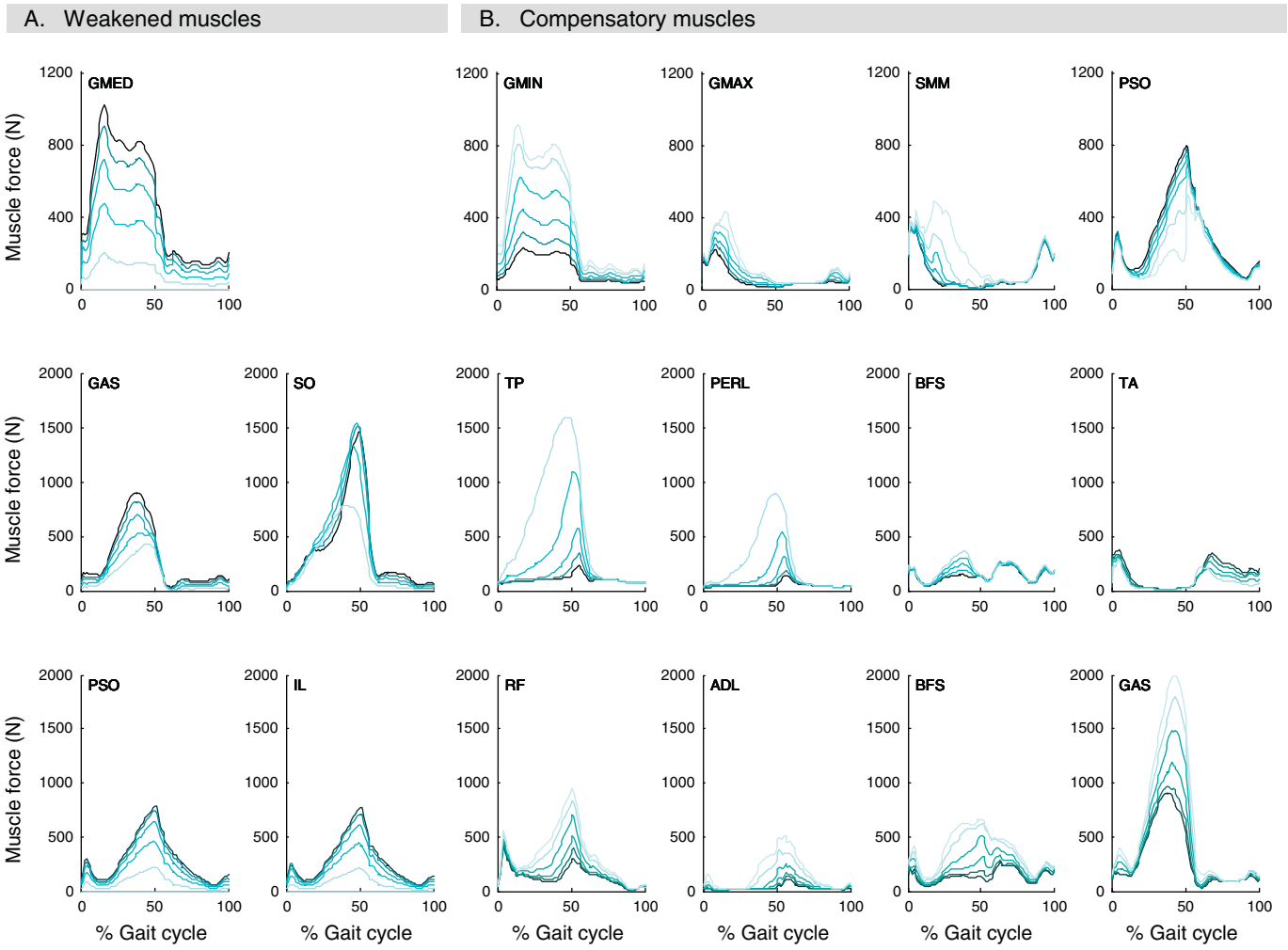

Fig. 4. Examples of the effect of local muscle weakness on muscle forces during gait. Top row: GMED weakness; middle row: plantarflexor (GAS + SO) weakness; bottom row: iliopsoas (PSO + IL) weakness. Lighter colors give strength values at increasing levels of weakness, from black = normal force (0% weakness) to light blue = total strength loss (100% weakness). 100% weakness was not possible for plantarflexors. Each line represents the average over 6 subjects. *Abbreviations*: GMED, gluteus medius; GMIN, gluteus minimus; GMAX, gluteus maximus; TFL, tensor fascia lata; SMM, semimembranosus; PSO, psoas; GAS, gastrocnemius; SO, soleus; TP, tibialis posterior; PERL, peroneus longus; BFS, biceps femoris short head; TA, tibialis anterior; IL, iliacus; RF, rectus femoris; ADL, adductor longus. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

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Table 1
Compensations that occur with weakness of different muscles/muscle groups.

Weak muscle/muscle group	Compensations		
	Activation of the weak muscle/muscle group itself	Muscles with increased activation	Muscles with decreased activation
GMAX	Down	GMED3 (GMED2 GMIN3 HAM ADD VAS QF PIRI)	
GMED	Up	GMIN SMM TFL BFS SAR GMAX1 GAS RF (VAS)	PSO GMAX2,3 SO
ILPS	Up	GMED1 GMIN SAR BFS TFL PEC GRA RF GAS TA	GMED2,3 SO
HAM	Up; but down for BFS in stance	SAR GRA ADD GMAX GAS TA PERT EXTD	ILPS (sw) PERL
RF	Down in stance; up in pre-swing	ILPS VAS SO TFL (GMED2,3)	SMT BFS GAS GRAC TA
VAS	Up	ADD GMAX GMIN1	HAM (sw) ILPS (sw)
TA	Equal up to 60% weakness, then down	EXTD PERT	None
PLFL	Up	TIBP FLD FLH PERB PERL BFS SMT SMM (GMED3)	TA EXTD
GAS	Equal up to 60% weakness, then down	SO BFS SMT SMM ILPS (GMED GMIN SAR)	TA
SO	Equal	GAS TIBP FLD FLH PERB PERL VAS RF	TA EXTD BFS ILPS SAR (GMIN)
ALL	Up in all muscles		

Muscles in (brackets) only have minor contributions. Lower numbers (GMAX1, GMED1, etc.) indicate more ventral parts, higher numbers more dorsal parts of the muscle. *Abbreviations*: GMAX: gluteus maximus, GMED: gluteus medius, ILPS: iliopsoas, HAM: hamstrings (semitendinosus, semimembranosus and biceps femoris long head), RF: rectus femoris, VAS: vasti (vastus medialis, lateralis, and intermedius), TA: tibialis anterior, PLFL: plantarflexors (gastrocnemius medialis, lateralis, and soleus combined), GAS: gastrocnemius (medialis and lateralis combined), SO: soleus, GMIN: gluteus minimus, QF: quadratus femoris, PIRI: piriformis, SMM: semimembranosus, TFL: tensor fascia lata, BFS: biceps femoris short head, SAR: sartorius, PEC: pectineus, GRA: gracilis, ADD: adductors, PERT: peroneus tertius, EXTD: extensor digitorum longus, TIBP: tibialis posterior, FLD: flexor digitorum longus, FLH: flexor hallucis longus, PERB: peroneus brevis, PERL: peroneus longus, SMT: semitendinosus, PSO: psoas, (sw): only in swing.

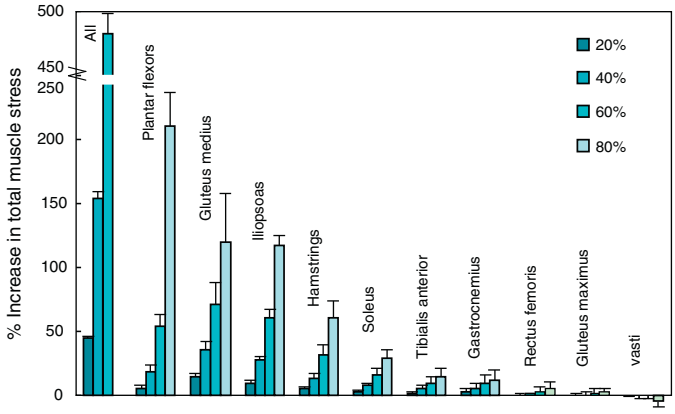

Fig. 5. Increase in total muscle cost with increasing levels of weakness (20–80% strength loss) for all evaluated muscle groups.

sensitive to weakness of hamstrings and iliopsoas: normal gait was not possible without these muscles in five and four subjects respectively.

Compensations for weakness of individual muscles included activating the weak muscle more and/or activating other muscles. When all muscles were weakened simultaneously, the activation increased in all muscles (Fig. 1B). When individual muscles were weakened, these muscles generally produced less force, as shown in Fig. 4A for a selection of weak muscles/muscle groups. Other muscles compensated by increasing their force (Fig. 4B, Table 1). The observed compensations were generally inefficient, because they generated unbalanced joint moments that required compensatory activation in other muscles. As a result, weakness of individual muscles led to changes in activation pattern and forces of many other muscles throughout the leg (Table 1, Fig. 4). Muscle force changes for all weakened muscle groups and their compensations are shown in an electronic appendix to this paper.

The compensations that occurred with muscle weakness led to increased muscle cost. Total muscle cost, summed over the gait cycle, increased with the severity of weakness and showed a significant variability depending on the muscle being weakened (Fig. 5). Total muscle cost increased most with weakness of plantarflexors, gluteus medius, and iliopsoas. Little change in total muscle cost was seen with weakness in the gluteus maximus and vasti.

4. Discussion

We developed a novel paradigm to evaluate the robustness of our musculoskeletal system to weakness. In a 'failure analysis', we progressively decreased the maximum force of muscles, and analyzed when and where 'things went wrong'. Hence the amount of weakness that could be tolerated reflects the overcapacity, or the factor of safety, of our musculoskeletal system to weakness, and how this differs between muscles. Our simulations revealed that normal gait was not possible with more than a 40% loss of strength. Most individual muscles/muscle groups could be weakened by approximately 80%, or could even be totally removed, while still allowing normal gait kinematics. Weakness of individual muscles was compensated by activating the weak muscle more and/or by activating other muscles. However, these compensations led to increases in muscle cost. Weakness of plantarflexors, hip abductors, and hip flexors affected the gait most, since weakness in these muscles could be tolerated only to a limited extent, and resulted in a large increase in total muscle cost. Gait appeared most robust to weakness of hip and knee extensors, which could tolerate weakness well and without a substantial increase in total muscle cost.

How much weakness can be tolerated clearly depends on the original strength of the model. We used initial strength values for all subjects as present in the generic model developed by Delp et al. [7], which are based on cadaver measurements and population averages. These values differ slightly from other models [12,13] and may not be perfectly representative for all populations. However, the differences between models are small compared to the large effects found in this study. Furthermore, our results are in line with relative joint loads during walking. If we compare the joint moments during walking in our subjects calculated from inverse dynamics to experimental strength measurements in similar groups [14,15], it is again the plantarflexors, hip abductors, and hip flexors that stand out as performing at the highest percentage of their capacity. For instance, peak ankle moment during gait in our subjects was 91 N m, while average strength in young adults is 130 N m [15], leading to a relative percentage of 70%. Similarly, peak hip abductor moment during gait in our subjects was approximately 52 N m, while average strength in the similar weight group of Eek et al. [14] was 129, leading to a relative use of 40%. Furthermore, the hip abduction moment is high over almost the entire stance phase. For hip flexors the relative peak load was 71%, although strength was measured in 90° flexion.

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Similar calculation for hip and knee extensors compared with normal strength [14] resulted in a relative use of only 23 and 20%, respectively. This comparison strengthens our finding that the 'safety factor' for plantarflexors, hip abductors, and hip flexors is much lower than for knee and hip extensors. Recent studies by Eek et al. [16] and Dallmeijer et al. [17] in healthy children and Requiao et al. [18] in adults also show that joint moments during gait when compared to maximum muscle strength are highest for the ankle plantar flexors. In these studies, relative hip abductor moment was not excessively high [16,17], but maintained for a long part of the gait cycle.

It may be surprising that total muscle cost did not increase at all with weakness of the vasti, and only very slightly with weakness of gluteus maximus. In these cases different compensations were seen throughout the gait cycle. For the vasti, a (small) increase in total muscle activation was seen during the first half of stance, in order to generate normal knee extension moments despite weakness. However, during pre-swing and initial swing, the passive force generated by the vasti decreased with weakness, reducing the muscle activity needed by knee flexors to compensate for this. A similar effect was seen in the gluteus maximus in terminal swing. These high passive forces in the vasti are a known limitation in the present model [12], and therefore this effect might by somewhat overestimated. When excluding the positive effects of weakness on passive forces in gluteus maximus and vasti, the effects of weakness in these muscles were still relatively small.

The increase in total muscle cost with weakness denotes an increased load on muscles which may lead to fatigue and damage of muscles. This may cause further weakening, and the prospect of a downward spiral of weakness and compensations. We used muscle stress squared as a measure of total muscle cost, and this same value was used in the force-sharing criterion to distribute joint moments over muscles. This total muscle stress squared expresses the load on muscles but is not a direct measure of metabolic cost. Therefore, other more economically optimal compensation strategies may be used by subjects besides those predicted by the model, and the choice of force-sharing criterion may have influenced our results. However, when the model is challenged to its maximum, as was the case with increasing muscle weakness in our simulations, the force-sharing problem becomes less relevant. In this case all suitable muscles are turned on to find a solution at all, independent of the optimization criterion used.

The muscles that were most vulnerable to weakness were generally important muscles during gait. Calf muscles are important for support and forward progression, iliopsoas is important for progressing the leg into swing, and gluteus medius is important for vertical support [19,20]. However, whether weakness could be tolerated was not only dependent on the muscle's importance for gait. Rather, the muscle's relative load during gait compared to its maximum capacity, as well as the availability of compensatory muscles, defined whether weakness could be tolerated or not.

Compensations for weakness were seen in many different muscles throughout the leg, which was sometimes non-intuitive. For example, weakness of the iliopsoas led to increased activation of the rectus femoris to assist in hip flexion, which necessitated increased activation of knee flexors such as biceps femoris short head and gastrocnemius. These findings indicate that when increased or decreased activation is seen in muscles during clinical gait analysis, the cause may be in muscle weakness elsewhere in the system, which may not always be clear at first view. The electronic appendix shows more examples of the effects of weakness and their compensations for all evaluated muscle groups.

When weakness is severe, patients may choose to alter their gait pattern, rather than to walk with normal kinematics using the compensations as found in this study. There is likely to be some threshold (or a variety of thresholds) of muscle stress beyond which kinematic compensations become more favorable than force generation compensations that maintain normal kinematics. Our simulations give insight to what extent normal kinematics are possible and at what cost, and where the first effects of weakness are likely to appear. For example, patients with abductor weakness are known to show excessive ipsilateral lateroflexion of the trunk during stance to reduce hip abductor moment. Further research could show whether and how such pathological gait patterns are an effective way to reduce muscle stress.

Our results may have important implications for designing strength training programs. Since patients can only perform and tolerate a certain amount of strength training, it is best to focus that effort where it can have the biggest payoff. We found that tolerance of weakness differs considerably between muscles. Since weakness is not well tolerated in the plantarflexors, hip abductors, and hip flexors, it may be most useful to target these muscles with strength training programs. In contrast, strength training of hip or knee extensors, although commonly performed [21], may merely enhance their already significant excess strength capacity. Studying the effect of these different strength training programs in patients could show whether our simulation results are indeed applicable in clinical practice. It should be noted that the effects of weakness may be different in pathological gait patterns than in normal gait. For example, a crouch gait pattern may pose a larger burden on the quadriceps, and therefore quadriceps muscle strength may be more important to maintain such a gait pattern compared to a normal gait pattern, to prevent further collapse of the knee during stance. More generally, the optimal strength training program may be dependent on the specific gait pattern of the patient and the accompanying relative loads on muscles. Future study should focus on the relative load on muscles in various patient populations and pathological gait patterns.

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Conflict of interest statement

None of the authors have a conflict of interest regarding the contents of this manuscript.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.gaitpost.2012.01.017.

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