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published in
Medicine and Science in Sports and Exercise
2011

DOI (link to publisher)
10.1249/MSS.0b013e3181fa7738

document version
Publisher's PDF, also known as Version of record

Effects of Fatigue of Plantarflexors on Control and Performance in Vertical Jumping

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ABSTRACT

BOBBERT, M. F., M. M. VAN DER KROGT, H. VAN DOORN, and C. J. DE RUITER. Effects of Fatigue of Plantarflexors on Control and Performance in Vertical Jumping. *Med. Sci. Sports Exerc.*, Vol. 43, No. 4, pp. 673–684, 2011. **Introduction**: We investigated the effects of a mismatch between control and musculoskeletal properties on performance in vertical jumping. **Methods**: Six subjects performed maximum-effort vertical squat jumps before (REF) and after the plantarflexors of the right leg had been fatigued (FAT) while kinematic data, ground reaction forces, and EMG of leg muscles were collected. Inverse dynamics was used to calculate the net work at joints, and EMG was rectified and smoothed to obtain the smoothed rectified EMG (SREMG). The jumps of the subjects were also simulated with a musculoskeletal model comprising seven body segments and 12 Hill-type muscles, and having as only input muscle stimulation. **Results**: Jump height was approximately 6 cm less in FAT jumps than in REF jumps. In FAT jumps, peak SREMG level was reduced by more than 35% in the right plantarflexors and by approximately 20% in the right hamstrings but not in any other muscles. In FAT jumps, the net joint work was reduced not only at the right ankle (by 70%) but also at the right hip (by 40%). Because the right hip was not spanned by fatigued muscles and the reduction in SREMG of the right hamstrings was relatively small, this indicated that the reduction in performance was partly due to a mismatch between control and musculoskeletal properties. The differences between REF and FAT jumps of the subjects were confirmed and explained by the simulation model. Reoptimization of control for the FAT model caused performance to be partly restored by approximately 2.5 cm. **Conclusion**: The reduction in performance in FAT jumps was partly due to a mismatch between control and musculoskeletal properties. **Key Words:** MUSCLE WORK, COORDINATION, OPTIMAL CONTROL, SIMULATION MODEL

It is often stated that control is of paramount importance for good performance in motor tasks. Control, also called coordination, may be operationalized as the stimulation of muscles as a function of time, which ultimately determines the resulting movement. Studies of human vertical jumping with forward dynamic simulation models of the musculoskeletal system have shown the obvious: the maximum jump height that can theoretically be realized is determined by the properties of the musculoskeletal system, and the extent to which this maximum jump height is approached is determined by control (e.g., see Bobbert and van Soest (6)). Achieving the maximum jump height requires optimization, that is, tuning of control to the musculoskeletal properties. It has previously been shown in a simulation study that a mismatch between control and musculoskeletal properties leads to an unbalanced increase in segment angular velocities, and this causes the shortening velocity of some muscles to be disproportionally high and the total work produced to be unnecessarily small (7).

The relationship between control and performance in jumping can easily be studied in simulation models but not so easily in human subjects because subjects are unable to manipulate their muscle stimulation at will during jumping. However, the effects of a mismatch between control and musculoskeletal properties may be investigated by changing muscle properties, and a straightforward method to change muscle properties is to induce muscle fatigue. Studies of the effect of muscle fatigue on control and performance may provide insight not only in the interplay between control, muscle properties, and performance but also in the potentially injurious effects of muscle fatigue during motor tasks in their own right. Bonnard et al. (8) have shown that subjects are able to maintain global power output in prolonged submaximal hopping despite development of fatigue in the plantarflexors by landing with more flexed knees and shifting load from the plantarflexors to the knee extensors (8). It has been speculated that alterations in landing strategies after development of fatigue may increase the risk of injury to, for example, the anterior cruciate ligament (e.g., see McLean

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Submitted for publication April 2010.
Accepted for publication August 2010.

0195-9131/11/4304-0673/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/MSS.0b013e3181fa7738

and Samorezov (15) and Thomas et al. (23)). This has inspired researchers to study the effects of selective fatigue of specific muscle groups (e.g., plantarflexors) on single-leg landing mechanics after a forward jump (e.g., see Thomas et al. (23)) and landing mechanics in running (14). Rodacki and coworkers conducted two studies of the effects of muscle fatigue on the push-off in vertical jumping; in the first study (19), all leg extensor muscles were fatigued by having subjects perform many vertical jumps, and in the second study (20), fatigue was induced selectively in either the knee extensors or the knee flexors. The authors provided a very thorough account of the effect of fatigue on jump height, kinematics, kinetics and EMG activity, but did not attempt to distinguish between effects that were due to the reduced mechanical output of the fatigued muscles and effects that were due to the presumed mismatch between control and musculoskeletal properties (20). The purpose of the present study was to try and make this distinction.

In this study, we had human subjects perform maximum-effort vertical squat jumps and studied the effects of selective fatigue of the plantarflexors of their right leg, induced by repeated maximum voluntary isometric contractions (MVC) on a dynamometer and subsequently maintained with a pressurized cuff around the right lower leg. The reason for choosing the plantarflexors as the muscles to be selectively fatigued was that the output of these muscles is considered to help prevent the angular velocities of the hip and knee joints and the shortening velocities of the hip and knee extensors from becoming very large and the mechanical output from becoming very low (7). We chose to induce fatigue in only one leg and study the effect on two-legged jumps because this allowed us to answer our main research question in a well-controlled and safe experimental setting. One alternative would have been to study one-legged jumping, but this was discarded because in most subjects, control of one-legged jumping is poor compared with control of two-legged jumping. The other alternative would have been to induce fatigue in the plantarflexors of both legs and to study two-legged jumping, but this was discarded after discovering in preliminary experiments that with bilateral fatigue of the plantarflexors, subjects had difficulty not only in pushing off but also in landing, which raised safety concerns. Hence, we induced fatigue in only one leg and studied the effect on two-legged jumps. In our study, we did not give the subjects the opportunity to practice jumping with their fatigued calf muscles, and we assumed that no adaptations in control would occur. To check this assumption, we recorded EMG and analyzed its timing and amplitude.

On the assumption that a mismatch between control and musculoskeletal properties is achieved by fatiguing the plantarflexors of the right leg, two hypotheses may be formulated. The first hypothesis is that loss of work of the fatigued plantarflexors also leads to loss of work of other, unfatigued muscles. The second hypothesis is that the loss in total work with fatigue of the right plantarflexors is unnecessarily high and can be partly remedied by reoptimization of control. The first hypothesis was tested in the subjects by studying the effect of the induced plantarflexor fatigue on the net work produced at the hip joint of the right leg, which is not spanned by the fatigued muscles. To test the second hypothesis, we simulated the jumps of the subjects in the unfatigued and fatigued conditions with a forward simulation model of the human musculoskeletal system. The simulation approach allowed us to analyze and explain the effects of fatigue of the plantarflexors of the right leg in terms of work of individual muscles and to investigate to which extent the loss of performance could be remedied by reoptimization of control.

MATERIALS AND METHODS

Outline of experimental procedures. Six physically active subjects (four men and two women), all actively engaged in various sports and experienced in jumping, participated in this study. Informed consent was obtained from all subjects in accordance with the policy statement of the American College of Sports Medicine, and the experiment received approval of the local ethics committee. Characteristics of the group of subjects (mean ± SD) were as follows: age = 26 ± 8 yr, body mass = 68.5 ± 5.9 kg, and height = 1.73 ± 0.08 m.

The protocol used for the experiments is schematically shown in Figure 1A. Each subject first performed five MVC with the right leg on a custom-built dynamometer. The contractions lasted 3 s and were interspersed by 2 min of rest. The plantarflexion moment was measured during the contractions, and the peak plantarflexion moment was selected to be used as reference. Second, the subjects were asked to perform practice jumps and find a preferred semi-squatted initial posture for maximum height vertical jumping without countermovement. The height of the hip in the preferred posture was measured and indicated on a yardstick, and in all subsequent jumps, the subjects were instructed to match this height in the initial posture. Subsequently, the subjects performed three maximum height squat jumps from the preferred initial posture, with approximately 5 s between consecutive jumps (PRE). A cuff (Erkameter 3000 with aneroid sphygmomanometer) was then applied to the lower leg. After 5 min of rest, the cuff was pressurized to more than 200 mm Hg, and the subjects performed three maximum height reference (REF) squat jumps, allowing us to study the potential effect of the presence of the pressurized cuff on jumping performance. After these jumps, the cuff was depressurized, and the subjects were transferred to the dynamometer to recover for 5 min. The cuff was then pressurized again, and the subjects performed a fatiguing protocol, consisting of 30 maximum-effort isometric plantarflexion contractions of 2-s duration alternated with 2 s of rest. With the pressurized cuff preventing recovery from fatigue (28), the subjects were released from the dynamometer to perform three fatigued (FAT) squat jumps. Immediately thereafter, the subjects were transferred back to the dynamometer to produce three MVC, allowing us to check whether any recovery had

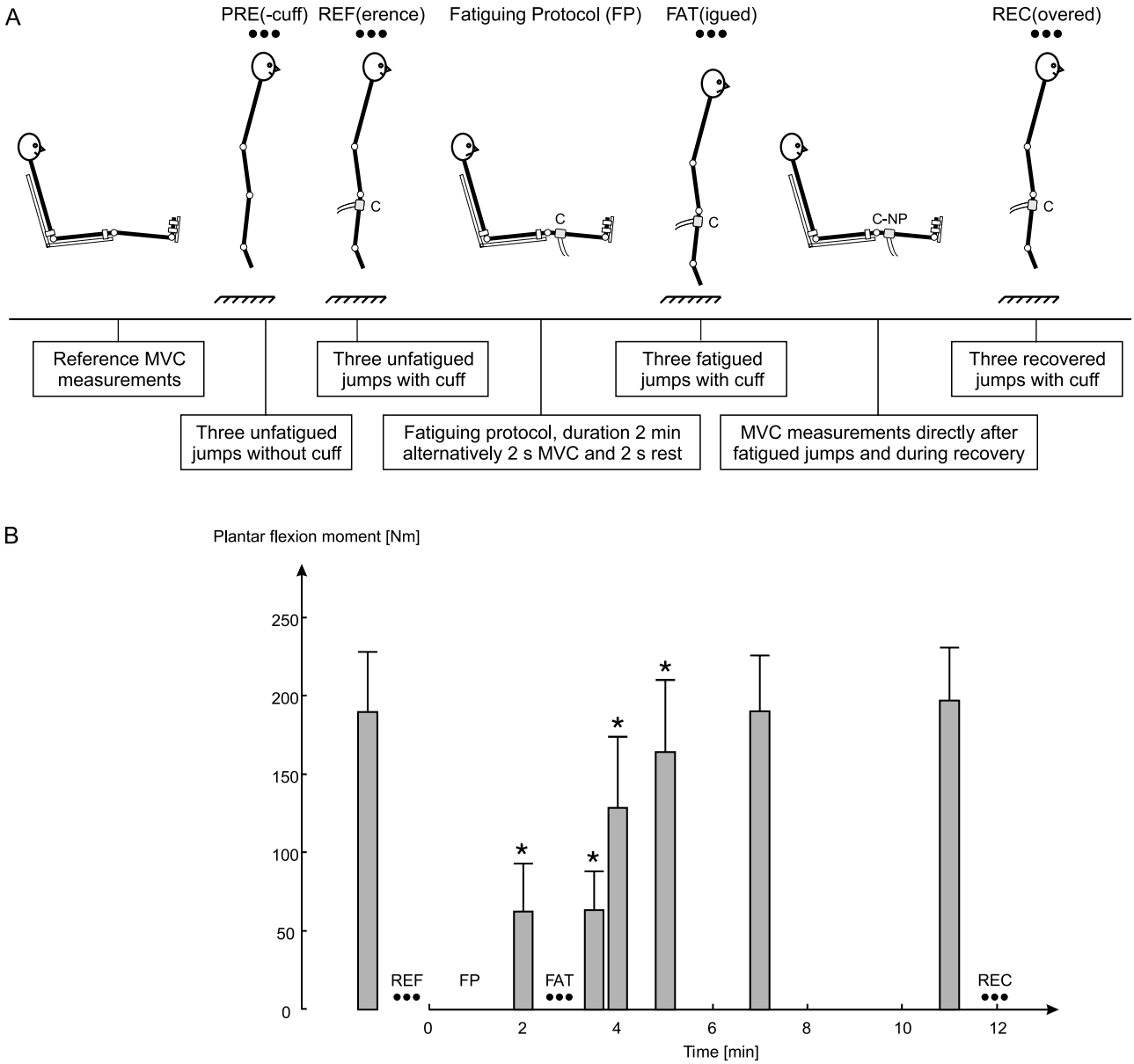

FIGURE 1—A, Experimental protocol. The experiment involved MVC of the right plantarflexors on a dynamometer, fatiguing contractions of the right plantarflexors, and maximum-effort vertical jumps (each dot over the subject's head represents one jump), with or without a pressurized cuff (C) around the lower leg. After the fatigued jumps, the subjects performed three MVC on the dynamometer with pressure on the cuff; thereafter, the cuff was depressurized (C-NP) to monitor recovery. For further details, see text. B, Mean ± SD values of isometric plantarflexion moments of the subjects (n = 6) during MVC in the different experimental conditions. *Significantly different from REF values (P < 0.05).

occurred during the squat jumps. The cuff was then depressurized, and MVC recovery measurements were conducted as indicated in Figures 1A and B. At the end of the session, the cuff was pressurized again, and the subjects performed three final recovered (REC) squat jumps, allowing us to check whether full recovery had occurred.

During jumping, sagittal-plane positional data of anatomical landmarks on both sides of the body were collected, ground reaction forces were measured with two force plates, and EMG was recorded from six muscles of the left and right leg. Jump height, defined as the difference between the height of the center of mass of the body (CM) at the apex of the jump and the height of CM in standing upright with heels on the ground, was calculated from the position data. Net joint moments and work of the individual legs were obtained by performing an inverse-dynamics analysis, combining kinematic information and ground reaction forces. Differences among conditions were tested for statistical significance using repeated-measures ANOVA. Details on experimental methods and procedures are provided in the succeeding sections.

Measurement of plantarflexion moment on the dynamometer. To measure the plantarflexion moment, subjects were seated in the dynamometer with their right

leg extended as shown in Figure 1A. The foot was strapped tightly to the footplate, and the footplate was oriented such that the sole of the foot was perpendicular to the lower leg. The plantarflexion moment was measured using a custom-made moment transducer. Moment values were displayed in real time on an oscilloscope placed in front of the subjects to provide visual feedback, and subjects were verbally encouraged to produce a maximum effort.

Kinematics and kinetics. Kinematic data were collected using an Optotrak (Northern Digital, Waterloo, Ontario) system, operating at 200 Hz. Infrared light–emitting diodes were placed on both sides of the body at the fifth metatarsophalangeal joint, calcaneus, lateral malleolus, lateral epicondyle of femur, greater trochanter, and acromion. Only sagittal-plane projections were used in this study. The marker trajectories were smoothed using a bidirectional low-pass Butterworth filter with a cutoff frequency of 8 Hz. The locations of the mass centers of upper legs, lower legs, and feet were estimated from the landmark coordinates, in combination with results of cadaver measurements presented in the literature (9). The location of the mass center of the upper body relative to the two markers on this segment was determined from two different equilibrium postures of the subjects, as explained elsewhere (5). With this information, the location of CM was calculated in all other body postures found during jumping. To obtain linear velocities and accelerations, the smoothed position–time histories were differentiated numerically with respect to time using a direct five-point derivative routine. Angles of body segments with respect to the horizontal were calculated from the smoothed marker position–time histories and differentiated to obtain angular velocities and accelerations.

Ground reaction forces were measured using two force platforms (Kistler 9281B; Kistler Instruments Corp., Amherst, NY), one underneath each leg. The output signals of the platforms were amplified (Kistler 9865E charge amplifier; Kistler Instruments Corp.), sampled at 200 Hz, and processed to determine the fore-and-aft and vertical components of the reaction force and the location of the center of pressure under each foot.

Net forces, moments, and work at the joints of each leg were calculated following a standard inverse-dynamics approach (11) using the measured ground reaction force vector in combination with locations of joint axes and segmental mass centers obtained from the positional data, linear and angular accelerations of segments derived from positional data, and segmental masses and moments of inertia calculated using regression equations (29).

Electromyography. In each leg, pairs of Ag–AgCl surface electrodes (Medicotest, Blue Sensor, type N-00-S) were applied to the skin overlying the m. soleus, m. gastrocnemius (caput mediale), m. vastus lateralis, m. rectus femoris, m. gluteus maximus, and m. biceps femoris (caput longum). The EMG signals were amplified and sampled at 1000 Hz (Porti-17t; Twente Medical Systems International, Oldenzaal, The Netherlands). Offline, they were high-pass filtered at 7 Hz to remove any possible movement artifacts, full-wave rectified, and smoothed using a bidirectional digital low-pass Butterworth filter with a 7-Hz cutoff frequency to yield smoothed rectified EMG (SREMG).

SREMG signals were normalized for the highest SREMG level found in the three REF jumps of each subject to allow for comparison of peak SREMG levels among conditions. Furthermore, to allow for comparison of timing of muscle activation among conditions, we determined for each muscle SREMG onset as explained elsewhere (3). Briefly, we fitted a line to two points on the ascending slope of the SREMG time history and extrapolated this line backward in time to where the SREMG level equaled the level observed in the equilibrium initial posture. For comparisons of SREMG onset patterns, we used the SREMG onset of m. gluteus maximus of the left, unfatigued leg as reference (3).

Statistics. Main effects of fatigue on several dependent variables, among which jump height, net joint work, SREMG onsets, and peak SREMG values, were tested to significance using a general linear model ANOVA for repeated measures; when a significant F value was found, *post hoc* pairwise comparisons of means were made using the least significant difference *post hoc* test (Statistical Package for the Social Sciences for Windows; SPSS Inc., Chicago, IL). The level of significance for all tests was 0.05. Because each subject performed three consecutive jumps in each condition, we included "trial" as a separate factor in the ANOVA. We will report per variable per condition a mean and an SD, where the latter is calculated over six values, each of which is the mean over the trials of one subject in a given condition.

Computer simulations. For simulations of jumps, we used a two-dimensional forward dynamic model of the human musculoskeletal system, schematically shown in Figure 2C (cf. van Soest et al. (27)). The model, which had muscle stimulation STIM as its only independent input, consisted of seven rigid segments representing the right foot, the right shank, the right thigh, the left foot, the left shank, the left thigh, and the head-arms-trunk. These segments were interconnected by hinges representing hip, knee, and ankle joints, and the distal part of the foot was connected to the ground by a hinge joint. In the initial posture, the rotational degree of freedom of each foot was fixed to mimic that the subjects had their heels on the ground, and we calculated the moment of the ground reaction force relative to the distal part of the foot that was needed to prevent angular acceleration. The rotational degree of freedom was released during simulations when the calculated moment of the reaction force on the foot dropped to zero, which represents the instant at which the heel came off the ground. Segment parameters were the same as those used in a model for simulation of two-legged jumping, which was previously described in full detail (25).

Within the skeletal submodel, six major muscle–tendon complexes (MTC) of each of the lower extremities were embedded: hamstrings (biarticular heads), m. gluteus maximus, m. rectus femoris, mm. vasti, m. gastrocnemius, and m.

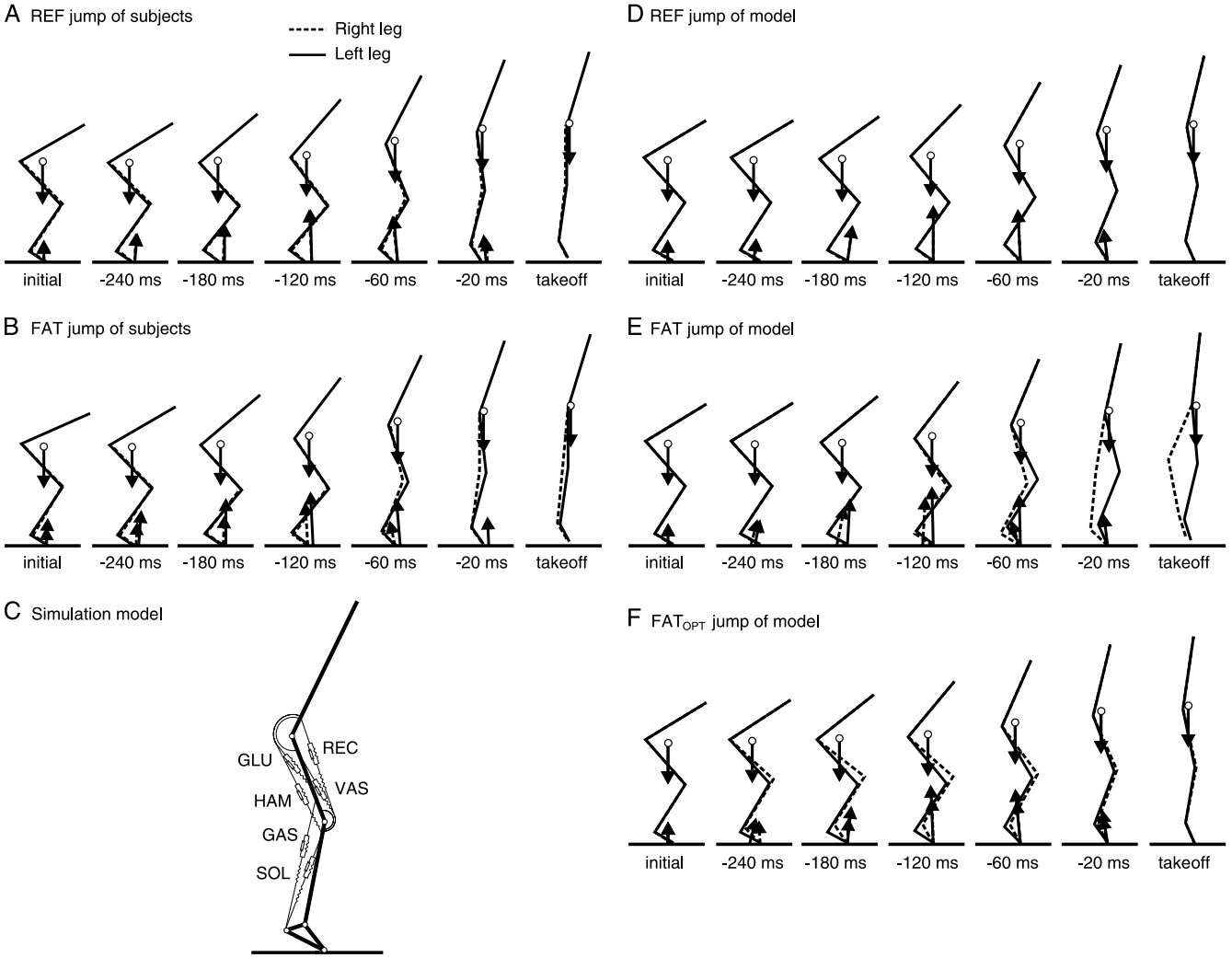

FIGURE 2—Stick diagrams of average body postures of the subjects (n = 6) for the jumps in different conditions (A, B), forward dynamic simulation model used (C), and stick diagrams for jumps of the model (D–F). Arrows pointing upward represent the ground reaction force vector on the individual legs plotted with the origin in the center of pressure; arrows pointing downward represent the force of gravity and are plotted with their origin in the CM (open circles). Time is expressed relative to the instant of takeoff (time = 0). The model consisted of seven interconnected rigid segments (right foot, right shank, right thigh, left foot, left shank, left thigh, and head–arms–trunk) and 12 MTC of the lower extremity (gluteal muscles (GLU), hamstrings (HAM), rectus femoris (REC), vasti (VAS), gastrocnemius (GAS), and soleus (SOL) of both the left and the right leg), all represented by Hill-type muscle models. Simulations were made for REF (reference model, optimal stimulation-time input) (D), FAT (reference model with weakened plantarflexors of the right leg and stimulation-time input taken from the REF condition) (E), and FAT$_{opt}$ (model with weakened plantarflexors of the right leg used for FAT condition, but with optimized stimulation-time input) (F).

soleus. Each MTC was represented using a Hill-type muscle model. This muscle model, which has also been described in full detail elsewhere (25), consisted of a contractile element (CE), a series elastic element (SEE), and a parallel elastic element. Briefly, the behavior of SEE and parallel elastic element was determined by a quadratic force–length relationship, whereas the behavior of CE was complex: CE velocity depended on CE length, force, and active state, with the latter being defined as the relative amount of calcium bound to troponin (10). Following Hatze (13), the relationship between active state and STIM was modeled as a first order process. STIM, ranging between 0 and 1, was a one-dimensional representation of the effects of recruitment and firing frequency of α-motoneurons.

At the start of each simulation, the model was put in the average initial posture chosen by the subjects, and the initial STIM levels were set in such a way that the net joint moments kept the system in static equilibrium. To find a unique solution for the initial STIM levels, we first assigned a STIM of 0.01 to the biarticular hamstrings, m. rectus femoris, and m. gastrocnemius, causing them to produce a small force that took up the slack in SEE. Subsequently, we calculated the STIM levels for the other muscles that ensured equilibrium of the system as a whole. During propulsion, STIM of each muscle was allowed to increase from its initial level toward its maximum of 1. The increase started at a switch time and occurred at a fixed rate of 5 s^{-1}. Under these restrictions, the motion of the body segments and therewith

the performance of the model depended on a set of switch times. The optimization problem, finding the combination of switch times that produced maximum jump height, was solved using a genetic algorithm (26). For each condition, the optimization ran for 1000 generations of a population of 100 chromosomes, with each chromosome being a bit string, coding a combination of stimulation onset times at a 1-ms resolution. It was confirmed that the algorithm converged to virtually identical solutions when started from different initial guesses.

The model was used to simulate three conditions. In one condition, REF, we found the optimal control solution for a maximum height two-legged squat jump. In a second condition, FAT, we reduced the force of m. soleus and m. gastrocnemius of the right leg by the amount observed in the fatigued MVC (see Results section) and found the STIM levels that produced equilibrium in the initial posture. We then applied the STIM onsets found for the REF condition to simulate a jump in which control had not been tuned to changed muscle properties. As will be explained later on, we also attempted two variations of these FAT jumps on the basis of the observations made in the subjects. Finally, in the third condition, FAT_{opt}, we found the optimal solution for the fatigued model.

RESULTS

Isometric plantarflexion moment. The peak plantarflexion moment produced by the subjects amounted to 190 ± 38 N·m in the initial MVC and dropped by approximately 70% to 61 ± 31 N·m during the fatiguing contractions (Fig. 1B). It remained at this low level during FAT jumps and returned to its original value within 2.5 min after removal of the cuff (Fig. 1B).

Jumps performed by the subjects. Initial height of CM and height of CM at takeoff did not differ among conditions (Table 1a), but a main effect of condition was found for jump height, and hence peak height reached by CM. Jump height was not different between PRE jumps and REF jumps but was approximately 6 cm less in FAT jumps ($P < 0.05$). There was no sign of improvement over the three consecutive FAT jumps; the average jump height in the third FAT jump was only 0.7 cm higher ($P = 0.59$) than that in the first FAT jump (results not shown). After removal of the cuff, the subjects quickly recovered; the REC jumps were approximately 4.5 cm higher ($P < 0.05$) than the FAT jumps but still approximately 1.5 cm ($P < 0.05$) less high than the REF jumps. The drop in mechanical energy of CM reached in FAT jumps compared with REF jumps closely corresponded to the drop in total work calculated by inverse-dynamics analysis (Table 1a). It seems safe, therefore, to analyze the changes in performance in terms of changes in the net work about individual joints. From here on, we will concentrate on the differences between REF and FAT jumps. The factor "trial" will receive no further attention because it had no statistically significant main effect or interaction effect on any of the variables.

Average body postures and ground reaction force vectors at selected instants during REF and FAT jumps are illustrated in Figures 2A and B. No difference was found in initial posture in the sagittal plane (joint angles were not different), but in FAT jumps, the subjects initially carried only 40% of body weight with their fatigued right leg (which means that in the frontal plane, they had their CM closer to the unfatigued left leg). During push-off, the ground reaction force produced by the fatigued leg was smaller than that produced by the unfatigued leg and dropped to zero approximately 20 ms before takeoff (Fig. 2B). At takeoff in FAT jumps, the right knee was more extended by approximately 4° ($P < 0.05$) than in REF jumps. No difference was observed between FAT and REF jumps in SREMG onset times of muscles relative to SREMG onset of m. gluteus maximus of

TABLE 1a. Mean ± SD Values of selected variables describing the squat jumps performed by the subjects ($n = 6$) in precuff (PRE), reference (REF), fatigued (FAT), and recovered (REC) conditions.

Variable	Unit	PRE	REF	FAT	REC
$z_{CM,initial}$	m	−0.18 ± 0.06	**−0.18 ± 0.06**	**−0.19 ± 0.07**	−0.18 ± 0.07
$z_{CM,to}$	m	0.10 ± 0.01	**0.10 ± 0.01**	**0.10 ± 0.01**	0.10 ± 0.01
$z_{CM,max}$	m	0.34 ± 0.05	**0.34 ± 0.05**	**0.28* ± 0.06**	0.33* ± 0.05
$\Delta E_{CM,tot}$	J·kg^{-1}	5.15 ± 0.62	**5.14 ± 0.79**	**4.61* ± 0.80**	4.91* ± 0.83
$W_{both\ legs\ total}$	J·kg^{-1}	5.62 ± 0.66	**5.52 ± 0.73**	**4.96* ± 0.82**	5.40 ± 0.91
Unfatigued leg					
$W_{left\ hip}$	J·kg^{-1}	0.93 ± 0.32	**0.96 ± 0.38**	**1.25* ± 0.39**	0.84* ± 0.37
$W_{left\ knee}$	J·kg^{-1}	0.91 ± 0.10	**0.89 ± 0.13**	**0.88 ± 0.15**	0.95 ± 0.17
$W_{left\ ankle}$	J·kg^{-1}	1.00 ± 0.14	**0.98 ± 0.14**	**1.07 ± 0.20**	0.98 ± 0.15
$W_{left\ leg\ total}$	J·kg^{-1}	2.85 ± 0.31	**2.83 ± 0.37**	**3.20* ± 0.47**	2.77 ± 0.47
Fatigued leg					
$W_{right\ hip}$	J·kg^{-1}	0.86 ± 0.45	**0.88 ± 0.52**	**0.51* ± 0.45**	0.81 ± 0.54
$W_{right\ knee}$	J·kg^{-1}	0.89 ± 0.19	**0.91 ± 0.20**	**0.95 ± 0.13**	0.93 ± 0.18
$W_{right\ ankle}$	J·kg^{-1}	1.02 ± 0.21	**0.91 ± 0.19**	**0.29* ± 0.13**	0.89 ± 0.23
$W_{right\ leg\ total}$	J·kg^{-1}	2.77 ± 0.35	**2.69 ± 0.36**	**1.75* ± 0.46**	2.62 ± 0.44

SD was calculated over six values, each of which was the mean over three jumps of one subject in a given condition. Bold values are to be compared with simulation results (Table 1b).
* indicates that value was significantly different from REF ($P < 0.05$).
$z_{CM,initial}$: height of the center of mass of the body in the equilibrium initial posture relative to standing upright; $z_{CM,to}$: height of the center of mass of the body at takeoff relative to standing upright; $z_{CM,apex}$: height of the center of mass of the body at the apex of the jump relative to standing upright (i.e., jump height); ΔE_{tot}: change in mechanical energy of center of mass from initial height to apex of the jump; W: work as indicated in subscript calculated using inverse-dynamics analysis.

TABLE 1b. Values of selected variables describing the squat jumps of the simulation model from initial postures observed in the subjects in reference (REF) and fatigued (FAT) conditions.

Variable	Unit	REF	FAT	$FAT_{F\_RL\_eq=0.4}$	$FAT_{q\_HAM\_0.8}$	FAT_{opt}
$z_{CM,initial}$	m	**−0.19**	**−0.19**	−0.19	−0.19	−0.19
$z_{CM,to}$	m	**0.07**	**0.08**	0.08	0.08	0.07
$z_{CM,max}$	m	**0.34**	**0.27**	0.27	0.27	0.30
$\Delta E_{CM,tot}$	J·kg^{-1}	**5.15**	**4.50**	4.49	4.49	4.72
$W_{both\ legs\ total}$	J·kg^{-1}	**6.32**	**5.73**	5.74	5.71	5.91
Unfatigued leg						
$W_{left\ hip}$	J·kg^{-1}	**1.33**	**1.16**	1.16	1.22	1.37
$W_{left\ knee}$	J·kg^{-1}	**0.97**	**1.23**	1.24	1.21	1.04
$W_{left\ ankle}$	J·kg^{-1}	**0.86**	**0.92**	0.92	0.93	0.87
$W_{left\ leg\ total}$	J·kg^{-1}	**3.16**	**3.31**	3.32	3.36	3.28
Fatigued leg						
$W_{right\ hip}$	J·kg^{-1}	**1.33**	**1.13**	1.12	1.05	1.01
$W_{right\ knee}$	J·kg^{-1}	**0.97**	**1.08**	1.08	1.08	1.13
$W_{right\ ankle}$	J·kg^{-1}	**0.86**	**0.21**	0.22	0.22	0.49
$W_{right\ leg\ total}$	J·kg^{-1}	**3.16**	**2.42**	2.42	2.35	2.63

Values in boldface are to be compared with experimental results (Table 1a). The FAT jump was obtained by reducing the isometric force of the right plantarflexors by 70%, by finding equilibrium, and by imposing the stimulation (STIM) onsets that were optimal for the REF model. The $FAT_{F\_RL\_eq=0.4}$ condition was the same as the FAT condition, but in equilibrium (eq) the force (F) of the right leg (RL) was only 40% of body weight. The $FAT_{q\_HAM\_0.8}$ condition was the same as the FAT condition, but the maximal STIM level of the right hamstrings was reduced to ensure that the active state of these muscles reached only 80% of its maximum during the jump. The FAT_{opt} condition was obtained by reoptimization of STIM onsets for the FAT model.

the left leg ($P > 0.05$ for all muscles, results not shown), but some differences were found in SREMG amplitudes (Fig. 3); the latter will be specified in the next section.

As can be seen in Table 1a, the reduction in height of FAT jumps was the net outcome of a reduction in total work of the fatigued right leg by 35% and an increase in total work of the left leg by 13%. Most of the reduction in total work of the right leg was due to a reduction in the net ankle work by 70%, but the net work at the hip joint of the right leg was also reduced by 40%. The reduction in the right ankle work in FAT jumps did not come as a surprise; muscle force of the plantarflexors had dropped by 70% because of the fatiguing protocol, and furthermore peak SREMG amplitudes of the right m. soleus and m. gastrocnemius were reduced by more than 35% ($P < 0.05$) in FAT jumps compared with REF jumps (Fig. 3). The reduction in the right hip work, however, could not be due to fatigue of the hip extensor muscles nor to a reduction in SREMG amplitude of the right m. gluteus maximus (Fig. 3). Also, no difference was found between FAT and REF jumps in angular displacement during the push-off in the right hip joint ($P > 0.05$). What we did find was a 20% reduction in SREMG amplitude of the right hamstrings in FAT jumps compared with REF jumps (Fig. 3). Furthermore, the average angular velocity of the right hip extension during the push-off was approximately 20% higher in FAT jumps than in REF jumps (and 165°·s^{-1} and 141°·s^{-1}, respectively, $P < 0.05$).

Although not of major interest in this study, let us briefly address the increase in total work of the unfatigued left leg. This increase was primarily due to an increase of net hip work by 10% in FAT jumps relative to REF jumps (Table 1a, $P < 0.05$). On average, the peak angular velocities of hip extension, knee extension, and plantarflexion were reduced, but the differences between FAT and REF jumps were not statistically significant. However, we did find a statistically significant 20% increase in left hamstrings activation in FAT jumps relative to REF jumps (Fig. 3).

Jumps of the simulation model. The simulated REF jump of the model matched the REF jumps of the subjects well in terms of vertical displacement of CM (compare Table 1b with Table 1a, respectively) and kinematics (compare Fig. 2D with Fig. 2A, respectively). The salient features of the effects of fatigue of the plantarflexors that had been observed in the subjects were reproduced by the FAT jump of the simulation model, obtained by reducing the maximal isometric force of the plantarflexors by 70% and using the STIM onsets that were optimal for the REF model. In the FAT jump of the model, jump height was reduced by 6.7 cm (Table 1b). Furthermore, in the FAT jump of the model, the ground reaction force of the right leg dropped to zero before takeoff, and when it did, the right knee was more extended than the left knee (Fig. 2E), just like in the FAT jump of the subjects (Fig. 2B). At takeoff, the right knee joint was hyperextended in the FAT jump of the model (Fig. 2E). The difference between FAT and REF jumps in right knee angle at takeoff was more pronounced in the simulated jumps than in the jumps of the subjects because the model did not have passive elements to prevent hyperextension of the knee joint. The reduction in jump height in the FAT jump of the model was the net outcome of a reduction in the total work of the fatigued right leg by 23% and an increase in total work of the left leg by 5% (Table 1b). Finally, just like in the subjects, the work of the "fatigued" right leg of the simulation model was reduced not only because of a reduction in the right ankle work but also because of a reduction in the right hip work (Table 1b).

In FAT jumps of the subjects, two unhoped-for changes were observed compared with REF jumps, which might have contributed to the reduction in work of the right leg and jump height. Firstly, the subjects initially carried only 40% of body weight with their right leg in FAT (Fig. 2B). Starting from a lower initial force, the muscles of the right leg will have tended to produce less work over the initial part of their shortening range because force development takes time (2). To test whether the initial weight distribution played an important role, we performed an additional simulation similar to the FAT simulation, but this time from an equilibrium posture (ep) in which the force (F) of the right leg (RL) was

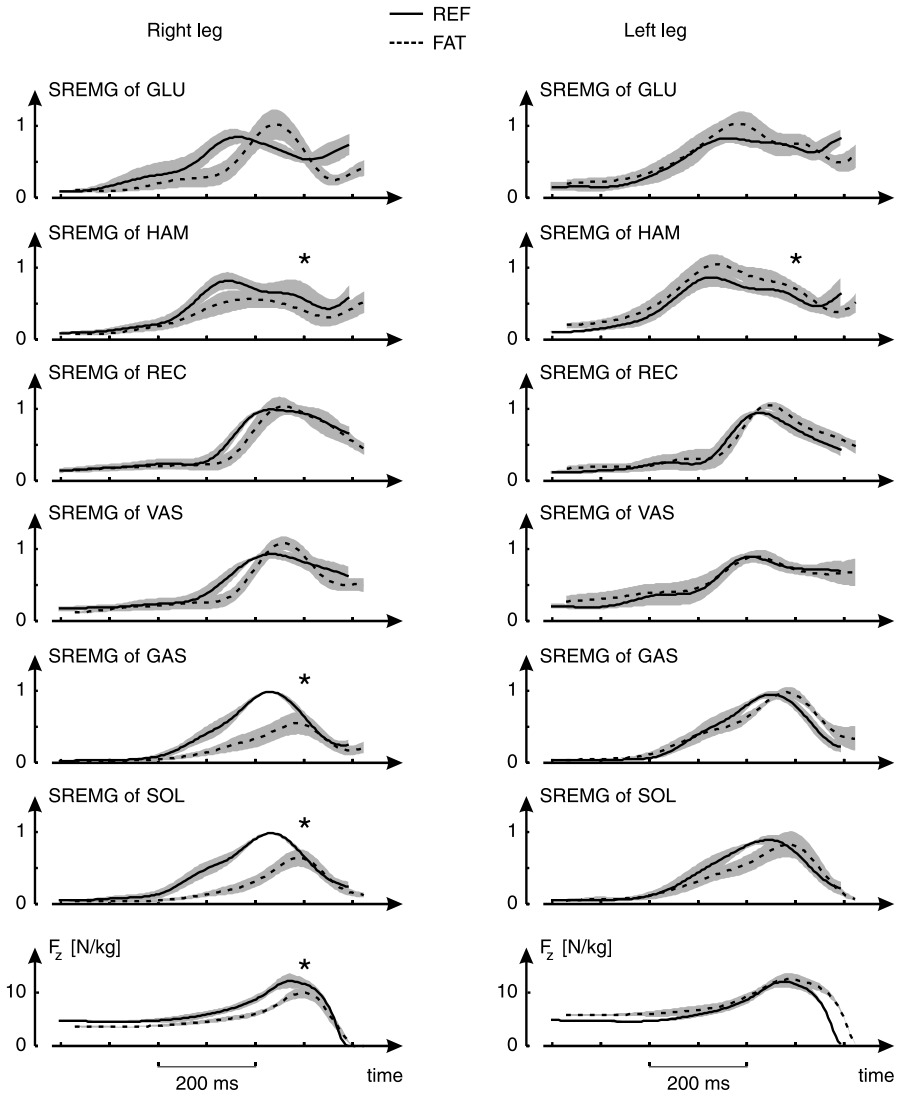

FIGURE 3—Average time histories of smoothed rectified EMG (SREMG) and vertical ground reaction force (F_z) of the subjects (n = 6) during the push-off in reference (REF) and fatigued (FAT) conditions. Muscle names as defined in Figure 2. Graphs were constructed as follows. SREMG histories of each subject were first normalized for the highest SREMG level found in the three REF jumps of that subject. After alignment of time histories at takeoff, mean and SEM values over subjects (gray areas) were calculated. Curves of REF and FAT conditions have been aligned at the average instant that SREMG onset of m. gluteus maximus (GLU) in the unfatigued leg was detected. *Difference between FAT and REF in peak value is statistically significant (P < 0.05).

40% of body weight and that of left leg 60%, just like in the subjects (condition $FAT_{F\_RL\_eq=0.4}$). Only minimal changes occurred in work and performance (Table 1b). It seems, therefore, that the initial weight distribution can be safely neglected when it comes to explaining the reduction in work of the right leg in FAT jumps. The second unhoped-for change observed in FAT jumps of the subjects was the reduction in peak SREMG level of the right hamstrings by 20% (Fig. 3). To test whether this played an important role, we performed an additional simulation similar to the FAT simulation, but this time we reduced the maximal STIM level of the right hamstrings to ensure that the active state of these muscles reached only 80% of maximum during the jump (condition $FAT_{q\_HAM\_0.8}$). Again, only minimal changes occurred in work and performance (Table 1b). Thus, the effect of reduced neural input to the

TABLE 2. Work ($J \cdot kg^{-1}$) produced by individual MTC of the simulation model. Conditions as defined in Table 1b; muscle names as defined in Figure 2.

Variable	REF	FAT	$FAT_{F\_RL\_eq=0.4}$	$FAT_{q\_HAM\_0.8}$	FAT_{opt}
Unfatigued leg					
Left GLU	1.10	1.18	1.19	1.18	1.15
Left HAM	0.62	0.61	0.61	0.62	0.68
Left REC	0.01	−0.05	−0.05	−0.03	−0.02
Left VAS	0.71	0.79	0.79	0.80	0.73
Left GAS	0.44	0.47	0.48	0.48	0.46
Left SOL	0.28	0.31	0.31	0.31	0.27
Left leg total	**3.16**	**3.31**	3.32	3.36	3.28
Fatigued leg					
Right GLU	1.10	1.06	1.06	1.07	1.03
Right HAM	0.62	0.56	0.56	0.46	0.52
Right REC	0.01	0.05	0.05	0.06	−0.02
Right VAS	0.71	0.67	0.67	0.68	0.67
Right GAS	0.44	0.07	0.07	0.07	0.29
Right SOL	0.28	0.01	0.01	0.01	0.14
Right leg total	**3.16**	**2.42**	2.42	2.35	2.63
Both legs total	**6.32**	**5.73**	5.74	5.71	5.91

Values in boldface are to be compared with experimental results (Table 1a).

right hamstrings can also be safely neglected in explaining the reduction in work of the right leg in FAT.

In the simulation model, the reduction in right ankle work in the FAT jump corresponded in magnitude to the reduction in work of the weakened plantarflexors (Table 2). The changes in work at the other joints cannot be explained this way because these joints were not spanned by weakened muscles; instead, the changes in work at the other joints were indirectly due to the effects of the reduction mechanical output at the right ankle on the movement pattern and therewith the contraction conditions of the muscles. The work produced by each individual muscle is equal to the integral of force with respect to MTC length, with force depending on CE length, CE contraction velocity, and active state. Let us focus on work of the right hip extensor muscles. In the FAT jump, work of these hip extensor muscles was reduced by 0.1 $J \cdot kg^{-1}$. The range of shortening of these muscles was increased in the FAT jump, which favored work production (Fig. 4). No difference was found in active state during shortening, but CE-shortening velocities were greater in the FAT jump (Fig. 4), which impeded work production due to the force–velocity relationship and caused work of the right hip extensors to drop despite the increased range of shortening. Note that the total reduction in work of the right hip extensor muscles (Table 2) was less than the reduction in right hip work (Table 1b). This should not come as a surprise; although the sum of net work over all joints must obviously be equal to the sum of work over all muscles, there is no formal relationship between net work at a joint and the work produced by the muscles spanning that joint. For example, if a biarticular muscle M is solely responsible for an extension moment at joint J and J extends over a certain angular displacement, joint work at J is positive. Whether muscle M itself contributes positive work, however, depends on its length change, and this length change is not only determined by the angular displacement at joint J but also by the angular displacement at the other joint that muscle M spans. From within a joint work approach, it has been said that a biarticular muscle may transfer energy between joints (17,18,24). In our simulation model, the "discrepancy" between the total reduction in work of the right hip extensor muscles (Table 2) and the reduction in right hip work (Table 1b) was partly due to the fact that the hip flexion moment of m. rectus femoris "absorbed" more work at the hip in the FAT jump than in the REF jump.

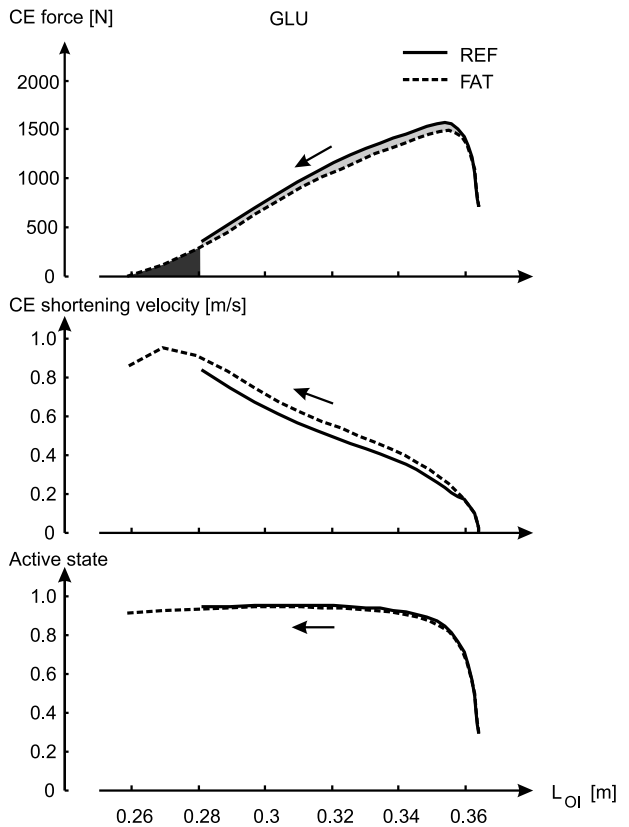

FIGURE 4—Force, shortening velocity, and active state of CE of m. gluteus maximus (GLU) as a function of origin-to-insertion distance (L_{OI}) for reference (REF) and fatigued (FAT) jumps of the model. Arrows indicate the direction of time. In the upper panel, the gray area represents a deficit and the black area a surplus of work in the FAT jump compared with the REF jump.

The simulation model also allowed us to test our second hypothesis, that is, that the loss in total work that occurs with fatigue of the right plantarflexors is unnecessarily high and can be partly remedied by reoptimization of control. In the optimal jump of the FAT model (FAT_{opt}), jump height was approximately 2.5 cm greater than in the REF jump (Table 1b). The gain was primarily due to an increase in work of the right leg, which in turn was primarily due to an increase in work of the weakened plantarflexors. The latter increase was achieved by earlier activation and consequent earlier shortening of these muscles at a lower velocity than in the REF jump (Fig. 2F). Thus, our second hypothesis was supported as well.

DISCUSSION

It has previously been shown in a simulation study that a mismatch between control and musculoskeletal properties leads to an unbalanced increase in segment angular velocities, causing the shortening velocity of some muscles to be disproportionally high and the total work produced to be unnecessarily small (7). In the present study, we set out to verify these effects of a mismatch between control and musculoskeletal properties in jumps of human subjects. We attempted to achieve this mismatch by fatiguing the plantarflexors of the right leg and by preventing the subjects from practicing with their fatigued muscles. We observed that the net work at the right hip joint during jumping was reduced in the subjects, and because the right hip joint was not spanned by fatigued muscles, we took this finding as support for our first hypothesis that loss of work of the fatigued plantarflexors also leads to loss of work of other, unfatigued muscles. We confirmed and explained this outcome by simulating the jumps of the subjects with a model

of the musculoskeletal system, and we used the model to show that reoptimization of control caused performance to be partly restored (from −6.7 cm to −4.2 cm; Table 1b). This suggests that the drop in performance in FAT was indeed partly due to a mismatch between control and musculoskeletal properties and not only by the reduction in work of the right plantarflexors, and therefore supports our second hypothesis that the loss of performance that occurs with fatigue of the right plantarflexors is unnecessarily high. We will first discuss some of the assumptions underlying interpretation of the findings and then some possible implications.

Our interpretation of the reduced performance of the subjects in FAT jumps compared with REF jumps builds on the assumption that control was not changed in the subjects. Specifically, we assumed that the subjects did not reduce the neural drive to their muscles in FAT jumps. Contrary to our assumption, we did find statistically significant changes in SREMG amplitude of some muscles. Peak SREMG amplitude of the right m. soleus and m. gastrocnemius was reduced substantially in FAT jumps compared with REF jumps (Fig. 3), presumably because of reduced central drive (21). This was not too much of a problem for our study. It contributed to the reduction in the mechanical output of the right plantarflexors, but this reduction itself was not of primary interest; it was just a means to achieve a mismatch between control and musculoskeletal properties. Peak SREMG of the right hamstrings was also reduced by approximately 20% in FAT jumps compared with REF jumps. This was potentially troublesome because we were aiming for an explanation of the work reduction by 40% at the right hip on the basis of factors other than reduced neural input to right hip extensors. However, as discussed in the next paragraph, our simulation results suggest that a 20% reduction in active state of the hamstrings hardly affected work at the right hip. Clearly, interpretation of the findings of the subject experiments without the help of a model is hazardous. Finally, we were surprised to find a statistically significant increase of SREMG in the left hamstrings in FAT jumps compared with REF jumps (Fig. 3).

If we want to use the simulation model to explain the effects of fatigue that were observed in the subjects, we need to assume, of course, that the simulation model is a valid representation of the real system. The model is in essence a simplification of the real system, and many of the properties of the real system, such as the force–length and the force–velocity relationships of individual muscles, cannot be measured *in vivo*. The parameter values of the model therefore had to be obtained by combining the results of experiments on human cadavers, experiments on isolated muscles of animals, and dynamometer experiments on human subjects (for details, see (1)) and have the status of educated guesses. Furthermore, the model was only two dimensional. Nevertheless, optimization of STIM(t) using only CM height as criterion has previously allowed us to reproduce various types of jumps of human subjects with the model, not only in terms of kinematics and kinetics but also in activation onset patterns of muscles (3). In the present study, the kinematics and kinetics of the simulated REF jumps also satisfactorily matched those of the jumps of the subjects (cf. Figs. 2A and D, Tables 1a and b), and reducing the isometric force of the plantarflexors of the right leg to simulate FAT jumps caused effects that were similar to those found in the subjects (cf. Figs. 2B and E, Tables 1a and b). The most important similarities between subjects and model were as follows: 1) the reduction in jump height in the FAT model was of similar magnitude as that observed in the subjects, 2) the reduction was due to the net outcome of an increase in work produced by the left leg and a decrease of work produced by the fatigued right leg, 3) work of the right leg was reduced because of both a decrease in work at the right ankle and a decrease in work at the right hip joint, and 4) the right knee joint was hyperextended at takeoff. Thus, although the use of a more detailed three-dimensional model (e.g., see Nagano et al. (16)) would have been more elegant to simulate the FAT jumps of the subjects, we feel that our simplified two-dimensional model did reproduce the salient features of the real system.

The main finding in the jumps of the subjects was that the work at the right hip joint was less in FAT jumps than in REF jumps, although this joint was not crossed by fatigued muscles. This finding was reproduced with the FAT jump of the simulation model, regardless of whether the initial weight distribution was asymmetrical (condition $FAT_{F\_RL\_eq=0.4}$; Table 1b), as in the subjects, or symmetrical (condition FAT; Table 1b). Furthermore, when we took our FAT model and reduced the maximal STIM level of the right hamstrings to ensure that the active state of these muscles reached only 80% of maximum during the jump (condition $FAT_{q\_HAM\_0.8}$), only a negligible reduction in right hip work occurred (Table 1b). Thus, we are not too concerned that the reduced neural drive to the right hamstrings observed in the subjects invalidates our interpretation of the reduced right hip work in FAT jumps. In an attempt to understand why right hip work in the FAT model was reduced, we analyzed work production by individual muscles (while realizing that the net work at a joint may be different from the total work of the muscles spanning that joint because of the action of biarticular muscles, as explained in the Results section). This analysis revealed that the reduction in work at the right hip joint in FAT jumps was primarily due to increased CE-shortening velocities of the hip extensors (Fig. 4), which impeded work production because of the force–velocity relationship. These findings support our previous general assertion that a mismatch between control and musculoskeletal properties leads to an unbalanced increase in segment angular velocities, causing the shortening velocity of some muscles to be disproportionally high and the total work produced to be unnecessarily small (7). Reoptimization caused an increase of the work of the fatigued plantarflexors, at the expense of a further drop in work of the unfatigued muscles of the right leg (Table 2) but to the benefit of an increase in total work of the right leg (Table 1b). The optimal solution was characterized by an

early plantarflexion and a virtually symmetrical termination of the push-off phase (Fig. 2F). Note that in the model, the increase in work of the left leg in FAT relative to REF was due to a reduction in the average push-off velocity and corresponding muscle-shortening velocities. At these lower velocities, the unfatigued muscles of the left leg generated more force according to the force–velocity relationship and hence produced more work. This effect has previously been shown to explain why a particular leg can produce more work when is it is used for propulsion in a one-legged jump than when it is used for propulsion in a two-legged jump (4). After reoptimization, the average push-off velocity increased slightly in FAT, and this explains the small drop in work of the left leg (Table 1b). In the subjects, the increased neural input to the hamstrings of the unfatigued left leg (Fig. 3) may also have contributed the work enhancement of that leg.

A final question is whether a mismatch between control and musculoskeletal properties may lead to injury of passive joint structures. In FAT jumps, hyperextension of the right knee joint occurred at takeoff, both in the subjects and in the simulation model (Figs. 2B and E). This hyperextension was indirectly due to the reduction in mechanical output of the fatigued plantarflexors and the consequent unbalanced increase of segmental angular velocities. In the subjects, the rotational energy of these segments is absorbed by passive structures crossing the knee joint; because we did not incorporate such passive structures in the simulation model, the hyperextension is exaggerated in the model (Fig. 2E). In several studies, it has been shown that forced hyperextension of the knee may lead to damage of soft tissues such as cruciate ligaments (e.g., see Fornalski et al. (12) and Schenck et al. (22)). However, our subjects did not report any discomfort in the knee in the FAT jumps, even though they had unnaturally extreme and selective fatigue of the plantarflexors and were given no opportunity to practice with their fatigued muscles and adapt their control (8). Therefore, we regard it highly unlikely that knee hyperextension at takeoff caused by a mismatch between control and muscle properties is a source of injuries in sporting activities. However, it is well possible that such a mismatch may lead to injurious mechanisms during landing or other activities involving large contact forces (15,23).

CONCLUSIONS

We have shown that fatigue of the plantarflexors of the right leg during jumping causes a loss in total muscle work that is greater than just the loss in muscle work of these plantarflexors. This loss in total muscle work is partly due to an arisen mismatch between control and musculoskeletal properties. When this mismatch is remedied by reoptimization of control, muscle work of the unfatigued muscles of the right leg increases, showing that a tuning of control to muscle properties benefits jumping performance not only when the muscles are strengthened (6,7) but also when they are weakened. However, even after reoptimization of control, muscle work of the unfatigued muscles of the right leg remains defective. This supports previous assertions (e.g., see Bobbert and van Soest (7)) that a high mechanical output of the plantarflexors is crucial for jumping; if the work of the plantarflexors drops, so does the work of the other muscles in the leg.

The authors did not receive funding for this work from any of the following organizations: the National Institutes of Health (NIH), Wellcome Trust, Howard Hughes Medical Institute (HHMI), or other(s).

The authors declare that there is no conflict of interest associated with this work.

The authors thank Richard Casius for his assistance with the optimizations.

The results of the present study do not constitute endorsement by the American College of Sports Medicine.

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