OPTIMAL CONTROL OF HUMAN RUNNING

A Dissertation Presented

by

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Department of Kinesiology
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ABSTRACT

OPTIMAL CONTROL OF HUMAN RUNNING

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Humans generally use two modes of locomotion as adults. At slow speeds we walk, and at fast speeds we run. To perform either gait, we use our muscles. The central questions in this dissertation were: (1) Why do humans run the way they do, and (2) How do the mechanical properties of muscle influence running performance? Optimal control simulations of running were generated using a bipedal forward dynamics model of the human musculoskeletal system. Simulations of running and sprinting were posed as two-point boundary value problems where the muscle excitation signals were optimized to maximize an optimality criterion. In the first study, minimizing the dimensionless muscle activations rather than the cost of transport generated the simulation that most closely agreed without experimental kinetic, kinematic, and electromyographic data from human runners. In the second study, sprinting simulations were generated by maximizing the model’s horizontal speed. Adjustments in the parameters of the muscle force-velocity relationship, in particular the shape parameter, increased the maximum speed, and provided support for previous
theories on limitations to maximum human sprinting speed. In the third study, virtual aging of the model’s muscles induced changes in the running biomechanics characteristic of older adults, and increased the stresses and strains of muscles where older runners are more frequently injured than young runners. Strengthening these muscles reduced their loading while still maintaining an economical gait with a relatively low joint contact force at the knee. The studies provide a framework for testing hypotheses on human movement without a strong dependency on experimental data, and provided new evidence on the validity of the simulation approach for studying human running, and on optimality criteria in human running, limitations to maximum sprinting speed, and relationships between aging, muscular properties, and running injuries.
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CHAPTER 1
INTRODUCTION

1.1. General Introduction

This dissertation concerns the biomechanics of human running. Running, like most movements, is achieved by coordination between the neural, muscular, and skeletal systems (Bernstein, 1967). The central nervous system sends excitatory control signals to activate the muscles, develop joint moments, actuate the skeleton, and generate the supportive and propulsive forces needed for locomotion. The musculoskeletal system is redundantly actuated, and there exists an infinitely large set of different muscle excitation combinations that could all potentially generate the net joint kinetics and limb motions needed to run. The selection of one particular combination from a vast pool is a decision-making process influenced largely by two components: the capabilities of the system, and the motor objective. This research focused on defining the motor objective for human running, and on how the capabilities of the muscular system influence the performance of these tasks.

1.2. Motivation

The present work was motivated by the popularity of running, both as a research topic in biomechanics (James, 1973; Vaughan, 1984a; Williams, 1985; Novacheck, 1998) and as an activity by the general population. Running is distinguished from walking by the presence of a flight phase, and by the in-phase fluctuations of the kinetic and potential energies. Humans generally run when they wish to move somewhere at a faster speed than is capable
by walking. Bipedal running is thought to be one of the major evolutionary pathways between humans and our closest genetic relatives (Bramble & Lieberman, 2004). Carrier (1984) proposed that the ability to run allowed human ancestors to hunt faster quadrupedal prey. This concern does not appear often today, but running is still an activity that people perform often during their lives, particularly if they are athletically inclined.

Additional motivation was provided by the current lack of answers to some general questions on the mechanics of running. Endurance running has high rates of injuries that, while multi-factorial, undoubtedly contain a biomechanical component within their etiology. Consequently, injuries have been widely studied from a biomechanical perspective, with the objectives of identifying causal and preventative factors (Hreljac, 2004, 2005). However, this research has not translated to a reduction in overall injury rates (Nigg, 2001), suggesting that the biomechanical basis of injury is not yet well understood. Older runners incur different injury patterns than young runners (McKean et al., 2006) and also have different muscular properties (Narici et al., 2008) and running biomechanics (Bus, 2003), but there are no causal links between these factors, nor any guidelines for preventing injuries in older runners. In addition to endurance running, sprint running has also been widely studied (Mero et al., 1992). One of the most important variables in sprinting is the maximum speed, but few studies have focused on the attainment of maximum speed. There is no consensus on mechanical limitations to sprinting speed, or on optimal training programs to improve speed.

1.3. Concepts and Background

The current lack of solutions to these problems is proposed in the present research to be due in part to the lack of predictive capabilities in most biomechanical models of running.
Running injuries and speed limits are intrinsically related to the internal loading of the body, namely the forces developed actively by muscle and passively by other structures. These forces cannot be measured \textit{in vivo} in most cases, and must be estimated using biomechanical models. Most models depend critically on input sets of experimental data. The popular inverse dynamics model of human locomotion can estimate the resultant internal kinetics of the joints given histories of segment positions and the external forces applied to the body (Elftman, 1939, 1940; Bresler & Frankel, 1950). Internal structural forces can then be estimated using static optimization procedures (Crowninshield & Brand, 1981b; Glitch & Baumann, 1997). However, inverse dynamics models are limited by their inability to predict outcomes from novel conditions for which no experimental data are known, by their sensitivity to the conditioning of the input experimental data (Bisseling & Hof, 2006; Alonso et al., 2007), and by inconsistencies in the dynamical behavior of the model and the source (human) system (Hatze, 2002).

An alternative approach is to use forward dynamics models and dynamic optimization. A forward dynamics model receives as inputs a set of control variables (typically joint torques or muscle excitations) and computes the resulting kinetics and kinematics of the anatomically-inspired model. Dynamic optimization generates simulated movement by solving for the control variables that minimize or maximize a quantity specified by the researcher (Chow & Jacobson, 1971; Davy & Audu, 1987). Forward dynamics simulations do not require input experimental data at each time step, and can be used to answer “what-if” questions that cannot be addressed with inverse dynamics. The lack of dependency on experimental data also eliminates some of the complications associated with human subjects. Issues with fatigue and subject safety are eliminated.
Specific factors that limit the model’s performance can be identified and manipulated. Variables that are time-consuming and complicated to alter in human subjects (e.g. muscle strength) can be quickly altered in the model. If such a model could be validated as a realistic analog of human running capabilities, its predictive nature would be useful for addressing the problems defined earlier.

Forward dynamics modeling and simulation have been used to study human running over the last two decades. Early studies specified initial conditions measured from human runners at heel-strike, but assumed constant muscle activations during the entire simulation and considered only the initial impact phase of ground contact (Gerritsen et al., 1995; Cole et al., 1996a). Later studies generated more realistic running simulations by solving data tracking problems (Wright et al., 1998; Neptune et al., 2000a,b; Thelen et al., 2005a; Chumanov et al., 2007; Miller & Hamill, 2009; Sasaki, 2010). With the data tracking approach, dynamic optimization is used to find the muscle excitation histories that minimize discrepancies between the model’s movement and that of human runners. The tracked data typically include the histories of the joint or segment angles and the ground reaction force.

Tracking problems are useful for studying individual muscle actions and internal loading, but their dependency on available experimental data compromises some of the predictive nature of forward dynamics. With a data tracking approach, predictive simulations of novel conditions can only be made by assuming a strong degree of similarity with a known condition, or by making assumptions about factors that do or do not change when a new condition is presented. Wright et al. (1998), Neptune et al. (2000b), and Miller and Hamill (2009) assumed that muscle activations do not change when running in different shoes. The validity of such assumptions can be difficult to establish. In addition, minimizing
discrepancies between simulated and experimental data may overestimate antagonistic muscle co-activation, and can predict unrealistically high levels of muscle activation and joint contact forces in order to accurately track the data (Neptune et al., 2000a).

An alternative approach to forward dynamics that depends less critically on experimental data in order to generate simulations would be useful for improving our understanding of human running mechanics, and for testing hypotheses related to performance and injury. One such approach is the optimal control or performance-based approach to forward dynamics, which determines the control variables that minimize or maximize a performance criterion (Pontryagin et al., 1965; Chow & Jacobson, 1971). The mathematical nature is identical to a data tracking problem, but rather than quantifying and minimizing the error with a target experimental data set, the performance criterion instead defines the suspected optimality criterion of the movement. This freedom allows researchers to investigate novel or theoretical conditions from which no experimental data are known, since the performance-based approach does not need these data in order to generate a movement. The challenges for the researcher are to define a mathematical model of the human musculoskeletal system that is capable of generating realistic movements, and to define an appropriate optimality criterion that results in realistic movements.

Previous studies have simulated human walking using the performance-based approach. In Anderson and Pandy’s (2001a) simulation, the initial posture of the model was from kinematic measurements of human subjects, and encouraged through penalty functions to finish the simulation in this same position (i.e. perform one stride). Between these initial and final points, the simulation minimized the cost of transport (metabolic energy expended per distance traveled). The resulting kinematics, kinetics, and muscle excitations of the
model were strikingly similar to real human walking, despite not tracking these data at all. The optimality criterion was motivated by evidence that humans optimize their speed and stride parameters to maintain a relatively low rate of cost of transport (Ralston, 1958; Cotes & Meade, 1960). Similar concepts were posed in earlier simulations by Chow and Jacobson (1971) and Davy and Audu (1987), who each used simpler models. Anderson and Pandy’s (2001a) model has subsequently been used to generate a wide-ranging set of novel data on human walking, such as a comparison between static and dynamic predictions of muscle forces (Anderson & Pandy, 2001b) and an examination of how individual muscle contribute to supporting the body weight while walking (Anderson & Pandy, 2003).

Running humans tend to select stride parameters that minimize the amount of metabolic energy used to run at a particular speed (e.g. Gutmann et al., 2006). Performance-based simulations of running may be possible by defining an optimality criterion that considers the rate of energy expenditure. However, there is no evidence that human runners absolutely minimize their energy expenditure rates when running at preferred conditions, and it is not clear exactly what quantity the neuromuscular system prioritizes for minimization. This criterion is not necessarily the cost of transport, as many different quantities could all result in relatively low costs. Unlike walking, the cost of transport in running does not depend heavily on speed (Margaria et al., 1963; Mayhew, 1977), and at fast speeds, anaerobic pathways dominate energy metabolism (Weyand & Bundle, 2005).

1.4. Problem Statement and Purpose

Although the biomechanics of human running have been widely studied, our understanding of injury mechanisms and factors limiting performance is incomplete.
Computer simulations using a performance-based approach could provide new insights into these topics with their ability to predict the outcomes without a strong dependency on experimental data, as outlined previously. However, the performance-based approach has not been widely adopted for running; simulations to date have focused largely on a data tracking approach and have thus been limited in their predictive capabilities.

The purpose of the research was to develop a computer-based mathematical model of the human musculoskeletal system for generating performance-based simulations of running without explicitly tracking templates of experimental data. Optimality criteria based on minimizing quantities related to energy expenditure and maximizing speed were used to generate simulations of running and sprinting, respectively. In the first study (Chapter 3), simulations were generated using a variety of performance criteria. In order to determine the most appropriate criteria for running, the simulated data were compared to data collected from human subjects as they ran in a laboratory. The second study (Chapter 4) presents an application of the sprinting simulation to investigating the speed-limiting effects of the force-velocity relationship. In the third study (Chapter 5), the running simulation was used to determine if age-related differences in running biomechanics can be explained by age-related changes in muscular properties. Chapter 6 discusses the significance of the work.

1.5. Assumptions

In developing the model and performing the simulations, it was assumed that:

1. The salient kinetic and kinematic features of human running occur primarily in the sagittal plane. A two-dimensional model confined to the sagittal plane is appropriate for simulating these features.
(2) The skeleton can be modeled as a set of rigid bodies that do not deform.

(3) The two-component Hill muscle model (Hill, 1938) accurately captures the functional aspects of force generation in skeletal muscles.

(4) Functionally similar muscles can be “lumped” (i.e. combined together) into a single muscle for the purposes of controlling a sagittal plane model.

(5) Neural control of locomotion is represented by an excitation signal sent to the muscle models. The signal is non-specific and thus represents the combined effects of all source of muscle excitation, including motor unit recruitment and rate coding.

(6) Motion of skin-mounted markers reflects the sagittal motions of the underlying skeletal segments with acceptable accuracy.

1.6. Operational Definitions

The following terms were used frequently in this document and are here operationally defined as follows.

• **Running**: locomoting at a submaximal speed with a flight phase, with most of the metabolic energy provided by aerobic pathways.

• **Sprinting**: locomoting at maximum speed with a flight phase, with most of the metabolic energy provided by anaerobic pathways.

• **State variable**: a time-dependent variable that describes the model’s state. Algebraically independent from all other state variables.

• **State space**: the set of all state variables. Specifies all possible states of the model.

• **Control variable**: a variable input to the system, with a value chosen from a permitted range. Example: magnitude and timing of muscle excitation.
• **Parameter:** a quantity that relates the function of the model to its variables.

• **Objective function:** a function of the state variables that returns a scalar value, which is minimized or maximized in a dynamic optimization problem.

• **Optimality criterion:** the suspected goal of the movement that governs the selection of control variables, formulated as a scalar equation.

• **Data tracking approach:** a dynamic optimization problem where the goal is to minimize discrepancies between simulated data and a set of experimental data.

• **Performance-based approach:** a dynamic optimization problem where the goal is to maximize the optimality criterion.

• **Cost of transport:** the metabolic energy consumed per unit distance traveled.

• **Metabolic power:** the metabolic energy consumed per unit time.

1.7. Hypotheses

The present work consists of three studies centered around simulating running motions with the model. The hypotheses of each study are stated here.

**Chapter 3: Optimal criteria for performance-based simulations of human running**

• **Hypothesis 1.1:** Compared to other criteria, an optimality criterion minimizing the cost of transport will result in simulated joint angles and ground reaction forces that most accurately match these same data from running humans.

• **Hypothesis 1.2:** The simulations will be dynamically stable in response to small perturbations to its initial conditions.
Chapter 4: Sensitivity of maximum sprinting speed to characteristic parameters of the muscle force-velocity relationship

- **Hypothesis 2.1:** Increasing the maximum shortening velocity, the eccentric plateau force, or the concentric shape parameter will increase the maximum sprinting speed.
- **Hypothesis 2.2:** The maximum sprinting speed will be most sensitive to the maximum shortening velocity.

Chapter 5: Virtual aging of the muscular system and its effects on running biomechanics

- **Hypothesis 3.1:** Adjusting the muscle model parameters to represent the aging process will induce changes in running biomechanics characteristic of older adults.
- **Hypothesis 3.2:** These adjustments will result in increased loading of the plantarflexors and hamstrings.
- **Hypothesis 3.3:** Strengthening the maximum isometric force of the plantarflexors and hamstrings will reduce their loading in the “older” running simulation.

The hypotheses are largely unchanged from the proposal stage of this dissertation. However, some of the hypotheses are more strongly emphasized than others. For example, hypothesis 1.2 is mentioned only briefly in Chapter 3, and is evaluated extensively in Appendix C.
CHAPTER 2

LITERATURE REVIEW

This chapter reviews the biomechanics of human running. Studies of experiments on human subjects are reviewed first (Section 2.1), followed by studies that synthesized running data using computer models (Section 2.2). The final two sections focus on topics related to the research: biomechanical limitations to maximum sprinting speed, with particular attention to limitations imposed by muscles (Section 2.3), and the biomechanics of running injuries, with an emphasis on injuries to older runners (Section 2.4).

2.1. Experimental Studies

The biomechanics of running have been widely studied by in vivo experimental approaches. Typical patterns of kinematics, kinetics, and EMG have been well documented in a number of review articles (e.g. Vaughan, 1984a; Williams, 1985; Novacheck, 1998) and descriptive studies (e.g. Elftman, 1940; Cavanagh & Lafortune, 1980; Winter, 1983; Cappellini et al., 2006) and will not be repeated here in great detail. Instead, the goals of this section are to review the sensitivity of these variables to adjustments in speed and stride parameters, and to identify biomechanical variables that are optimized when humans run at a given speed using self-selected stride parameters.

2.1.1. Lower Extremity Kinematics

Three basic kinematic descriptors of human locomotion are speed, stride length, and stride frequency. Speed is the rate of displacement of the center of mass in the horizontal
Stride length is the displacement of the center of mass during one stride cycle. Stride frequency is the number of strides completed per unit time. The product of stride length ($SL$) and stride frequency ($SF$) gives the speed $v$:

$$v = SL \cdot SF \quad \text{(Eq. 2.1)}$$

A change in speed can thus be accomplished by a change in stride length, a change in stride frequency, or concurrent changes in both parameters. When speed is constant, increasing stride length by a certain percentage necessarily decreases stride frequency by this same percentage, and vice-versa. Stride length levels off at a moderate speed for most runners and may decline slightly at very fast speeds, while stride frequency continues to increase up until the maximum sprinting speed (Fig. 2.1; Luhtanen & Komi, 1978; Hay, 2002).

Equation 2.1 and Fig. 2.1 indicate that although a given speed can be attained by many combinations of stride length and stride frequency, humans tend to use a particular set of stride parameters to run at a particular speed. This tendency begs the question of why humans use one particular combination when others are theoretically possible. When stride parameters are unrestrained and speed is specified, runners tend to select stride parameters that minimize the metabolic power (Högberg, 1952; Hamill et al., 1995) and the metabolic cost of transport (Gutmann et al., 2006). Metabolic power is the amount of metabolic energy consumed per unit time. The cost of transport (CoT) is the energy consumed per unit distance, or equivalently the metabolic power divided by speed. Margaria et al. (1963) found that the metabolic power is a linear function of running speed, resulting in a CoT that is invariant with respect to speed. This conclusion is widely cited, although it was based on a group of only three subjects and later studies presented contrary evidence (Mayhew, 1977; Steudel-Numbers & Wall-Scheffler, 2009). In any case, the speed-CoT for running is much
shallower than it is for walking (e.g. Ralston, 1958; Fig. 2.2) and it seems clear that humans optimize their stride parameters in order to maintain a low rate of metabolic energy expenditure.

Other kinematic variables are not optimized at a preferred speed or stride parameter set. Shock attenuation between the leg and head, quantified by the frequency domain of segment accelerations, increases with increasing speed (Shorten & Winslow, 1992) and increasing stride length (Derrick et al., 1998). The sagittal ranges of motion and peak angular velocities of the hip, knee, and ankle increase with increasing running speed (Mann & Hagy, 1980; Kivi et al., 2002). Increasing stride length increases joint ranges of motion during stance (Derrick et al., 1998). However, the strength of long-range correlations in the stride duration is minimized at a preferred, comfortable running speed (Jordan et al., 2006). A long-range correlation means that a stride with a particular duration is likely to be followed by another stride of similar duration.

2.1.2. Ground Reaction Forces

The ground reaction force (GRF; centroid of the pressure distribution along the foot) in running has been widely studied. The magnitude of the vertical GRF impact peak increases with increasing running speed (Hamill et al., 1983; Nigg et al., 1987) and increasing stride length (Derrick et al., 1998; Mercer et al., 2005). The magnitude of braking and propelling peak forces and impulses also increase with increasing speed (Hamill et al., 1983; Munro et al., 1987). Running in harder or softer shoes has no consistent effect on GRF peaks (Bates et al., 1983; Nigg et al., 1987), although barefoot runners tend to adopt a flatter footfall pattern that reduces or eliminates the impact peak (Divert et al., 2005).
2.1.3. Joint Moments and Mechanical Energetics

Segment kinematics, GRF, and anthropometric parameters can be combined in a mathematical model to calculate the resultant internal forces and moments at the joints that must have been present for the model to exhibit the segment kinematics. This procedure is called inverse dynamics (Elftman, 1939, Bresler & Frankel, 1950). Further processing can be performed to estimate quantities such as the mechanical power of muscle groups (e.g. Elftman, 1940; Winter, 1983) and the flow of mechanical energy within and between segments (e.g. Robertson & Winter, 1980; Chapman & Caldwell, 1983a).

Peak joint moments and powers in the sagittal plane generally increase with increasing running speed, reflecting the greater mechanical output needed from muscles at faster speeds (Belli et al., 2002). Running with longer strides increases peak joint moments and powers during stance, particularly at the knee (Derrick et al., 1998). The effects of stride length and frequency on swing phase joint kinetics has not been widely studied, but increasing sprinting speed (which is accomplished primarily by increasing stride frequency; Fig. 2.1) increases the peak joint powers and the amount of energy that must be transferred into and out of the lower extremity during swing (Chapman & Caldwell, 1983b). Simple computer models that minimized the peak hip joint power (Yanai & Hay, 2004) or the mechanical work performed by the legs on the center of mass (Srinivasan & Ruina, 2006) predicted manifolds of speed and stride length that resembled those of human runners, but the models neglected many salient features of the human musculoskeletal system, such as muscular dynamics, feet, and knee joints.
2.1.4. Muscle Activity

Electromyographic (EMG) signals have been measured during running using both surface and indwelling electrodes. Mann and Hagy (1980) reported that EMG magnitudes of the quadriceps and hamstrings increased with increasing running speed (jogging to running to sprinting). This trend is also true for most muscles of the lower extremity (Cappellini et al., 2006; Gazendam & Hof, 2007). However, Mero and Komi (1987) reported no changes in EMG magnitudes when sprinting speed was increased to supra-maximal levels. This finding is perhaps expected because the supra-maximal speed was attained using an external towing rig, not by increasing the muscular effort.

EMG responses to changes in stride parameters have not been widely studied. Swanson and Caldwell (2000) compared EMG magnitudes for treadmill running at a level grade and at a 16° incline. Stride frequency increased when running uphill, as did the stance phase EMG magnitudes of gastrocnemius, soleus, rectus femoris, vastus lateralis, and gluteus maximus. When stride frequency was held constant between conditions, the uphill EMG amplitudes were more similar to the level condition, suggesting that at least part of the changes in EMG were due to the change in stride frequency.

EMG is useful for its accessibility, but is limited by a variety of methodological and interpretative complications and does not directly reflect muscle activation or force (Kamen & Caldwell, 1995). Numerous methods for estimating individual muscle forces from EMG have been developed (e.g. Hof & van den Berg, 1981; Lloyd & Besier, 2003) but their sensitivity to running speed and stride parameters has not been presented. Few studies have attempted to more directly quantify human muscle actions during running (or any movement) due to the invasive nature of the techniques. Komi (1990) summarized his laboratory’s
design and implementation of a surgically implanted force transducer for measuring Achilles tendon forces during human movement. Data were presented for a 73-kg male running at a variety of speeds. A peak tendon force of 3750 N was reported during late stance for running at 3.9 m s\(^{-1}\), although a peak force of 6000 N at nearly the same speed was also presented in another figure. No explanation for the discrepancy was given. Peak tendon force increased with increasing speed but plateaued and declined at the fastest speeds (above 6.0 m s\(^{-1}\)). The peak force was greater for forefoot running than for heel-toe running at the same speed.

2.1.5. Upper Body Biomechanics

The great majority of running biomechanics research has focused on the lower extremity and the pelvis. The dynamics of the upper body have been minimally described. The primary role of the arms is to offset the transverse angular momentum of the lower extremities, such that the angular momentum about the body’s center of mass is kept small to avoid hindering progression. This role is independent of speed (Hinrichs, 1987). The arms also provide between 5-10% of the center of mass vertical linear momentum, with the percentage increasing at faster speeds (Hinrichs et al., 1987).

2.1.6. Conclusions on Optimality Criteria

Yanai and Hay (2004) concluded that the selection of stride lengths and frequencies during running has characteristics of a constrained optimization problem. Hamill et al. (1995) argued that if humans are self-optimizing physiological systems, then the minimum cost of some optimality criterion governing the system’s response to given task may be
identified. Based on the review of literature, the only variable that is optimized when humans run with unrestrained stride parameters is the rate of metabolic energy consumption.

Variables with no strong evidence as optimality criteria include joint kinematics and ranges of motion, GRF, joint moments and powers, and EMG signals. These variables are adjusted to run at a new speed or set of stride parameters, but are not minimized when stride parameters are unrestrained.

2.2. Computer Modeling and Simulation

Computer modeling and simulation have become popular tools for studying human motion (Neptune, 2000; Pandy, 2001). Simulations are perhaps most useful for studying variables or conditions that cannot easily be measured on live human subjects. Vaughan (1984b) distinguished computer modeling as the process of developing and validating a model, and computer simulation as the application of the validated model to a research question. A model is a system that is used to represent part of another system. A computer model is a specific type of model that represents the source system as a set of equations that are solved using a computer program.

This section reviews studies that have used computer models to simulate human running, as well as some models of walking. The methods, assumptions, and limitations of the models are emphasized. A goal is to highlight the utility of a simulation approach that depends only minimally on inputs sets of experimental data.

2.2.1. Simple Mechanical Models
The earlier models of human running were in a class known as simple mechanical models. These models consider the body as a series of masses connected by rheological elements such as springs and dampers. The equations of motion are sometimes simple enough to be solved analytically.

The simplest mechanical model of human running is the mass-spring model (Fig. 2.3a). It consists of a point mass representing the body mass, attached to the ground by a massless linear spring. The spring represents the stiffness characteristics of the body during foot contact. Its stiffness is defined as the ratio between the GRF and the displacement of the center of mass. With this definition, the spring stiffness is not constant but changed as the stiffness characteristics of the body change, such as when running at different speeds.

The utility of the mass-spring model as an energy saving mechanism in running was first studied by Cavagna et al. (1977). Upon impact, the spring compresses and stores elastic potential energy. As the spring recoils during push-off, the stored energy is transformed to kinetic energy and returned to the body. With an ideal spring, the energy return is 100% efficient. Mammalian tendons provide similar energy savings, although they are only 90-95% efficient (Biewener & Roberts, 2000; Alexander, 2002).

The mass-spring model has subsequently been used to study body stiffness, mechanical energy, and stride parameters during running. Cavagna et al. (1988) found that when humans run at low speeds, the stride frequency was equal to the natural frequency of the mass-spring system. At faster speeds, the stride frequency was below the natural frequency. They attributed this result to an increase in push-off force at faster speeds, which keeps the stride frequency below the natural frequency to reduce the mechanical power needed to accelerate the limbs. The mass-spring model has revealed that in order to maintain
an economical trajectory of the center of mass, the body stiffness during running increases with increasing speed (McMahon & Cheng, 1990), decreasing stride length (Farley & Gonzalez, 1996), and decreasing surface stiffness (Ferris et al., 1999).

A shortcoming of the mass-spring model is that it cannot simulate the initial impact peak of the vertical GRF that is characteristic of heel-toe running (Fig. 2.4). More complex models are needed to represent this dynamic. Derrick et al. (2000) used a mass-spring-damper model to simulate the stance phase of running. The model consisted of a lower mass attached to the ground by a spring and damper in parallel, and an upper mass attached to the lower mass by another spring (Fig. 2.3b). The lower mass was assigned the mass of the lower extremity and was intended to represent the “effective” mass accelerated during the impact phase (Derrick, 2004). The upper mass was assigned the remainder of the body mass. The spring stiffness were optimized to track the vertical GRF generate by human subjects running at a fixed speed with either 80, 100, or 120% of their preferred stride length. As stride length increased, the stiffness of the upper spring decreased while the stiffness of the lower spring increased. The authors suggested that as stride length increased, the stiffness of the lower extremity increased in order to prevent the limb from collapsing. Chu and Caldwell (2004) later used the mass-spring model to explain inter-subject variations in shock attenuation when running downhill. Good shock attenuators made better use of the damping capabilities of the lower extremity. The authors hypothesized that this difference could be related to a difference in muscle mechanical properties.

Nigg and Liu (1999) used a more complex model (Fig. 2.3c) to investigate why vertical GRF impact peaks do not change when running in harder or softer shoes (Nigg et al., 1987). The stiffness and damping parameters of the foot-ground interface were adjusted to
represent shoes with different midsole hardness. The vertical impact peak could be modulated by adjusting the other stiffness and damping parameters, which abstractly represented the activations and mechanical properties of muscles. However, the loading rate of the vertical impact peak increased with harder shoes regardless of other adjustments. Both findings were consistent with the experimental evidence. In another study using the same model (Liu & Nigg, 2000), the authors found that vertical impact forces were sensitive not only to total body mass, but to the ratios of bone and soft tissue masses.

Simple mechanical models are attractive for their simplicity and versatility. The models have only a handful of components and their equations of motion are easy to formulate and solve. They are capable of accurately representing some salient features of human running, in particular the vertical GRF, and useful insights into the mechanics of running in a much more complicated system (the human body) can be drawn from their output. Since the equations of motion are integrated forward in time when solving them, the models are predictive in nature and do not explicitly require time histories of experimental data to perform simulations.

The simplicity of the models also limits their capabilities. Due to their small number of components, the models are usually limited to making conceptual conclusions on body function as a whole, or on the function of general, abstract components of the body. By a similar argument, the appropriate assignment of parameter values for masses, spring stiffness, and damping rates can be challenging since they often do not represent a well-defined anatomical structure of the body. For example, the linear springs and dampers are often intended to represent the function of muscles, whose activation and contractile dynamics are highly nonlinear (Zajac, 1989).
2.2.2. Musculoskeletal Indeterminacy

A useful outcome from mechanical analyses of human motion is the estimation of individual time-varying muscle forces. Muscle forces provide information on the nervous system’s strategy for performing a movement, and are needed to determine the internal loading environment of the body (Erdemir et al., 2007). Muscle forces cannot be determined solely from the equations of motion of the skeleton because the human musculoskeletal system is mechanically indeterminate. At the hip, for example, 22 muscles span the joint, but it has only six degrees of freedom. Similar indeterminacies are present at the other joints.

This indeterminacy problem, which is referred to as the “force sharing” or “general distribution” problem, has been solved using a variety of musculoskeletal modeling techniques (Crowninshield & Brand, 1981a). Two of the most popular techniques in recent years have been static optimization, an extension of inverse dynamics, and dynamic optimization, an extension of forward dynamics.

2.2.3. Static Optimization

A static optimization model receives as inputs the joint moments at an instant in time and outputs the individual muscle forces that minimize a user-defined cost function while replicating the input joint moments. A model of musculoskeletal geometry is therefore also needed in order to transform the muscle forces into joint moments: the product of the muscle force and its moment arm, summed over all muscles, equals the joint moment. The “static” part of the name refers to the fact that the solution is determined for each time step without explicit dependency on solutions at other time steps (Hardt, 1978). Despite this
independence, muscle force histories predicted for locomotion tend to be smooth and continuous (Crowninshield & Brand, 1981b; Glitsch & Baumann, 1997).

The most widely recognized static optimization study of human locomotion is undoubtedly Crowninshield and Brand (1981b). The study was not the first to predict muscle forces during walking (Seireg & Arvikar, 1975), nor was it the first to do so with a nonlinear cost function (Pediotti et al., 1978). It was the first, however, to support the choice of cost function (minimization of the sum of cubed muscle stresses) with a strong physiological basis. The predicted muscle force histories compared favorably with the amplitudes of EMG linear envelopes, and the predicted hip joint contact force was close to in vivo measurements from an instrumented hip replacement. To date, these were the most realistic predictions of muscle forces during human walking.

Glitsch and Baumann (1997) were the first to estimate muscle forces during running using static optimization, although their experimental data were somewhat atypical (a single subject running at 5.0 m s\(^{-1}\) using a forefoot strike pattern). The authors used a full 3D musculoskeletal model of the pelvis and lower extremity with 47 muscles and investigated the sensitivity of solutions to (a) the form of the cost function and (b) the degrees of rotational freedom allowed at the joints. They concluded that minimizing the sum of squared muscle stresses while modeling the hip as a spherical joint and the knee and ankle as “intermediate” joints (a non-specific geometry somewhere between two and three degrees of freedom) produced the most realistic results, although no quantitative criteria were defined to support this conclusion.

More recent studies used static optimization to predict the loading of the skeleton during running. By making assumptions regarding the magnitude of ligament forces and the
moment arms of bone contact forces, the bone contact force vectors can be calculated if the muscle and resultant joint forces are known (Crowninshield & Brand, 1981a). Sasimontonkul et al. (2007) predicted bone contact forces on the distal tibia, a common site of tibial stress fractures, during the stance phase of running at an average speed of 3.75 m s$^{-1}$. The peak compressive and shear forces averaged 9.0 and 0.6 times body weight. Compressive forces were primarily due to soleus and gastrocnemius forces. Shear forces were due primarily to internal (muscle) forces in early and late stance and to the external joint reaction force in mid-stance. The shear components of muscle forces acted in the same direction as the net shear joint reaction force, which reduced the magnitude of the tibial shear due to bone contact. Edwards et al. (2008, 2009) combined static optimization with finite element modeling to investigate locations of stress concentrations on the femur and the effects of stride length on tibial stress fracture potential. Locations of high femoral stresses corresponded to areas where runners most often sustain stress fractures. Running with a stride length 10% shorter than preferred reduced the estimated probability of incurring a tibial stress fracture by 3-6%.

Yokozawa et al. (2007) used static optimization to predict muscle activations for six subjects running on level ground and uphill at 3.3, 4.2 and 5.0 m s$^{-1}$. Their study was the first to simulate a full stride of running using static optimization. They also included muscle contractile dynamics by using a Hill-based muscle model with force-length-velocity relationships as well as series and passive elastic properties. Most previous studies have modeled the muscles as ideal force generators that neglect these features of muscle. The cost function was the sum of cubed muscle activations. Most of the predicted muscle activations compared well with EMG linear envelopes. Muscle activations increased when running
uphill, which may explain experimental observations that the required metabolic power increases when running uphill. The authors also proposed a special role for rectus femoris to support the forward tilt of the pelvis when running uphill.

The strengths of static optimization are its computational speed and the ease of implementing a complex musculoskeletal model. Unlike simple mechanical models, no forward integration is involved in performing the simulations, and even a 3D model with dozens of muscles can be solved on a single CPU in well under an hour. However, static optimization has also been criticized on a number of fronts. There is considerable debate on whether the popular cost function of minimizing muscle stresses is appropriate for particular movements (e.g. Prilutsky, 2000). Validating the predicted muscle forces quantitatively is difficult, as EMG provides at best a qualitative comparison. Further validation is generally not possible, as a “gold standard” in the form of in vivo human muscle forces are usually not available (although this weakness is true of any method for muscle force prediction). Prilutsky et al. (1997) compared static optimization predictions to plantarflexor muscle forces in cats measured using tendon strain gauges. No cost function predicted muscle forces with average errors under 26% of the measured forces. Finally, since joint moments calculated by inverse dynamics are needed as inputs, the output from static optimization models depends critically on the acquisition and processing of experimental data (Patriarco et al., 1981; Davy & Audu, 1987) and the definition of the musculoskeletal model (Glitch & Baumann, 1997; Hatze, 2002), and the models can only be applied to further analysis of experimentally recorded movements. They cannot be used to predict outcomes from conditions for which no experimental data are known.
2.2.4. Forward Dynamics

Forward dynamics is essentially inverse dynamics in reverse: a mathematical model is defined, some variables that result in joint moments are input, and the resulting kinematics are output. Like inverse dynamics and static optimization, forward dynamics requires a mathematical model of the musculoskeletal system. The nervous system is often included as well. However, forward dynamics lacks many of the limitations (but also most of the strengths) of inverse dynamics and static optimization. Some important terms in forward dynamics are *state equations*, *state variables*, and *control variables*. The state equations define the model’s dynamics, such as the equations of skeletal motion. The state variables are the dependent variables of the state equations, and the control variables are the independent variables. To perform simulations, values for the control variables are input and the state equations are solved for the state variables. The equations are usually too complex to solve analytically, so approximate solutions are determined numerically. The control variables can consist of joint moments, muscle forces, or muscle excitations. The control variables do not have to be taken from experimental data; any arbitrary signal can be input as a control and the resulting kinematics calculated. The central task for the researcher is to determine control variables that produce realistic movements.

Walking has been studied using forward dynamics models since the early 1970s (Chow & Jacobson, 1971). Running has received comparatively less attention, with more recent origins. Taga et al. (1991) were the first to simulate running with a forward dynamics model, although the study focused more heavily on walking. The model (Fig. 2.5a) was 2D and had five segments (trunk and bilateral thighs and legs) with no feet and no individual muscles. The joints were actuated by moments developed by a neural network whose
dynamics were coupled to those of the skeletal system via terms representing feedback from the peripheral nervous system. The parameters of the neural network were manipulated until the model exhibited a stable gait that resembled human walking over multiple strides. When a single parameter controlling the scaling and frequency of neural output was increased, the model transitioned to a run (identified by the emergence of a flight phase) at 1.9 m s$^{-1}$, which is close to the speed at which running is less metabolically costly than walking in humans (Hreljac, 1993).

The first forward dynamics simulations that focused specifically on human running used 2D, one-legged models and simulated the early impact phase of stance (Gerritsen et al., 1995; Cole et al., 1996a). The limitations to early stance was motivated by suspected links between the impact phase and running injuries. Gerritsen et al. (1995) used the model shown in Fig. 2.5b to determine the effects of the initial kinematic state and initial muscle activation levels on peak impact forces. Muscle activations were assumed to be constant across the entire 40-ms impact phase. The authors performed simulations with thousands of combinations of initial activations that all produced initial joint moments equaling those calculated inversely from human subjects. Regardless of the initial muscle activations, the simulated vertical GRF impact peak varied by only 10% of the model’s body weight. However, when the joint moment constraint was removed and all muscles were maximally activated, the impact peak rose to 600% of the body weight. Sensitivity analyses of the kinematics revealed that the vertical impact peak was most sensitive to the initial vertical velocity of the model at heel-strike. The results suggest that antagonistic muscle co-activation has little effect on impact forces compared to resultant joint moment magnitudes and the vertical velocity at impact.
Cole et al. (1996a) used a similar model and simulations to further investigate the effects of muscles on vertical GRF impact peaks. Simulations were performed with three models: one with no muscles and rigid body segments, a second with muscles but still rigid segments, and a third with muscles and non-rigid segments. The non-rigid model had wobbling masses that could oscillate along the muscle lines of action. Adding muscles to the rigid body model reduced the impulses of bone contact forces at the hip, knee, and ankle, while adding wobbling masses further reduced the impulses (Fig. 2.6). The results suggest that muscle activity and soft tissue vibrations can attenuate joint loading, and may explain why runners do not have an elevated risk of knee osteoarthritis (Chakravarty et al., 2008).

2.2.5. Data Tracking Simulations

Gerritsen et al. (1995) and Cole et al. (1996a) made assumptions about muscle activity that limited the accuracy of their simulations. Specifically, they assumed muscle excitation and activation levels were constant during the impact phase. Calculations of joint moments (Winter, 1983) and measurements of EMG signals (Gazendam & Hof, 2007) suggest this assumption may be erroneous. It also prevents the simulation of a full stance phase or gait cycle, during which muscle activations are certainly not constant. Consequently, some of their results were not characteristic of data from real human runners. Most noticeably, the simulated vertical GRF did not have the same time course as experimental data, and the magnitudes of the impact peaks in Cole et al. (1996a) were about 30% larger than the mean impact peak typically seen in experimental studies.

Later studies generated more realistic simulations of running using dynamic optimization. This procedure finds the control variables that minimize a user-defined
objective function. Wright et al. (1998) were the first to simulate human running using dynamic optimization. Their goal was to determine if the invariance of vertical GRF impact peaks to shoe midsole hardness (Nigg et al., 1987) could be explained by passive changes in muscle forces. Muscle excitations were optimized using a data tracking approach where the objective function was the sum of the mean square errors between simulated and experimental joint angles and GRF. The optimization was performed with the parameters of the foot-ground interface set to represent a “soft” running shoe. The optimization was performed with nine different sets of initial kinematics, and each of these nine simulations was then repeated with the muscle excitations held constant, but the shoe parameters adjusted to represent a “hard” shoe.

The simulated impact peaks did not differ consistently between the soft and hard shoe conditions and on average were the same. The vertical GRF loading rate increased in the harder shoe for all nine simulations. For all nine simulations, peak force in the tibialis anterior muscle increased and peak force in the peroneal muscle decreased, but changes in the peak hamstrings force were inconsistent. Since the muscle excitations were the same for both shoe conditions, the changes in peak muscle forces were attributed to changes in the model’s kinematic state induced by the harder shoe, namely an increase in knee flexion velocity. The authors concluded that impact forces during running can be regulated by passive changes in muscle forces (i.e. changes in a muscle’s kinematic state with no change in its activation). Miller and Hamill (2009) used a similar approach to investigate the effects of shoe midsole hardness on impact forces and the internal loading of the ankle joint. They concluded that changes in internal musculoskeletal loading when running in different shoes are not reflected by the magnitude of external impact forces. Running in harder shoes
increased the peak compressive and shear ankle joint contact forces in subject-specific simulations of a male and a female runner, but changes in the vertical GRF impact peak were inconsistent between the two. The authors suggested that relationships between internal loading, external loading, and footwear should be defined on a subject-specific basis.

Neptune et al. (2000a) extended the model and data tracking approach of Wright et al. (1998) to a simulation of the entire stance phase. After validating the model’s ability to accurately track experimental GRF and joint angles with realistic muscle excitation patterns, the authors applied the model to studying the effects of training interventions on the shearing force within the patellofemoral joint, a suspected force of knee pain in runners (Neptune et al., 2000b). Nine simulations of the stance phase were generated by tracking the kinematics of nine different subjects running at 4 m s$^{-1}$, although the same generic model was used for all nine simulations. Simulations were then repeated with the model’s parameters adjusted to represent three common training interventions for treating patellofemoral pain: a 10% increase in vastus medialis strength, a 5-ms delay in the onset timing of vastus medialis excitation, and a medially arched shoe orthotic insert. The insert was modeled by increasing the stiffness of the medial ground contact elements. All three interventions reduced the peak patellofemoral contact force by up to 35 N and decreased the average force by up to 18 N. Further, these reductions were additive when simulations were repeated with multiple simultaneous interventions. The results provided quantitative evidence on the effects of frequently prescribed interventions for a common running injury.

Sasaki (2010) recently generated data tracking simulation of running using a 2D forward dynamics model, at a fairly slow speed (2.4 m s$^{-1}$, an 11.2-minute mile pace), to determine contributions of individual muscles to the axial knee joint contact force. The vasti
muscles accounted for most of the joint contact force in first half of stance, while gastrocnemius and hamstrings made the greatest contribution in the second half of stance. Gluteus maximus and soleus, which do not cross the knee, made secondary contributions through their effects on the GRF.

Other researchers have used dynamic optimization to simulate human sprinting. Thelen et al. (2005a) generated a 3D data tracking simulation of the swing phase of sprinting to study the mechanics of the hamstrings, a site of frequent injury in sprint athletes. The model included muscles involved in hip flexion/extension, hip ab/adduction, and knee flexion/extension. A procedure called computed muscle control was used to determine the muscle activations that minimized the tracking error between the simulated histories of these joint angles and experimental data from an adult male sprinting on a treadmill at speeds from 7.9-9.3 m s⁻¹. This procedure assumes that joint moments are distributed into individual muscle forces by minimizing a function of the muscle activations at each time step, similar to static optimization, but greatly enhances the computational speed of forward dynamics (Thelen et al., 2003). The analysis focused on the biceps femoris muscle model. Most of the changes in length of this muscle during swing were accounted for by eccentric lengthening of its contractile component, which generated large strains. Speed had no effect on peak biceps femoris strain, although the peak biceps femoris force rose from 934 N to 1195 N between the slowest and fastest speeds. Similar to Neptune et al. (2000b), the simulations provided quantitative data on muscle mechanics at a common site of injury that could not be determined purely from experiments on human subjects. Chumanov et al. (2007) used the same model and simulation approach to determine the contributions of individual muscles on hamstring mechanics during swing. Peak biceps femoris strain was more sensitive to small
changes in the force of lumbo-pelvic muscles than changes in knee and ankle muscle forces. Changes in hip flexor forces induced large changes in biceps femoris strain (up to 3.7 mm per N of force) on the contralateral leg. The authors interpreted the results as evidence that hamstring train and rehabilitation programs should focus on strengthening the lumbo-pelvic muscles. A follow-up study (Chumanov et al., 2010) included the stance phase in order to address the question of whether sprinters are more injury prone in stance or swing. Peak hamstrings strains occurred during swing and were invariant with respect to speed, although peak biceps femoris force increased with increasing speed. The hamstrings muscles performing negative mechanical work only during the swing phase. The authors concluded that hamstrings mechanics are more likely to cause injuries in swing than in stance.

The eight simulation studies cited here comprise the bulk of dynamic optimization studies of human running. Other studies investigated muscle function during running near the preferred gait transition speed (e.g. Sasaki & Neptune, 2006), but this is an unusually slow speed that humans do not often run at. The simulations as a whole have provided unique insights into the muscle mechanics of human running that could not be gained purely from experimental measurements on human runners. However, in each study a data-tracking problem was solved in order to generate the simulations. This simulation approach limits the predictive nature of forward dynamics by constraining the model to follow a particular movement pattern. Consequently, the data tracking approach cannot be used to study conditions for which no experimental data are known.

To simulate conditions for which the movement pattern is unknown a priori, assumptions must be made about the kinematics, kinetics, and muscle activity of the unknown condition in relation to a measured condition. For example, Neptune et al. (2000b)
assumed that changes in muscle strength, excitation timing, and orthotic inserts do not alter muscle excitation patterns during stance. Wright et al. (1998) and Miller and Hamill (2009) assumed that running in shoes with different midsole hardness does not alter muscle excitations during impacts. Thelen et al. (2005a) and Chumanov et al. (2007, 2010) made an assumption on how joint moments are distributed into individual muscles based on a minimization criterion. The validity of these assumptions can be difficult to establish. Consequently, a data tracking approach to forward dynamics simulations is not well suited to studying “what if?” questions where the movement pattern and muscle excitation strategy are unknown and may differ from a measured condition.

2.2.6. Performance-Based Simulations

An alternative approach to forward dynamics simulations that does not depend on solving a data-tracking problem is to define an objective function that formulates the suspected motor objective, or optimality criterion, of the movement. Even though no experimental data are explicitly tracked with this approach, the model should still move like a human as long as two criteria are met:

1. The model appropriately represents the neural, muscular, and skeletal systems
2. The optimality criterion appropriately defines the nervous system’s criterion for activating particular muscles during the movement in question.

Simulations of this type have been referred to by many names, and in this work will be referred to as performance-based simulations. This type of simulation can be used to investigate “what if?” conditions without a strong dependency on experimental data, because
these data are not entered into the objective function. However, experimental data are still useful for validating the simulation results.

Performance-based simulations have been widely used to study jumping, since the movement presents a relatively unambiguous optimality criterion of maximizing the peak height (Pandy et al., 1990; van Soest et al., 1993; Selbie & Caldwell, 1996). It has also been used to simulate sprint cycling (van Soest & Casius, 2000) and walking (Chow & Jacobson, 1971; Anderson & Pandy, 2001a; Hase & Yamazaki, 2002; Sellers et al., 2003; Ackermann & van den Bogert, 2010; Umberger, 2010) but has not often been extended to running. Sellers and Manning (2007) simulated sprinting in 2D musculoskeletal models of a variety of extinct and extant animals, including humans, by finding the muscle activations that maximized the horizontal center of mass displacement over three seconds. The human simulation achieved a maximum speed of 7.9 m s\(^{-1}\), which is comparable to sub-elite human subjects, but the segments kinematics, GRF, and muscle activations were not compared to data from human sprinters. The model included no biarticular muscles or activation dynamics, which could be expected to have an effect on sprinting efforts. The authors performed a sensitivity analysis between the muscle strength and the maximum sprinting speed, although data were only presented for the tyrannosaurus rex simulation.

Hase and Yokoi (2002) simulated running at a range of speeds between 3.0-5.5 m s\(^{-1}\) using a 3D model with 60 muscles. The controller was a neural network representing a central pattern generator with peripheral feedback that controlled each degree of freedom, similar to Taga et al. (1991). Simulations were generated by optimizing the neural network parameters to maximizing a complex optimality criterion that included the cost of transport, the loading rate of contractile tissues, speed, and the duration of stance. A simulation that
only maximized speed was also performed. Since the neural network output generated net joint moments rather than muscle forces, the individual muscle forces were determined using static optimization at each time step. The model was able to simulate at least seven consecutive steps at a speed of 4.0 m s\(^{-1}\) due to the peripheral feedback terms included in its control, but the simulation results were not compared to data from human runners, and the model achieved a relatively slow maximum speed of only 5.5 m s\(^{-1}\).

Recently, Sellers et al. (2010) tested the effect of tendon compliance on the speed and metabolic cost of running using performance-based simulations. The model was taken from the AnyBody project at Aalborg University. The model itself was 3D and had 70 muscles with the energetics model of Minetti and Alexander (1997), but the joint motions were restricted such that segments could only move in the sagittal plane. Two simulations were generated: one that maximized the average speed, and one that minimized the cost of transport. The second simulation was repeated with the tendon stiffness increased 100-fold for all muscles, for the Achilles tendon, and for all muscles but the Achilles tendon. The sprinting simulation achieved a speed of 5.6 m s\(^{-1}\), while the energy-minimizing simulation ran at 4.0 m s\(^{-1}\) with a cost of transport of 1.3 J m\(^{-1}\) kg\(^{-1}\). Stiffening all the tendons, only the Achilles tendon, and all tendons but the Achilles raised the cost of transport to 4.1, 3.4, and 2.5 J m\(^{-1}\) kg\(^{-1}\), respectively. The results demonstrated the important role of tendon compliance in economical locomotion, although the nominal cost of transport (1.3 J m\(^{-1}\) kg\(^{-1}\)) was far below the cost of transport for human running (around 3-5 J m\(^{-1}\) kg\(^{-1}\); Mayhew, 1977).

The greatest limitations of the three noted performance-based running simulations were (1) none of them compared the simulated kinematics, GRF, and muscle excitations to
experimental data from human runners, and (2) all assumed the appropriateness of a single optimality criterion, without posing a rationale for this criterion and without testing other potential criteria. The review of literature found no additional studies that generated performance-based simulations of human running with musculoskeletal models.

Despite their limitations, the performance-based running simulations highlight the utility of this simulation approach for investigating aspects of human movement that are difficult to study with a purely experimental approach. The lack of dependency on experimental data for tracking purposes endows these simulations with considerable prediction power. For example, the sprinting simulations of Sellers and Manning (2007) presented data on (1) the sensitivity of maximum sprinting speed to changes in muscle mass and strength, and (2) the maximum running speeds of extinct dinosaurs. Item #1 would be difficult to study in human subjects due to difficulties in quantifying muscle mechanical properties in vivo, and would require a time-consuming longitudinal study that would be difficult to control. Item #1 could not be studied by data tracking or static optimization approaches since no experimental data exist for the conditions with altered muscle mass. Item #2 is obviously impossible to study by any other approach, beyond estimates from anthropometry or time travel.

2.3. Limitations to Maximum Speed

Maximizing human sprinting potential is a topic of interest for athletes and coaches as well as biomechanists. Sprinting has been widely studied (Mero et al., 1992; van Ingen Schenau et al., 1994) with some studies focusing on limits to maximum speed. The purpose
of this section is to summarize the base of knowledge on biomechanical limits to maximum human sprinting speed.

2.3.1. Predicting Maximum Speed

Although elite human sprint performances continue to improve (Fig. 2.7), all runners exhibit similar relationships between speed and time during a sprint, although the time to accelerate from a dead stop to top speed varies among individuals and is a major factor in determining race success. In one of the earliest studies of sprinting, Furusawa et al. (1927) measured the position of a runner during a maximum-effort 200-yard sprint using a series of magnetic timing gates. The velocity-time data were fit well by an exponential function:

$$v(t) = fga \left(1 - e^{-\frac{t}{a}}\right)$$

(Eq. 2.2)

where $v(t)$ is the instantaneous velocity at time $t$, and $g$ is the gravitational acceleration. $f$ and $a$ were curve-fitting constants. The authors suggested that $f$ was a dimensionless metric of the runner’s build, strength, skill, and fitness, and that $a$ was a measure of physiological effort per unit distance traveled. Vaughan (1983a,b) used a similar approach to develop an equation of motion for sprinting in the horizontal direction and suggested the model could be used to pinpoint strengths and weaknesses throughout a run.

The average speed of a run decays exponentially with the duration of the run. Elite sprinters run much faster than elite milers, but elite milers do not run much faster than elite marathoners. This relationship is a function of the pathways for aerobic and anaerobic energy metabolism used predominately in longer and shorter runs (Bundle et al., 2003):

$$\bar{v}(d) = v_{aer} + (v_{an} - v_{aer})e^{-kd}$$
where $\bar{v}(d)$ is the average speed during a run of duration $d$, $v_{aer}$ and $v_{an}$ are the maximum speeds that can be supported by aerobic and anaerobic metabolism, and $k$ is a constant.

2.3.2. Stride Parameters

The equations of Furusawa et al. (1927), Vaughan (1983a,b), and Bundle et al. (2003) are useful for predicting and assessing sprint performance, but do not provide many insights into specific factors that limit maximum speed. The stride parameters measured by Luhtanen and Komi (1979) and Hay (2002) suggest that maximum speed is limited by the maximum stride length, and ultimately by the maximum stride frequency (recall Fig. 2.1).

Maximum stride length is dictated primarily by stature and leg length (Alexander, 1984), which can be altered in maturation but not by training. The force-length relationship of skeletal muscle (Ramsey & Street, 1940; Gordon et al., 1966) may also affect the maximum stride length by limiting muscular force during long strides, but there is little evidence in the muscle mechanics and strength and conditioning literature that the force-length relationship can be altered in long-term training. Herzog et al. (1991) compared the moment-length relationships of rectus femoris between sprint and endurance athletes and found that the groups had diametrically shaped moment-length profiles (sprinters were strongest at short lengths, while the endurance athletes were strongest at long lengths), but it was unclear if this difference was inherent to the athletes or induced by training.

Stride frequency, the rate at which the legs move through the stride cycle, is intuitively related to the muscle force-velocity relationship (Hill, 1938; Wilkie, 1950). Increased cycle rates necessitate faster changes in muscle length, and require muscles to develop sufficient forces as they lengthen and/or shorten at faster velocities. There is
convincing evidence that the shape of the force-velocity relationship can be altered by training (Fitts & Widrick, 1996; Andersen et al., 2005; Malisoux et al., 2006), and faster athletes tend to have greater proportions of the fast-fatigable (glycolytic) type of muscle fibers than slower athletes (Costill et al., 1976). Consequently, the force-velocity relationship has been proposed as a primary, and perhaps the primary, limiting factor of maximum human sprinting speed.

2.3.3. Muscle Force-Velocity Relationship

The force-velocity relationship of skeletal muscle was first studied by Hill (1938), Katz (1939), Fenn and Marsh (1935), and Wilkie (1950). The relationship states that the force developed by a muscle’s contractile elements decays to zero as the shortening velocity increases, and increases to a plateau force as the lengthening velocity increases (Fig. 2.8). These two sides of the force-velocity relationship are often referred to as the concentric and eccentric limbs, respectively.

Before the mechanics and mathematical character of the force-velocity relationship were well understood, Furusawa et al. (1927) used the term “muscle viscosity” in reference to the viscoelastic resistance of muscles to stretch. The authors derived an equation of sprint performance (Eq. 2.2) by assuming that muscle viscosity was the primary source of resistance that a sprinter must overcome. Since the equation fit the experimental data well, the authors concluded that muscle viscosity is the primary limiting factor in maximum sprinting speed. However, A.V. Hill’s later work (Hill, 1938) showed that this initial concept of muscle viscosity was flawed; muscle does not behave viscoelastically as a simple dashpot. Using early motion capture techniques, Fenn (1930a) calculated the kinetic and potential
energies of the arms and legs during sprinting. He concluded that the external resistances to running, namely the work performed against gravity and against the ground, are not negligible compared to internal resistance as Furusawa et al. (1927) had assumed. In his next study, Fenn (1930b) used one of the first force platforms to demonstrate that the horizontal force exerted by a sprinter against the ground is not constant. This finding was contrary to another assumption made by Furusawa et al. (1927).

Research on biomechanical limitations to maximum sprinting speed was then relatively dormant for decades. Over 50 years later, Chapman and Caldwell (1983b) studies the mechanical energetics of the swing limb as a potential limiting factor in maximum speed. Kinematic data were recorded in two dimensions from an international-level competitor while sprinting on a treadmill at five speeds from 6.7-9.5 m s\(^{-1}\). The internal forces, moments, powers, and energy transfers of the swinging limb were calculated using inverse dynamics (Chapman & Caldwell, 1983a). The stance phase was neglected because GRF were not available. The total mechanical energy of the swing limb increased with speed initially but plateaued at the two fastest speeds. During the last quarter of swing, the eccentric muscle power of the knee reduced the kinetic energy of the leg to prepare the leg and foot for ground contact. The eccentric peak of the knee muscle power increased with speed, but also plateaued at the two fastest speeds. The authors described this finding as an energy management problem, with the knee flexors needing to remove the kinetic energy of the limb that was generated earlier in swing by the hip flexors. They implicated the force-velocity relationship in the energetics results at the knee, which suggests that the knee flexors were operating near the eccentric plateau at the two fastest speeds. This suggestion was supported by the stride lengths and frequencies used by the subject to run at the fastest
speeds. Between the second-fastest and fastest speeds, the stride frequency decreased slightly, suggesting that the subject had reached her fastest possible stride frequency at the second-fastest speed. The fastest speed was achieved by a small increase in stride length.

Surprisingly, these four studies, the latest of which was published 27 years ago, comprise the bulk of experimental evidence on biomechanical limitations to maximum sprinting speed. In a review article, van Ingen Schenau et al. (1994) concluded that maximum speed is limited by ability of the hip flexor muscles to rotate the legs forward at faster and faster rates. However, they presented no experimental evidence to support this statement.

2.3.4. Ground Reaction Forces

The magnitude of ground reaction forces (GRF) generally increases with increasing running speed (Hamill et al., 1983; Nigg et al., 1987), although the peak vertical force levels off at faster sprinting speeds (Kuitunen et al., 2002). Weyand et al. (2000) reported that the average vertical GRF magnitude, scaled by body mass, was significantly greater for runners with faster top speeds. The absolute duration of swing was similar between runners of all sprinting ability levels. The authors concluded that faster top speeds are achieved by generating greater vertical GRF forces, not by swinging the legs more rapidly. A more recent study reached a similar conclusion, but added that the ability to generate sufficient vertical forces is limited by the available ground contact time, not by the isometric strength of the lower limb muscles (Weyand et al., 2010). However, no range of motion data were reported (faster runners did have longer absolute stride lengths), and the possibility that the faster runners moved through greater ranges of motion in the same time as slower runners
cannot be ruled out. The authors proposed that the major factor by which the force-velocity relationship limits maximum sprinting speed is the rate at which muscles can develop a large vertical GRF to support the body during stance.

2.3.5. Summary of Speed Limitations

In summary, the theory that maximum sprinting speed is limited by the force-velocity relationship of skeletal muscle has a large volume of support in the biomechanics literature. The limiting nature of the relationship manifests itself in the abilities to generate sufficient muscle powers and/or vertical support forces at high stride frequencies. Theories of particular muscle groups, namely the hip and knee flexors, which limit speed through their force-velocity relationships, have been proposed. To better understand limitations to maximum sprinting speed, a greater understanding of muscle mechanics during sprinting, in particular the kinematic states of particular muscles during particular periods of the stride, as well as the sensitivity of maximum speed to changes in the force-velocity relationship, are needed.

2.4. Running Injuries

Injuries are one of the most widely studied topics in running biomechanics. The growth in popularity of running injury research was driven in part by the “running boom” of the 1970s and ‘80s, when many new runners, some of whom were woefully out of shape, began to develop injuries. Injuries were no longer just a concern for elite track or marathon competitors. Consequently, running research began to shift from sport science and analyses of elite performers (e.g. Mann & Herman, 1985; Williams et al., 1987) to clinical orthopedics
and analyses of recreational runners (e.g. Heiderscheit et al., 2002; Milner et al., 2006). Despite this volume of research, the reported incidence rates of running injuries since the 1980s has not declined (Fig. 2.9).

Most running injuries arise internally from mechanical trauma to musculoskeletal structures. Neural injuries have been documented but are quite rare (McKean, 2008). Hreljac (2004) stated that all running injuries can be attributed to training errors. A biological tissue that is stressed beyond a particular threshold sustains mechanical damage. If the tissue is allowed sufficient recovery (time and nutrition) before it is stressed again, it will adapt to the damage and become stronger, such that a greater stress stimulus is needed to incur the same degree of damage (Wolff’s Law for bone; Davis’s Law for soft tissues). If recovery is insufficient for adaptation, the tissue will accumulate damage and an injury will eventually develop. Running injuries can be described from this perspective by a stress magnitude-frequency relationship (Fig. 2.10): high magnitude stresses can be sustained if they are applied infrequently, while high frequency stresses can be sustained if they are of low magnitude. Stressing the body at relatively low magnitudes and frequencies leads to positive adaptation, while stresses too great in magnitude and/or too high in frequency lead to injury. Nigg and Bobbert (1990) argued that comparisons between different loading conditions can potentially reduce the frequency of running injuries. To this end, biomechanical variables related to the internal and external loading of the body have been widely studied in relation to the etiology, treatment, and prevention of running injuries. The variables that have received the most attention are the vertical impact peak of the GRF, peak joint angles, and muscular strength.
2.4.1. Vertical Impact Peak

The vertical impact peak (Fig. 2.4, first peak) is often suspected to cause running injuries due to its large magnitude in relation to the body weight, as well as its rapid loading rate. The impact peak is sometimes called a “passive” peak because it occurs too quickly after heel-strike for an active muscle response to absorb the impact force, which must then be attenuated by passive internal structures. This theory was first posed as an injury mechanism by James et al. (1978). Animal models have demonstrated an association between increased impact peak magnitudes and incidence rates of knee osteoarthritis (Radin et al., 1982). Consequently, many running shoes are designed with cushioned midsoles intended to protect the musculoskeletal system from harmful impact forces.

The definition of a passive impact peak is flawed because it does not consider the possibility of anticipatory muscle activity prior to the impact phase (Bobbert et al., 1992). In human runners, there is no consistent relationship between shoe midsole hardness and the vertical impact peak (Bates et al., 1983; Nigg et al., 1987). Nigg et al. (1995) noted that most evidence linking impact forces and injuries is either speculative or inferred from animal models. Further, no study has established a causal relationship between impact forces and running injuries. A series of five studies at Wake Forest University, published over 12 years, compared GRF peaks between hundreds of runners with particular injuries and healthy runners with no injuries. The magnitude of the impact peak did not differ significantly between the healthy and injured runners in any of these studies (Messier & Pittala, 1988; Messier et al., 1991, 1995; McCrory et al., 1999; Duffey et al., 2000). Studies of runners with and without a history of tibial stress fractures also showed no association impact peak magnitudes and injury (Crossley et al., 1999; Bennell et al., 2004; Milner et al., 2006).
The stress fracture and Wake Forest studies were retrospective designs and thus could not infer causal relationships between their outcome variables and injuries. Most running injury studies have used this type of design, but the few published prospective studies on running injuries have also shown no difference in impact peaks between healthy and injury-prone runners (Noehren et al., 2007; Willems et al., 2007). In an inverse dynamics modeling study Scott and Winter (1990) found that estimates of peak forces at chronic running injury sites were insensitive to changes in the magnitude of the impact peak. A review of literature found only one study (retrospective design) where runners with a history of injuries had greater impact peaks than lifetime injury-free runners (Hreljac et al., 2000). In clinical studies, runners do not have high rates of knee osteoarthritis compared to non-runners, despite experiencing thousands of impacts on a consistent basis (Lahr, 1996; Shrier, 2004; Cymet & Sinkov, 2006; Chakravarty et al., 2008). Most of these studies also controlled for a selection bias (non-runners who stopped running due to knee pain). Based on the current biomechanical and clinical evidence, impact forces in excess of twice the body weight are not necessarily detrimental to the musculoskeletal health of a runner.

2.4.2. Joint Kinematics

At the extremes of a joint’s range of motion, a large passive resistive moment develops due to the stretching of muscles and ligaments and contact forces between boney prominences. Runners who experience greater peak joint angles during the stride may therefore be at greater risk for a passive tissue strain or bone contact injury.

In contrast to studies of the vertical impact peak, peak joint angles have consistently distinguished between healthy and injury-prone runners, particular in the frontal and
transverse planes. The retrospective Wake Forest studies consistently reported greater maximum ankle pronation velocities in the injured cohorts, although the actual pronation of the subtalar joint could not be measured with the kinematic model used.

The joint actions of ankle pronation and knee internal rotation are mechanically coupled during closed-chain motion due to the oblique orientation of the subtalar joint. Knee internal rotation is often suspected as a source of passive strain at the knee. Consequently, a disruption in the timing of knee and subtalar motions has been proposed as an injury mechanism (Hamill et al., 1992). McClay and Manal (1997, 1998) reported angle-angle plots that presented the coupling of rearfoot pronation and knee internal rotation in groups of runners classified as “normal” or “excessive” pronators. The expected coupling relationships based on subtalar anatomy were not observed, although again the actual subtalar joint angle was not measured. In a prospective study, Noehren et al. (2007) reported greater peak knee internal rotation and hip adduction angles in runners who developed iliotibial band syndrome (ITBS) compared to healthy runners who developed no injuries. A modeling study on the same subjects found that the ITBS runners placed greater estimated peak strains and strain loading rates of the iliotibial band compared to the healthy runners (Hamill et al., 2008). Similar results were reported in a retrospective study that used the same model (Miller et al., 2007). Donoghue et al. (2008) reported greater peak ankle eversion, ankle dorsiflexion, and leg abduction angles during stance in runners with chronic Achilles tendinopathy.

Recently, techniques from nonlinear dynamics have been used to assess the structure of stride-to-stride fluctuations in joint kinematics and the coupled motion between different body segments. From these studies, a functional role for stride-to-stride variability has emerged. Specifically, a certain degree of variability in the kinematic coordination pattern
used to perform a stride appears to be a natural component of healthy, pain-free running, whereas a lack of variability can indicate a pathological state (van Emmerik et al., 2004). Retrospective studies have supported this theory in relation to running injuries; runners with injuries or a history of injury tend to be less variable in their kinematic coordination than health runners (Hamill et al., 1999; Heiderscheit et al., 2002; Miller et al., 2008). Hamill et al. (1999) suggested that relatively low variability in runners could indicate a narrow range of coordination patterns that allow for pain-free running, and/or the repetitive use of the same degrees of freedom in the neuro-musculo-skeletal system. The former theory implies the presence of an injury, but not its cause, while the latter suggests a possible role for coordination variability in repetitive tissue stress and injury development. However, this theory remains to be tested in prospective studies.

2.4.3. Muscular Strength

Muscular strength (or a lack thereof) has received considerable attention as an injury mechanism in runners. The computer simulations of Cole et al. (1996a) suggested a beneficial role for muscles in attenuating joint loading. It would seem beneficial then for runners to have a high level of muscle mass. However, high amounts of muscle mass in an absolute sense are not conducive to endurance running performance, and most distance runners do not engage in regular intensive resistance training.

Clinical evidence has shown a correlation between muscular weakness and injury potential in runners. The Wake Forest studies found that runners with ITBS, patellofemoral pain, or anterior knee pain were weaker than healthy runners in a variety of isometric and isovelocity measures of knee extensor strength. Deficits in hip abductor strength have been
documented in athletes with ITBS (Fredericson et al., 2000) and patellofemoral pain (Niemuth et al., 2005; Cichanowski et al., 2007).

Longitudinal studies of resistance training and injury prevention or rehabilitation are rare, although a few examples exist. Fredericson et al. (2000) reported that 22 of 24 athletes with ITBS returned to pain-free running after a six-week training program that targeted gluteus medius. Eccentric strength training of the plantarflexors is an effective therapy for Achilles tendinopathy (Kingma et al., 2007). However, the authors noted that it is difficult to isolate the effect of strength training from contributions of other uncontrolled factors.

2.4.4. Older Runners

The injury studies noted in this chapter focused exclusively on younger runners generally between the ages of 18-35. As the participants of the “running boom” approach elderly status (typically ages 65+), the population of older runners is rapidly growing (Jokl et al., 2004). McKean et al. (2006) reported injury rates in younger and older runners. The older runners incurred more injuries to soft tissues (calf muscles, Achilles tendon, and hamstrings) while the younger runners were more prone to knee injuries, shin splints, and tibial stress fractures.

In addition to different injury rates, older runners also exhibit different running biomechanics. When running at the same speed, older runners use shorter stride lengths (Bus, 2003; Cavagna et al., 2008a; Fukuchi & Duarte, 2008) and experience greater vertical GRF impact peaks (Bus, 2003) than younger runners. Older runners hit the ground with a more flexed knee and also flex and extend the knee through a smaller range of motion during the stride (Bus, 2003; Fukuchi & Duarte, 2008). Cavagna et al. (2008a,b) calculated the
mechanical work performed on the center of mass ("external work") in younger and older runners. The older runners performed less external work per stride, and their required external work was more sensitive to increases in speed than the younger runners. The older runners also deviated more from the idealized mass-spring model in the exchange of kinetic and potential energies of their centers of mass. The authors attributed these differences to age-induced changes in concentric muscle strength and joint range of motion, although they had no direct measures of these variables. They also suggested that older runners may optimize their gaits to reduce musculoskeletal loading.

The mechanical properties of skeletal muscles also differ between young and older adults. In general, older muscles are smaller, weaker, slower, and have more compliant active stiffness and less compliant passive stiffness than younger runners (Thelen, 2003). Since muscles develop the active forces that consume metabolic energy and perform mechanical work on the skeleton, these differences in muscle mechanical properties may explain the differences in biomechanics and injury rates between young and older runners. Several authors have posed this hypothesis to explain their biomechanical data (Bus, 2003; Cavagna et al., 2008a,b), but it has not been tested directly. Muscle mechanical properties can be altered through resistance training even in very old individuals (Caserotti et al., 2008). In order to avoid injuries while still attaining the health benefits of running, older runners may need to follow different training strategies than younger runners. However, appropriate strategies for avoiding injuries in older runners are currently unknown.

In summary, musculoskeletal injuries have been a frequent problem for recreational runners despite a tremendous volume of biomechanical research on their causes, treatments, and prevention. Ground reaction forces have generally not distinguished between healthy...
and injury-prone runners, while joint kinematics and muscular strength have. Regarding older runners, specific questions in need of greater clarity are: (1) Can age-related changes in muscle mechanical properties explain differences in running biomechanics between young and older runners? (2) How does the internal loading environment of the body differ between young and older runners? Do these differences explain the injury patterns of young and older runners? (3) Can older runners return to a “young” running gait by increasing their muscular strength? Is this change beneficial for injury prevention?
Figure 2.1. Stride lengths (solid line) and frequencies (dashed line) at a full range of steady running speeds. Adapted from Luhtanen and Komi (1978) and Hay (2002).

Figure 2.2. The cost of transport as a function of locomotor speed for walking (dashed line) and running (solid line). Adapted from Ralston (1958), Mayhew (1977), and Steudel-Numbers and Wall-Scheffler (2009).
Figure 2.3. Simple mechanical models of human running. (a) The mass-spring model of Cavagna et al. (1977). (b) The mass-spring-damper model of Derrick et al. (2000). (c) The four-mass model of Nigg and Liu (1999).

Figure 2.4. Vertical ground reaction force during the stance phase of running, simulated by the mass-spring model (dashed line) and the mass-spring-damper model (solid line).
Figure 2.5. Musculoskeletal models used to simulate running by (a) Taga et al. (1991) and (b) Gerritsen et al. (1995). Circles at the joints represent torque generators. Lines spanning joints represent skeletal muscles.

Figure 2.6. Simulated joint contact impulses from Cole et al. (1996a) at the (a) hip, (b) knee, and (c) ankle joints when using a rigid body model without muscles (R), a rigid body model with muscles (RM), and a non-rigid model with muscles (NRM). BW = body weight.
Figure 2.7. World record progression in the 100-meter dash by men since 1912 (diamonds) and women since 1922 (squares) for races ratified by the International Amateur Athletics Federation. Current record holders as of December 2010 are Usain Bolt for men (9.58 s) and the late Florence Griffith-Joyner for women (10.49 s). Records retracted for violation of IAAF rules were excluded.

Figure 2.8. The force-velocity relationship of skeletal muscle.
**Figure 2.9.** Average incidence rates of injuries (% of all runners studied) from studies published over the last three decades (PubMed search for “running AND injury AND incidence OR frequency”). Studies were assigned to the decade during which data were collected. Incidence rates were weighted for the number of subjects in each study.

**Figure 2.10.** The stress magnitude-frequency relationship for repetitive loading of biological tissues. Adapted from Hreljac (2004).
CHAPTER 3

OPTIMALITY CRITERIA FOR PERFORMANCE-BASED SIMULATIONS OF HUMAN RUNNING

3.1. Introduction

The mechanics and energetics of human running have been topics of interest for many years. Greek vase paintings circa 500 B.C. illustrate distinct postures of endurance and sprinting athletes (Gardner, 1967). Aristotle noted in his De Incessu Animalium that humans run faster when they swing their arms (Farquharson, 1913). Borelli (1989) included running in his classic descriptions of muscle actions and limb motions from the 17th century. More recently, modeling and simulation have played prominent roles in focused quantitative analyses of running. Hill (1928) built a scale model of a sprinter to study the effect of air resistance in a wind tunnel, and Elftman (1940) used a mathematical model to estimate the mechanical work of uni- and biarticular muscles. The simple mass-spring model is often used to study the role of limb compliance in running (McMahon, 1985). In the 1990s, researchers began to use anatomically inspired computer models to simulate running (Taga et al., 1991; Gerritsen et al., 1995; Cole et al., 1996a).

Most computer simulations of human running have focused on data tracking problems, in which the model’s control variables are optimized to replicate a measured motion as accurately as possible (Wright et al., 1998; Neptune et al., 2000a,b; Thelen et al., 2005a; Chumanov et al., 2007; Miller & Hamill, 2009; Hamner et al., 2010; Pandy & Andriacchi, 2010; Sasaki, 2010). Performance-based simulations, which track no data but
instead maximize the quality of the simulated movement in the context of a specified optimality criterion, have been fruitful in the study of jumping (Hatze, 1983; Pandy and Zajac, 1991; Van Soest & Bobbert, 1993; Selbie & Caldwell, 1996; Anderson & Pandy, 1999; Ashby & Delp, 2006) and walking (Chow & Jacobson, 1971; Anderson & Pandy, 2001a; Ackermann & van den Bogert, 2010; Umberger, 2010). The lack of strong dependency on experimental data for tracking purposes endows performance-based simulations with considerable predictive power (e.g. Hatze, 1983), but the technique has only rarely been extended to running (e.g. Sellers et al., 2010).

One limitation to generating performance-based simulations of running is that there are currently no generally accepted optimality criteria for human running. Criteria for optimal performances are straightforward for explosive tasks like jumping or sprinting, but are somewhat ambiguous for sub-maximal tasks like jogging. Experiments on human runners have indicated a potential optimality candidate in the energetic cost of running. The stride length (or frequency) that humans naturally select to run at a particular speed is one that maintains a low rate of energy expenditure (Högberg, 1952; Cavanagh & Williams, 1982; Hamill et al., 1995; Gutmann et al., 2006). The variable of interest is often the “cost of transport”, defined as the amount of metabolic energy consumed per unit distance travelled. However, it is not clear what specific quantity the neuromuscular system prioritizes for minimization, if minimizing the cost of transport governs all salient biomechanical features of running, or if any quantity at all is absolutely minimized. Recent simulations of walking indicated that the pattern of movement is sensitive to the form of the optimality criterion (Ackermann & van den Bogert, 2010), suggesting that optimality criteria should not be arbitrarily defined for movements that lack a clear, unambiguous goal. Previous
performance-based simulations of running have assumed a single criterion, without quantitatively comparing simulation results to data from human runners, and without evaluating other potential optimality criteria (Hase & Yokoi, 2002; Sellers et al., 2010).

Therefore, the purpose of the study was to evaluate a set of potential optimality criteria for generating performance-based simulations of human running. A biological correlate of this purpose is to investigate what quantity the nervous system prioritizes when activating muscles during running. Based on experimental evidence (Gutmann et al., 2006), we hypothesized that minimizing the cost of transport would result in the most realistic simulations of running at a sub-maximal speed.

3.2. Methods

We studied the task of running at a steady sub-maximal speed over level ground. The approach taken was to (1) measure experimental data from experienced runners, (2) develop a computer model, (3) perform a data tracking simulation to establish the model’s ability to exhibit the salient features of human running, and (4) generate performance-based simulations to investigate optimality criteria.

3.2.1. Experimental Data

Experimental data were recorded from 12 adult females (mean±sd: age = 27±6 years, height = 1.66±0.05 m, mass = 61.0±4.7 kg; training = 24.8±6.4 km wk⁻¹). All protocols were approved by the local institutional review board and all subjects provided written informed consent. Subjects ran along a level 30-m runway at a self-selected “normal and comfortable” speed while wearing a set of seven retro-reflective markers that defined the trunk, thigh, leg,
foot, and toes segments in the right sagittal plane. Subjects also wore a set of bipolar surface electromyogram (EMG) sensors on gluteus maximus, rectus femoris, vastus lateralis, biceps femoris (long head), tibialis anterior, gastrocnemius (medial head), and soleus (Hermens et al., 2000). Ground reaction forces (GRF) were collected at 2000 Hz using a strain gauge force platform near the middle of the runway (OR6-5, AMTI, Watertown, MA, USA). Marker positions and EMG were sampled synchronously with the GRF at 200 and 2000 Hz, respectively, using an eight-camera optical motion capture system (Oqus 300, Qualisys, Gothenburg, Sweden) and a portable data-logging unit (Myomonitor IV, Delsys, Boston, MA, USA). Subjects completed five trials each, with one full stride recorded per trial.

Marker positions were lowpass filtered at 12 Hz and used to calculate lower extremity joint and segment angles in the sagittal plane. GRF were lowpass filtered at 75 Hz. EMG were bandpass filtered (20-300 Hz), detrended, full-wave rectified, then lowpass filtered at 5 Hz to calculate linear envelopes. The linear envelopes were scaled by the average envelope magnitude from maximum isometric contractions. All data were averaged over trials, then over subjects. Due to technical difficulties, EMG were only available from eight subjects.

During treadmill running at the same speed as their overground trials (±0.1 m s⁻¹), rates of oxygen consumption and carbon dioxide production were measured from ten of the subjects using a metabolic cart (TrueOne 2400, ParvoMedics, Sandy, UT, USA). Gross rates of metabolic energy consumption were calculated from the gas exchange data (Weir, 1949).

### 3.2.2. Musculoskeletal Model

A two-dimensional, bipedal model of the human musculoskeletal system (Fig. 3.1) was developed in the form of a Fortran computer program. The model was conceptually similar to other computer models used to simulate human locomotion (e.g. Cole et al., 1996a;
Umberger, 2010). The model consisted of nine rigid segments (trunk, thighs, legs, feet, and toes; de Leva, 1996) and 18 muscle actuators (nine actuators per each lower limb; iliopeoas, glutei, vasti, biceps femoris (short head), tibialis anterior, soleus, rectus femoris, hamstrings, and gastrocnemius). Segment inertial parameters and muscle parameters were bilaterally identical. Muscle paths were defined by second-order polynomials fit to length-angle data from OpenSim 2.0 (Delp et al., 2007). Muscle moment arms were defined using the virtual work method (An et al., 1984). Muscle activation and contractile dynamics were modeled using a Hill-based muscle model (Hill, 1938); see Appendix A for more details. The equations of motion were derived symbolically using Autolev 4.1 (OnLine Dynamics, Sunnyvale, CA, USA). State variables were updated using a variable-stepping Kutta-Merson algorithm (Fox, 1962) with absolute and relative integrator error tolerances of 1.0•10⁻⁵.

Passive restoring torques at the hips, knees, and ankles restricted the joints to realistic ranges of motion (Riener & Edrich, 1999). The passive torque at the metatarsophalangeal (MTP) joint was a linear function of angular position and velocity. The hip and knee joints were lightly damped (0.1 Nm per rad s⁻¹). The ankle and MTP joints were heavily damped (0.5 and 1.0 Nm per rad s⁻¹) due to the small masses of the foot and toes segments. Ground contact elements on the heel, toe, and MTP joint of each foot generated GRF to prevent the model from moving through the floor. The vertical force was generated by a nonlinear spring-damper:

\[ F_{gc-y} = A_{gc} |y_{gc}|^3 \exp(B_{gc} \dot{y}_{gc}) \]  
\[ \text{(Eq. 3.1)} \]

and the horizontal force was an approximation of Coulomb friction:

\[ F_{gc-x} = -\mu F_{gc-y} \tanh\left(\frac{\dot{x}_{gc}}{\gamma}\right) \]  
\[ \text{(Eq. 3.2)} \]
where $x_{gc}$ and $y_{gc}$ are the horizontal and vertical positions of the contact element. The ground contact model parameters were $A_{gc} = 2.5 \cdot 10^8$ N m$^{-3}$, $B_{gc} = -0.85$ N s m$^{-1}$ (A.J. van den Bogert, personal communication, 2009) and $\mu = 0.8$, $\gamma = 0.1$ m s$^{-1}$ (Song et al., 2001).

The effect of arm swing was modeled as a vertical sinusoidal force applied at the shoulder. The arm swing force had a period of two cycles per stride and was defined by an amplitude parameter and a phase shift parameter.

The model’s gross rate of metabolic energy consumption was the sum of the muscular, resting, and arm swing energy rates. Muscle energy expenditure rates were calculated from the muscle model state variables (activation and contractile component length) according to Umberger et al. (2003). The energy expenditure of active contractile component lengthening was adjusted according to Umberger (2010). The resting energy rate, representing the viscera, brain, heart, and respiratory muscles, was assigned a value of 1.13 W kg$^{-1}$, which was the mean rate of energy expenditure by the human subjects during upright standing. The arm swing force was assigned an energy rate of 0.76 W kg$^{-1}$ based on the estimated cost of swinging the lower limb (Marsh et al., 2004; Modica & Kram, 2005) and the relative masses of the upper and lower limbs (de Leva, 1996). The cost of transport was the ratio of the gross energy rate and the average horizontal speed of the center of mass.

Each muscle received a time-varying excitation signal that was a piecewise linear function of 21 nodal values. The nodes were spaced evenly over the time for one stride and could take on any magnitude from 0-100% excitation. To enforce periodicity, the 21st node was assigned the same excitation magnitude as the first node. The excitations to the left side muscles were copies of the right side, but phase shifted by half the stride duration.
3.2.3. Simulations

Both data tracking and performance-based simulations were generated. In all simulations, a set of control variables was optimized to maximize the objective function $J$:

$$J = OC - \left( 0.01\varepsilon_{\theta} + 0.0001\varepsilon_{\omega} + 0.001\varepsilon_{pas} \right)$$  \hspace{2cm} \text{(Eq. 3.3)}$$

where $OC$ is the optimality criterion and the other right-hand side terms are penalty terms. $\varepsilon_{\theta}$ and $\varepsilon_{\omega}$ were the sums of the squared differences between the initial and final segment angular positions and velocities, respectively, and encouraged periodic kinematic states. Since human running is not strictly periodic (e.g. Hamill et al., 1999), these penalties were set to zero if a segment’s path was periodic to within $3^\circ$ and $20^\circ$ s$^{-1}$. $\varepsilon_{pas}$ was the sum of the integrals of the squared passive joint moments, and discouraged joint hyperextension. The weighting coefficients on the penalty terms were adjusted in preliminary simulations and set to the smallest values that produced nearly periodic strides.

**Data tracking simulations.** A forward dynamics simulation of one stride was generated using a data tracking approach similar to Neptune et al. (2000a). In this simulation, $OC$ in Eq. 3 was the average mean square error between the simulated and experimental hip, knee, and ankle angles and the horizontal and vertical GRF components:

$$OC = -\frac{\sum_{j=1}^{n} \sum_{i=1}^{5} \left( \frac{X_{ij}^{exp} - X_{ij}^{sim}}{SD_i} \right)^2}{5n}$$  \hspace{2cm} \text{(Eq. 3.4)}$$

where $X_{ij}^{sim}$ and $X_{ij}^{exp}$ are the values of simulated and experimental variable $i$ at time step $j$. $SD_i$ is the between-subjects standard deviation of variable $i$ averaged over one stride. There were 190 control variables: the $9 \times 20 = 180$ muscle excitation parameters, the seven initial angular velocities, the arm force amplitude and phase shift parameters, and the passive
stiffness of the MTP joint. The initial positions were specified as the experimental means from the last time step prior to foot contact (vertical GRF < 15 N). The initial velocities were allowed to vary within one standard deviation of the experimental means. The arm force amplitude could not exceed 25% of the model’s weight. The MTP joint’s stiffness could not exceed 300 Nm rad\(^{-1}\). The initial muscle activations were calculated by numerically solving the equation for activation dynamics (He et al., 1991) over the stride duration. The initial muscle model contractile component lengths were calculated by assuming the initial contractile component velocities to be zero. To reduce computation time, one complete stride was reconstructed from simulation of one step by assuming bilateral symmetry (Anderson & Pandy, 2001a). No constraints were imposed on the center of mass motion, speed, or stride length.

**Performance-based simulations.** Performance-based simulations were generated using an approach similar to Anderson and Pandy (2001a) and Umberger (2010). The control variables were the same 190 variables from the tracking problem, plus the stride duration, which was allowed to vary within one standard deviation. Initial conditions and the bilateral symmetry assumption were the same as those in the tracking problem.

Three different optimality criteria were evaluated in the performance-based simulations. The first criterion minimized the cost of transport and was termed “MinCoT”:

\[
OC = \left( \frac{E_{\text{mus}}}{m_{\text{body}} \Delta x_{\text{CoM}}} \right)^2
\]

(Eq. 3.5)

where \( E_{\text{mus}} \) is the muscle energy expenditure, \( m_{\text{body}} \) is the mass of the model (61 kg), and \( \Delta x_{\text{CoM}} \) is the change in horizontal position of the model’s center of mass. The rationale for this criterion is: (1) it maximizes the potential distance covered during a run to exhaustion by
minimizing the rate at which energy is consumed at the whole-body level (Carrier, 1984; Steudel-Numbers & Wall-Scheffler, 2009), and (2) when speed, stride length, or stride frequency is specified, running humans self-select the other two parameters such that the cost of transport is nearly minimized, relative to other potential choices (Gutmann et al., 2006).

The second criterion minimized the muscle activations and was termed “MinAct”:

\[
OC = -\frac{1}{t_f} \sum_{i=1}^{m} \left( \int_{0}^{t_f} A_i^2 dt \right) 
\]

(Eq. 3.6)

where \( A_i(t) \) is the time-varying activation of the \( i^{th} \) muscle in an \( m \)-muscle system, and \( t_f \) is the stride duration. This criterion is based on the rationale that by using the unweighted muscle activations (all varied nondimensionally on \([0,1]\)), exhaustion of any single muscle and a consequential fatigue-induced change in movement are avoided, regardless of the size or strength of the muscle. In addition, this criterion was recently shown to generate realistic simulations of walking using a similar model, where it was referred to as “minimum fatigue” (Ackermann & van den Bogert, 2010).

The third criteria minimized the muscle stresses and was termed “MinStress”:

\[
OC = -\frac{1}{t_f} \sum_{i=1}^{m} \left( \int_{0}^{t_f} \left( \frac{F_i}{PCSA_i} \right)^2 dt \right) 
\]

(Eq. 3.7)

where \( F_i(t) \) is the force in the \( i^{th} \) muscle and \( PCSA_i \) is its physiological cross-sectional area (Haxton, 1944). The rationale for this criterion is also the argument of avoiding fatigue at the individual muscle level (Crowninshield & Brand, 1981b), but is based on a mechanical variable (muscle force) whereas Eq. 3.6 is based on activation, which is related to a biochemical variable (the proportion of bound calcium ions). All optimality criteria were
scaled such that their magnitudes were approximately equal. Equation 3.5 was divided by 4.0, Eq. 3.6 was multiplied by 2.0, and Eq. 3.7 was divided by 2,500,000.

**Optimization algorithm.** All optimizations were performed using a parallel simulated annealing algorithm (Higginson et al., 2005) on a cluster of eight 3.0-GHz CPUs (Pentium 4 HT, Intel, Santa Clara, CA, USA). For the tracking problem, the optimization parameters, in the algorithm’s notation (Goffe et al., 1994; see Appendix E), were: N = 190, NS = 24, NT = 100, initial T = 100, and RT = 0.85. The initial guess was 0.5 for all controls. The performance-based simulations began from the data tracking solution as an initial guess, with parameters N = 191, NS = 8, NT = 100, initial T = 25, and RT = 0.85 (Appendix E). Optimizations terminated when a temperature reduction did not improve the optimal solution by at least 1%. This criterion was met after eight days elapsed time for the tracking problem and typically three days elapsed time for each performance-based simulation.

3.2.4. Evaluation

The accuracy of the speeds, stride parameters, and costs of transport of the simulations were compared by calculating their percent differences relative to the means of the experimental data. The simulated joint angles and GRF were compared by calculating their root mean squared errors (RMSE) relative to the means of the experimental data. All RMSE were scaled by the average between-subjects standard deviation (SD) so that the joint angles and GRF could be compared on the same scale. The RMSE are therefore reported in multiples of the SD. Temporal similarity between the simulated muscle activations and the EMG linear envelopes was assessed by calculating zero-lag cross-correlations (ZLCC):
\[ ZLCC = \frac{\sum_{i=1}^{n} (x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum_{i=1}^{n} (x_i - \bar{x})^2} \sqrt{\sum_{i=1}^{n} (y_i - \bar{y})^2}} \] (Eq. 3.8)

where \( \bar{x} \) and \( \bar{y} \) are the mean values of two time-varying signals \( x(t) \) and \( y(t) \) that both have \( n \) points, and ZLCC is the strength of the correlation. The simulation that most closely matched the experimental data based on these comparisons (i.e. smallest percent differences, smallest scaled RMSE, and largest ZLCC) was deemed the most accurate.

### 3.3. Results

#### 3.3.1. Subject Performance

The subjects ran at an average speed of 3.80±0.51 m s\(^{-1}\) with a stride length of 2.61±0.27 m and a stride frequency of 1.45±0.08 Hz. The average gross metabolic power was 16.0±3.4 W kg\(^{-1}\), which corresponds to a cost of transport of 4.2±0.9 J m\(^{-1}\) kg\(^{-1}\).

#### 3.3.2. Data Tracking Simulation

The tracking simulation ran at 3.76 m s\(^{-1}\) using a stride length of 2.59 m. Both values were within 2% of the mean values of the human subjects (3.80 m s\(^{-1}\) and 2.61 m), even though neither variable was explicitly tracked. The tracking solution matched the means of the experimental data to within 1.3 between-subjects SD on average (Fig. 3.2). The hip, knee, and ankle joint angles had average tracking errors of 1.0, 0.9, and 0.6 SD, respectively. The horizontal and vertical GRF components had tracking errors of 2.2 and 1.6 SD. This accuracy is similar to other studies that used similar models for tracking simulations of human running (e.g. Sasaki, 2010).
The tracking simulation expended energy at a very high rate. The metabolic power (36.8 W kg\(^{-1}\)) and cost of transport (9.8 J m\(^{-1}\) kg\(^{-1}\)) were over twice the means of the human subjects (16.0 W kg\(^{-1}\) and 4.2±0.9 J m\(^{-1}\) kg\(^{-1}\)). The high rate of energy expenditure was due to the high muscle excitation magnitudes (Fig. 3.3). All nine muscles were excited to at least 97% at some point during the stride cycle. The timing of the optimized muscle excitations generally agreed with the on/off timing of the EMG data, except for a lack of soleus excitation in late swing and a quick spike in hip extensor excitation in early swing. The ZLCC between the muscle activations and the EMG linear envelopes averaged 0.50±0.29, with a range of 0.19 (hamstrings) to 0.89 (soleus). The magnitude of the muscle activation usually exceeded the EMG linear envelope, although it is difficult to make a one-to-one comparison between these magnitudes due to issues with the collection and processing of EMG data (Kamen & Caldwell, 1996).

### 3.3.3. Performance-based Simulations

All three performance-based simulations ran at speeds within 5% of the average speed of the human subjects. The speeds, stride lengths and frequencies, and costs of transport for all three simulations were within one SD of the means for the human subjects. These data are summarized in Fig. 3.4. The MinAct simulation had the most accurate cost of transport (4.4 J m\(^{-1}\) kg\(^{-1}\); +6% above the mean), followed by the MinStress simulation (4.7 J m\(^{-1}\) kg\(^{-1}\); +12%) then the MinCoT simulation (3.6 J m\(^{-1}\) kg\(^{-1}\); -15%). The segment paths of all the simulations visually resembled the mean segment paths of the human subjects (Fig. 3.5).

More detailed comparisons are presented as RMSE with the experimental means in Fig. 3.6. Recall that none of the experimental data were tracked in the three performance-
based simulations. The hip angles for the MinAct and MinCoT simulations had RMSE (0.9 and 1.0 SD) that were 44% and 38% smaller than the MinStress simulation (1.6 SD). The knee angle RMSE for the MinStress and MinAct simulations (0.7 and 0.8 SD) were 46% and 38% smaller than the MinCoT simulation (1.3 SD). The MinAct simulation had the smallest ankle angle RMSE (0.8 SD), 38% smaller than the MinStress simulation (1.3 SD) and 47% smaller than the MinCoT simulation (1.5 SD). The GRF components were less accurate than the joint angles (all RMSE ≥ 2.7 SD). The MinStress simulation had the most accurate horizontal GRF (RMSE = 2.7 SD), 2% smaller than the MinCoT simulation (2.8 SD) and 15% smaller than the MinAct simulation (3.2 SD). The MinAct simulation had the most accurate vertical GRF (RMSE = 4.3 SD), 10% smaller than the MinCoT simulation (4.8 SD) and 15% smaller than the MinStress simulation (5.1 SD).

Most of the differences in joint angle accuracy appeared in the swing phase (Fig. 3.7). Peak joint angles during stance varied by under 5°. The peak hip flexion angle during 60-80% of the stride (mid-swing) was 14° less flexed for the MinStress simulation than for the MinAct and MinCoT simulations. The MinStress simulation flexed the hip from 90-100% of the stride (late swing) while the MinAct and MinCoT simulations extended the hip. The peak knee flexion angle in mid-swing was 16° less flexed for the MinCoT simulation than for the MinAct and MinStress simulations. The peak ankle dorsiflexion angles in swing were 9 and 10° greater for the MinAct and MinStress simulations than for the MinCoT simulation. The inaccuracies of the horizontal and vertical GRF were primarily due to the duration of the stance phase, which was shorter in the simulations (31% of the stride) than the human subjects (38±5%). The impulse of the vertical GRF (vertical GRF integrated over time) was within 10% of the experimental mean for all three performance-based simulations.
When comparing the muscle excitation strategies of the simulations (Fig. 3.8), the performance-based simulations generally had lower muscle excitation magnitudes than the data tracking simulation. Zero-lag cross correlations between the muscle activations and the EMG linear envelopes averaged 0.58±0.36, 0.67±0.36, and 0.59±0.39 for the MinCoT, MinAct, and MinStress simulation, respectively. ZLCC between subjects (i.e. each subject compared to every other subject) averaged 0.65±0.35. In all three simulations, the lowest ZLCC was found for rectus femoris (average 0.16) and the highest for the plantarflexors (average 0.94). The data tracking simulation excited all muscles to at least 97% at some point during the stride while the performance-based simulations excited only two to four muscles to above 80% (iliopsoas, soleus, and gastrocnemius for the MinCoT simulation; iliopsoas, vasti, soleus, and gastrocnemius for the MinAct simulation; soleus and gastrocnemius for the MinStress simulation). The MinCoT simulation avoided use of the largest knee muscles (vasti and hamstrings) during swing, and avoided using soleus to prepare the foot for heel-strike in late swing. The magnitude of glutei excitation during stance and the duration of iliopsoas excitation during the entire stride were also lower for the MinCoT simulation than for the other two performance-based simulations. The excitation strategies of the MinAct and MinStress simulations were generally similar, although the MinStress simulation used more tibialis anterior excitation during mid swing and used both glutei and hamstrings to extend the hip in late swing, while the MinAct simulation used primarily hamstrings.

As expected by the less intense use of the muscles, nearly all muscles expended considerably less metabolic energy in the performance-based simulations than in the data tracking simulation (Fig. 3.9). For example, the tracking simulation consumed just over 150
J with vasti, while all the performance-based simulations consumed less than 65 J. The lone exception was seen in soleus, which expended slightly more energy in the MinStress simulation (42 J vs. 41 J). Within the performance-based simulations, the MinAct simulation expended almost twice as much energy as the MinCoT simulation with vasti (62 J vs. 35 J) and hamstrings (58 J vs. 30 J). The MinStress simulation expended a moderate amount of energy with these two muscles (45 J for vasti, 46 J for hamstrings). The relatively high cost of transport for the MinStress simulation was explained by slightly greater energy expenditure by the other seven muscles (on average, 6 J more than the MinAct simulation and 8 J more than the “minimum energy” simulation). The MinStress simulation expended more energy with soleus (42 J) than the MinCoT and MinAct simulations (both 26 J).

3.4. Discussion

It has been suggested that humans run in a way that minimizes the rate at which metabolic energy is consumed at the whole-body level (Alexander, 1989; Srinivasan, 2009). To date, this theory has been motivated primarily by measurements taken from humans running under various conditions (Högberg, 1952; Cavanagh & Williams, 1982; Hamill et al., 1995; Gutmann et al., 2006; Steudel-Numbers & Wall-Scheffler, 2009). We note that the theory that humans absolutely minimize the cost of transport cannot be confirmed conclusively from a finite set of experimental measurements. What is clear from these studies is that humans run in a way that incurs a relatively low cost when running under “natural” or “comfortable” conditions. However, the cost of transport is not necessarily prioritized for absolute minimization. The present simulation results indicate that, at least in simulated running, a variety of variables can be minimized to incur low costs of transport,
even if the cost of transport itself is not explicitly minimized. All three optimality criteria tested in the performance-based simulations resulted in energy expenditure rates well within the range of the human runners (Fig. 3.4). Which criterion is the most likely optimality candidate for human running? To answer this question, we will first address the accuracy of the simulations, and will then analyze and compare various aspects of the simulation results.

The motions and energy expenditure rates of the simulations, at the whole-body level, were realistic in comparison to the experimental data (Figs. 3.4 and 3.5). Although a perfect match between the full simulated and experimentally measured data sets is not expected due to the reduction in degrees of freedom inherent to the modeling process (Hatze, 2002), deviations in the model’s design from the human neuro-musculo-skeletal system and discrepancies between the simulated and experimentally measured data must be addressed before the present results can be generalized to human running. The model simulated sagittal plane motion only and lacked the muscles that primarily control motions in the frontal and transverse planes. The results of this study are therefore limited to the tasks of support and propulsion, which are controlled by the major flexor/extensor muscles (Hamner et al., 2010).

The vertical GRF of the performance-based simulations had large impact peaks about 2.8 times body weight in magnitude, larger than the typical 1.5-2.5 times body weight in human runners (Cavanagh & Lafortune, 1980). A more realistic impact peak appeared in the tracking simulation (2.2 times body weight), suggesting that a penalty term in the objective function for the performance-based simulations could reduce this inaccuracy. Perhaps humans optimize their running gaits in part to maintain low impact forces, in which case such a penalty term would be appropriate. However, the large impact peaks could simply be due to inadequacy within the model rather than the objective function. Including wobbling
masses on the body segments (e.g. Gruber et al., 1998) did not greatly improve the impact forces, nor did adjusting the parameters of the ground contact model or allowing additional excitation nodes. We chose not to include an impact penalty because the focus of this study was on energy expenditure. An impact penalty encourages adjustments in muscle excitations to reduce the impact force (e.g. the high cost of transport in the tracking solution), but excessive impact force is due primarily to non-muscular forces (Neptune et al., 2004).

The model of the foot may also have influenced the simulated GRF. While our foot model had only two segments, it has been suggested that the human foot has at least four functional segments (Wolf et al., 2008). We chose to use a two-segment model because it is used widely in studies of running biomechanics, and because appropriate parameters for active and passive control over a more complex foot model could not be identified either experimentally or in the literature.

The optimized muscle excitations agreed well with the EMG timing data (Fig. 3.8). In the present simulations, no constraints were placed on any of the muscle excitation parameters. Other studies using similar models have restricted excitations to be similar to processed EMG data (e.g. Sasaki & Neptune, 2006) or have inversely calculated excitations using static optimization (e.g. Chumanov et al., 2007; Hamner et al., 2010). One unexpected result was that the plantarflexors were maximally excited for simulations of running at a sub-maximal speed. Other studies of walking and running at much slower speeds have also featured near-maximal excitations of the plantarflexors (Anderson & Pandy, 2001a; Sasaki & Neptune, 2006). Hamner et al. (2010) recently simulated running at 4.0 m s\(^{-1}\) and predicted near-maximal excitations for nearly all muscles in the model. It may be that human muscles, in particular the plantarflexors, are in reality stronger than the maximum isometric forces.
typically identified for Hill-based muscle models. The relative simplicity of the foot model may also have contributed to this finding. We note that EMG magnitudes from soleus do not increase dramatically as running speed increases from 3.5-4.5 m s\(^{-1}\) (Gazendam & Hof, 2007) and that the simulations still expended energy at realistic rates.

An additional limitation relates to the neuromuscular control scheme, which was designed for simulations of one step only. The present simulations performed only one step and reconstructed strides by assuming bilateral symmetry of the limbs (Anderson & Pandy, 2001a). This assumption reduced CPU time by approximately 50%. The model can perform an actual stride (two steps) if CPU time is doubled, but tends to fall over if additional steps are attempted. Although not reported here, Floquet analysis of the model’s center of mass trajectory indicated that although the model was stable from a theoretical mechanics definition, it lacked the flexibility seen in human running for responding to perturbations to the locomotor system (Appendix B). This finding is not surprising since the control scheme did not feature any feedback mechanisms. Other models have included such mechanisms but require more complex control schemes that are coupled with the musculo-skeletal dynamics (Taga et al., 1991; Hase & Yokoi, 2002). Simulations of multiple strides would require such a scheme, but would not have added substantially to our results.

The hypothesis that minimizing the cost of transport would result in the most realistic simulation of running was not supported. Specifically, minimizing either muscle activations or muscles stresses produced more realistic patterns of knee and ankle motion than minimizing the cost of transport (Figs. 3.6 and 3.7). The “MinAct” simulation was in general the most realistic one, as it compared most accurately to the experimental data in three of the five mechanical variables, namely the hip angle, the ankle angle, and the vertical GRF (Fig.
3.6). The “MinStress” simulation was most realistic in the other two variables (knee angle, horizontal GRF) while the “MinCoT” simulation was not the most accurate for any variable. Absolutely minimizing the cost of transport discouraged the use of large, strong muscles such as vasti, hamstrings, and soleus, which led to a lack of control of the knee and ankle during swing. These results suggest that avoiding fatigue of any one muscle by minimizing activation is an important control policy for human running, perhaps more important than absolutely minimizing the energy expended. Ackermann and van den Bogert (2010) recently reached a similar conclusion from simulations of walking, although most of their differences were observed in the stance phase. These theories are unfortunately difficult to test in human subjects, since muscle stress is impractical to measure in vivo (Komi, 1990) and since activation is largely a conceptual variable that cannot easily be measured in vivo (Gasser & Hill, 1924), although it could perhaps be computed from EMG data (Hof, 1984).

Although all three performance-based simulations expended energy at reasonably realistic rates (Fig. 3.5), the cost of transport for the “MinCoT” simulation was 15% lower than the mean of the human subjects, while the cost of transport for the “MinAct” simulation was only 6% above the mean. The cost of the “MinStress” simulation was 12% above the mean. These results are sensitive to the assigned values of metabolic rates for the arm swing force and the resting upright stance condition. If these combined estimates were off by more than 40%, the cost of transport for the MinCoT becomes most accurate. Arm swing is accomplished primarily by the elbow and shoulder muscles (Hinrichs, 1990), which are considerably less massive and consume less energy than the large muscles of the lower limbs. The estimated arm swing metabolic rate (0.76 W kg\(^{-1}\)) would need to be off by 102% in order for the MinCoT simulation’s cost of transport to become 1% closer to the
experimental mean than the MinAct simulation. Pontzer et al. (2009) recently reported that suppressing arm swing in running humans had no significant effect on the cost of transport, and that the excitation timings of antagonistic deltoid muscles were simultaneous rather than alternating. These results were consistent with their hypothesis that arm swing is largely a passive motion that does not require substantial metabolic power.

Regarding the resting metabolic rate, the assigned value (1.13 W kg$^{-1}$) was the metabolic rate for upright stance in the human subjects. It would need to be off by 69% for the MinCoT simulation’s cost of transport to become 1% closer to the experimental mean than the MinAct simulation. It might be expected that the metabolic rates for the heart, respiratory muscles, brain, and viscera increase from upright stance to running. This theory cannot be tested directly in humans, as it requires highly invasive measurements. However, Ellerby et al. (2005) reported that 90% of the increased cardiac output of guinea fowl between rest and fast running near the animal’s maximum oxidative capacity was directed to the lower limb muscles. The remaining 10% supplied the heart and respiratory muscles, which account for about 1% of the total muscle mass in humans. Visceral and brain blood flow did not differ significantly between rest and strenuous running. Based on these findings, we are confident that the arm swing and resting metabolic rates are accurate enough to have confidence in the grading of the various costs of transport in the simulations.

The differences between the “MinAct” and “MinStress” simulations were relatively small in comparison to differences with the “MinCoT” simulation. This finding suggests that maintaining low mechanical stresses of the muscles is also a potentially important control policy for human running, and supports theories of maximizing muscle endurance / minimizing muscle fatigue by minimizing muscle stress (Crowninshield & Brand, 1981b).
As noted in the methods, both “MinAct” and “MinStress” weighed all muscles equally regarding their contributions to the optimality criterion, such that intense use of any one muscle, regardless of its size and strength, was discouraged. The differences between these simulations can be explained by the variables that determine muscle stress and activation. Muscle stress is determined by muscle force, which is affected by the muscle’s kinematic state as well as its excitation. Activation is determined by the excitation history only. Muscle stress in particular is a variable which the nervous system likely has direct knowledge of via Golgi tendon organs, although it may also presumably have some time-delayed knowledge of muscle activation by its modulation of motor unit recruitment and rate-coding. Since the “MinAct” simulation was in general more realistic than the “MinStress” simulation in comparison to the experimental data, we suggest that minimizing activations rather than stresses is a more likely optimality candidate if one or the other is to be selected.

While the stride frequency of the data tracking solution was imposed by specifying the stride duration, it was allowed to vary as a control in the performance-based simulations. The freely chosen stride frequency in human running does not always minimize the rate of energy expenditure. A stride frequency slightly higher than the freely chosen one requires slightly less metabolic power in roughly 10-20% of habitual runners (Morgan et al., 1994). The performance-based simulations featured slightly higher stride frequencies (1.50 Hz on average) than the human subjects (1.45 Hz on average). This finding supports the conclusion of Morgan et al. (1994) that some runners may gain an energetic benefit by using shorter, faster strides. Interestingly, this adjustment may also be beneficial for skeletal health (Edwards et al., 2009).
A final note relates to the energy expenditure rate of the tracking simulation, which was unrealistically high (Fig. 3.4). Large amounts of energy were expended due to very high individual muscle excitations, along with relatively high levels of antagonistic co-activation (Fig. 3.8). Other tracking simulations of human running have had similar results (Sasaki & Neptune, 2006; Hamner et al., 2010). Compared to no or minimal co-activation, antagonistic co-activation allows for a higher level of control over shaping the joint torques used to track the experimental data. This result suggests that data tracking simulations should include some sort of conservative term or constraint set to avoid over-estimating muscle excitations. Accurate tracking of segment kinematics and GRF does not on its own ensure realistic use of individual muscles. This consideration is particularly important if variables like energy expenditure, muscle force, and joint contact force are of interest. One option is to include a penalty term for intense muscle use when tracking data (Davy & Audu, 1987). When the tracking optimization was repeated with the sum of the squared muscle activation integrals added to the objective function, the model ran at 3.74 m s\(^{-1}\) with a cost of transport of 7.5 J m\(^{-1}\) kg\(^{-1}\) and an average tracking error of 1.4 SD, compared to 3.76 m s\(^{-1}\), 9.8 J m\(^{-1}\) kg\(^{-1}\), and 1.3 SD with tracking only. The excitation magnitudes could also be limited on a temporal and muscle-by-muscle basis, but these limits may be difficult to define appropriately if EMG data are unavailable, and may be inappropriate if the work is exploratory in nature. Even if muscle energy expenditure is not included explicitly in an objective function, the use of a muscle energy expenditure model (e.g. Umberger et al., 2003) is still valuable in these types of simulations, as it provides useful metrics for comparisons with human metabolic data.

In conclusion, sagittal plane simulations of human running were sensitive to the form of the optimality criterion defined as a task objective. A tracking simulation established the
ability of the model to run with realistic mechanics, but over-estimated metabolic energy consumption. Minimizing fatigue by minimizing muscle activations generated more realistic motion of the hip, knee, and ankle than minimizing the cost of transport, while still expending whole-body metabolic energy at a realistic rate. We suggest that minimizing fatigue at the individual muscle level is an important priority of the neuromuscular system during running, perhaps more so than minimizing the absolute energetic cost of the movement. Avoiding high muscle stresses and external impact forces may be important considerations as well.
Figure 3.1. Schematic of the musculoskeletal model, with nine segments (trunk, thighs, legs, feet, toes) and nine muscles per each lower limb (iliopsoas, glutei, vasti, biceps femoris, tibialis anterior, soleus, rectus femoris, hamstrings, gastrocnemius).
Figure 3.2. Joint angles and GRF components vs. the stride cycle from the human subjects and the data tracking simulation. Shaded area is +/- one between-subjects standard deviation around the mean of the human subjects. Solid line is the simulation data. The stride begins and ends at heel-strike. Vertical dashed lines indicate toe-off.
Figure 3.3. Optimized muscle excitations (thin lines), activations (thick lines), and EMG linear envelopes (dashed lines) for the tracking simulation. ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at heel-strike. Vertical dashed lines indicate toe-off. Linear envelopes were scaled by values from maximum isometric contraction. No EMG data were available for ILP and BF.
Figure 3.4. Speeds, stride parameters, and optimality criteria (cost of transport, sum of squared activation integrals, and sum of squared muscle stress integrals) for the human subjects and the simulations. Errors bars are one standard deviation between subjects. “Track” is the data tracking simulation. “MinCoT”, “MinAct”, and “MinStress” are performance-based simulations. The optimality criteria for muscle activations and muscle stresses are not shown for the human subjects because these variables could not be measured in vivo.
Figure 3.5. Stick figure profiles of the mean experimental data (Subjects), the data tracking simulation (Track), and the three performance-based simulations (MinCoT, MinAct, and MinStress). The profiles begin at a right-foot heel-strike, end at the next right-foot heel-strike, and are traced every 10% of the stride.
Figure 3.6. Root mean square errors (RMSE) between the mean experimental data and the simulations. Errors are expressed in multiples of between-subjects standard deviations (SD). “Hip”, “Knee”, and “Ankle” are the hip, knee, and ankle joint angles. “HGRF” and “VGRF” are the horizontal and vertical components of the ground reaction force.
Figure 3.7. Joint angles (left panels) and ground reaction force components (right panels) from the performance-based simulations. Shaded areas are two between-subjects standard deviations around the mean of the human subjects. Vertical dashed lines indicate toe-off.
Figure 3.8. Optimized muscle excitations (thin lines), activations (thick lines), and EMG linear envelopes (dashed lines) for all simulations. ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at heel-strike. Vertical dashed lines indicate toe-off. Linear envelopes were scaled by values from maximum isometric contractions. No EMG data were available for ILP and BF.
Figure 3.9. Metabolic energy expended by individual muscles in the simulations. ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius.
CHAPTER 4

SENSITIVITY OF MAXIMUM SPRINTING SPEED TO
CHARACTERISTIC PARAMETERS OF THE MUSCLE
FORCE VELOCITY RELATIONSHIP

4.1. Introduction

Sprinting is perhaps the most exciting form of human running. There is something intrinsically interesting about watching humans (or other animals) sprint at maximum effort, as evidenced by the popularity of athletic events such as the Olympic 100-m dash and the Kentucky Derby. One of the most important variables in sprinting is the maximum speed, which in this study is defined as the fastest average speed of the center of mass that can be sustained during the steady-speed phase of a sprint.

Maximum speed varies widely among individuals depending on a plurality of factors (van Ingen Schenau et al., 1994). Strength (e.g. maximum isometric muscle force) is certainly important, but so is the rate at which that strength can be used. It has long been suspected that the velocity-dependent nature of force production in skeletal muscles (Hill, 1922, 1938; Fenn & Marsh, 1935) ultimately limits the maximum human sprinting speed (Furusawa et al., 1927). The well-known force-velocity relationship of muscle (Fig. 4.1) is characterized by three parameters: Hill’s dynamic constants $a$ and $b$ on the concentric limb (Hill, 1938), and the plateau force $F_{ecc}$ on the eccentric limb (Katz, 1939). The ratio $b/a$ defines another parameter of interest: the maximum shortening velocity $v_{max}$ at which the muscle produces no force. $F_{ecc}$ is typically scaled by the maximum isometric force $F_o$ to
create the nondimensional parameter $C_{ecc}$. Independent changes of each parameter have different effects on the shape of the force-velocity relationship (Fig. 4.1). $a$ is often referred to as the “shape parameter”; as $a$ increases, the concentric and eccentric hyperbolas become more shallow. As $b$ or $v_{max}$ increases, the entire concentric hyperbola shifts to the right, with greater shifts at faster velocities. As $C_{ecc}$ increases, the maximum eccentric force increases and the muscle force approaches the eccentric asymptote more gradually.

These features of the force-velocity relationship can potentially limit performance in sprinting (or other explosive tasks, e.g. Domire & Challis, 2010) by constraining the amount of force that muscles can produce in a given kinematic state. In a previous simulation study, we identified the force-velocity relationship as the most critical of the mechanical properties of skeletal muscle in limiting the maximum sprinting speed (Miller et al., 2010). To follow up this general conclusion, a more specific set of questions is: which muscles are important for sprinting, and to what extent are these muscles impacted by the force-velocity constraint? Over the last 30 years two different theories have emerged on specifically how the force-velocity relationship limits sprinting speed: the “energy management” theory of Chapman and Caldwell (1983a,b), which focuses on the swing phase and flexor muscle groups, and the “time to generate force” theory of Weyand et al. (2000, 2010), which focuses on the stance phase and extensor muscle groups. While not necessarily mutually exclusive, it is unknown which theory, if either, plays the dominant role in limiting maximum speed. Analyses of the abilities of various muscles to produce force in stance and swing when the characteristic parameters of the force-velocity relationship are adjusted could help clarify this issue.

In this study, we expanded on the noted previous work by examining the sensitivity of maximum sprinting speed to the various parameters that define the force-velocity
relationship, with a focus on the mechanics of individual muscles. The force-velocity relationship in humans is sensitive to training status (e.g. Thorstensson et al., 1977; Andersen et al., 2005), but it is generally not possible to modify specific selected parameters \textit{in vivo}. In addition, training studies can be confounded by other variables that may affect sprinting ability, such as muscle strength and fiber type composition. We therefore used a computer simulation approach to determine the effects of incremental adjustments in force-velocity parameters on maximum sprinting speed. We hypothesized that increasing any of the three force-velocity parameters would increase the maximum sprinting speed. In addition, since sprinting presumably requires muscles to shorten at fast velocities, we hypothesized that maximum sprinting speed would be most sensitive to $v_{\text{max}}$.

4.2. Methods

4.2.1. Experimental Data

Experimental data were recorded from 12 adult females (mean±sd: age = 27±6 years, height = 1.66±0.05 m, mass = 61.0±4.7 kg). All protocols were approved by the local institutional review board and all subjects provided written informed consent. Subjects sprinted at maximum effort along a level 30-m runway while wearing a set of seven retro-reflective markers that defined the trunk, thigh, leg, foot, and toes segments of the right lower limb in the sagittal plane. Subjects wore a set of bipolar surface electromyography (EMG) sensors on gluteus maximus, vastus lateralis, tibialis anterior, soleus, rectus femoris, biceps femoris (long head), and gastrocnemius (medial head). Ground reaction forces (GRF) were collected at 2000 Hz from a strain gauge force platform positioned in the middle of the runway (OR6-5, AMTI, Watertown, MA, USA). Marker positions and EMG were sampled
synchronously with the GRF data at 200 Hz and 2000 Hz, respectively, using an eight-camera optical motion capture system (Oqus 300, Qualisys, Gothenburg, Sweden) and a portable data-logging unit (Myomonitor IV, Delsys, Boston, MA, USA). Subjects completed five trials each, with one full stride recorded per trial.

Marker positions were lowpass filtered at 12 Hz and used to calculate lower extremity joint and segment angles in the sagittal plane. GRF were lowpass filtered at 75 Hz. EMG were bandpass filtered (20-300 Hz), detrended, full-wave rectified, then lowpass filtered at 5 Hz to calculate linear envelopes. The temporal synchrony of the EMG linear envelopes and the muscle model activations was assessed by calculating zero-lag cross-correlations. All data were averaged over trials, then over subjects. Due to equipment malfunction, EMG data were only available from eight of the subjects.

### 4.2.2. Musculoskeletal Model

A two-dimensional, bipedal model was developed in the form of a Fortran computer program. The model consisted of 58 ordinary differential equations representing the dynamics of the musculoskeletal system (Chapter 3). The model consisted of nine rigid segments (trunk, thighs, legs, feet, and toes; de Leva, 1996) and nine muscle actuators per each lower extremity (iliopsoas, glutei, vasti, biceps femoris (short head), tibialis anterior, soleus, rectus femoris, hamstrings, and gastrocnemius). Muscle activation and contractile dynamics were modeled using a Hill-based muscle model (Hill, 1938); see Appendix A for more details. Metabolic energy expenditure was calculated according to Umberger et al. (2003) assuming primarily anaerobic metabolism. The force-velocity equations of van Soest
and Bobbert (1993) were used (Fig. 4.1). The dimensionless parameters $a_R$ and $b_R$ were defined according to Winters & Stark (1985):

\[ a_R = \frac{a}{F_o} = 0.1 + 0.4FT \]  \hspace{1cm} (Eq. 4.1)

\[ b_R = \frac{b}{L_o} = a_R v_{\text{max}} \]  \hspace{1cm} (Eq. 4.2)

where $F_o$ is the maximum isometric muscle force, $FT$ is the proportion of fast-twitch muscle fibers, and $L_o$ is the optimal contractile component length. Muscle-specific values for these parameters are listed in Table 4.1. See Appendix A for details on their derivation.

Passive restoring torques at the hips, knees, and ankles restricted the joints to realistic ranges of motion (Riener & Edrich, 1999). The passive torque at the metatarsophalangeal (MTP) joint was a linear function of angular position and velocity. The hip and knee joints were lightly damped (0.1 Nm per rad s$^{-1}$), while the ankle and MTP joints were heavily damped (0.5 and 1.0 Nm per rad s$^{-1}$) due to the small masses of the foot and toes segments. Ground contact elements on the heel, toe, and MTP joint of each foot generated GRF to prevent the model from moving through the floor. The force was generated by a viscoelastic spring in the vertical direction and by an approximation of Coulomb friction in the horizontal direction. The effect of arm swing was modeled as a vertical sinusoidal force applied at the shoulder. The arm swing force had a period of two cycles per stride and was defined by an amplitude parameter and a phase shift parameter.

Each muscle received a time-varying excitation signal that was a piecewise linear function of 21 nodal values. The nodes were spaced evenly over the time for one stride and could take on any magnitude from 0-100% excitation. To enforce periodicity, the 21$^{st}$ node
was assigned the same excitation magnitude as the first node. The excitations to the left side muscles were copies of the right side, but phase shifted by half the stride duration.

**4.2.3. Simulations**

Simulations of one stride of sprinting were generated using the optimization procedure described in Chapter 3. Starting from a data-tracking solution, an initial simulation was generated using nominal force-velocity parameters, followed by a set of additional simulations in which these parameters were systematically adjusted. In all simulations, the model’s control variables were optimized via simulated annealing (Higginson et al., 2005) to maximize an objective function where the primary term was the average horizontal speed of the model’s center of mass (Eq. 4.3). The 200 control variables were the 9x20=180 muscle excitation parameters, the seven initial angular positions, the nine initial velocities, the amplitude and phase shift of the arm swing force, the angular stiffness of the metatarsophalangeal (MTP) joints, and the stride duration. The initial kinematics were bounded within one standard deviation of the means from the experimental data at the first time step of foot contact (vertical GRF < 15 N). The stride duration was bounded within one standard deviation of the mean duration of the subjects (0.44-0.56 s). The arm swing and MTP joint parameters were bounded within realistic ranges based on literature data (Miller et al., 2009; Stefanyshyn & Nigg, 1997).

**Initial simulation.** In the initial simulation, the objective function was the average horizontal velocity of the center of mass, minus a set of penalty functions:

\[
J = \frac{\Delta x_{CoM}}{t_f} - \left(0.01\varepsilon_\theta + 0.0001\varepsilon_{\omega} + 0.001\varepsilon_{pas} + \varepsilon_{grf}\right) \quad \text{(Eq. 4.3)}
\]
where $\Delta x_{\text{CoM}}$ is the change in horizontal position of the center of mass and $t_f$ is the stride duration. The weighting coefficients on the penalties were adjusted in preliminary simulations and set to the smallest values that produced nearly periodic strides. $\varepsilon_\theta$ and $\varepsilon_\omega$ were the sums of the squared differences between the initial and final segment angular positions and velocities, respectively, and encouraged periodic kinematic states. $\varepsilon_{\text{pas}}$ was the sum of the integrals of the squared passive joint moments, and discouraged joint hyperextension. Since we were interested in the maximum steady speed, $\varepsilon_{\text{grf}}$ was the ratio between the absolute braking and propelling impulses of the horizontal GRF component, with the larger impulse in the numerator, minus one to make the optimal value of zero.

**Force-velocity adjustments.** Following the initial simulation, additional simulations were performed with either $v_{\text{max}}$, $a_R$, or $C_{\text{ecc}}$ adjusted in isolation while the other two parameters were held at their nominal values. For all muscles, the nominal $v_{\text{max}}$ and $C_{\text{ecc}}$ were 12 $L_o \, s^{-1}$ and 1.45, respectively, while muscle-specific nominal $a_R$ values are listed in Table 4.1. The ranges of parameter manipulation were identified from a literature search using the PubMed database (www.pubmed.gov). Studies were included if they measured or estimated one or more parameters in live human subjects. From this search, the parameter ranges were set at $v_{\text{max}} = 4$-$14 \, L_o \, s^{-1}$, $a_R = 0.1$-$0.5$, and $C_{\text{ecc}} = 1.25$-$1.65$. $a_R$ and $C_{\text{ecc}}$ were adjusted in increments of 0.2 (three simulations each). Values for $v_{\text{max}}$ were less consistent in the literature, so $v_{\text{max}}$ was adjusted in finer increments of 2 $L_o \, s^{-1}$ (six simulations).

The initial horizontal velocity of the hip heavily influenced the maximum sprinting speed. Theoretically, the model could achieve a supra-maximal speed by always selecting the maximum value for this variable (akin to an external “towing force”; Mero & Komi, 1986). Conversely, speed could be limited if the allowed range of this variable was too
narrow (akin to a treadmill belt set at a sub-maximal speed). To avoid these situations, if an optimization converged with an initial horizontal hip velocity above 98% of the maximum permitted value, the optimization was repeated with a higher upper bound (± two between-subjects standard deviations).

4.3. Results

4.3.1. Subject Performance

The average sprinting speed of the subjects was 6.42±0.61 m s$^{-1}$. The average stride length and stride frequency were 3.21±0.30 m and 2.01±0.24 Hz, respectively. The mean time histories of experimental joint angles and GRF (Fig. 4.2) and EMG timing (Fig. 4.3) were consistent with other studies (Mann & Hagy, 1980; Thelen et al., 2005b).

4.3.2. Initial Simulation

The model sprinted at 6.75 m s$^{-1}$ with a stride length of 3.39 m and a stride frequency of 1.99 Hz. The model’s speed was 5% faster than the mean speed of the subjects, who may have been constrained by the length of the indoor runway. The joint angles and GRF of the simulation generally fell within two standard deviations of the mean experimental data (Fig. 4.2) and the optimized muscle excitations compared favorably to the EMG on/off timing (Fig. 4.3), even though none of the experimental data were explicitly tracked. The stance duration of the simulation (28% of the stride) was shorter than the mean of the human subjects (37±5%) but within the range observed (26-42%). The zero-lag cross-correlation between the EMG linear envelopes and the muscle model activations averaged 0.49, with a range of 0.26 (rectus femoris) to 0.81 (soleus).
4.3.3. Sensitivity to Force-Velocity Parameters

Across all the additional simulations (12 simulations total), the sprinting speed varied from 5.22-7.17 m s⁻¹. The metabolic cost of transport averaged 5.52 J m⁻¹ kg⁻¹ and did not correlate with speed \((p = 0.55; R^2 = 0.11)\). These results are comparable to estimates in sprinting humans of around 6 J m⁻¹ kg⁻¹ (Weyand & Bundle, 2005) and agree with the invariance of the cost of transport with respect to speed noted by (Margaria et al., 1963), although they studied slower running speeds.

**Maximum shortening velocity.** As \(v_{max}\) was increased incrementally from 4 to 14 \(L_o\) s⁻¹, the maximum sprinting speed increased steadily from 5.2 to 6.9 m s⁻¹ (Fig. 4.4). Most of the increase in speed was accounted for by an increase in stride length, which increased from 2.73 to 3.46 m (+27%) as speed increased between \(v_{max} = 4\) and 10 \(L_o\) s⁻¹ then plateaued for the three fastest simulations (Fig. 4.4). Stride frequency increased from 1.91 Hz at \(v_{max} = 4\) \(L_o\) s⁻¹ to 1.99 Hz at \(v_{max} = 14\) \(L_o\) s⁻¹ (+4%). The duration of the stance phase, in absolute or relative units, was relatively unaffected and did not show a consistent pattern with \(v_{max}\), ranging from 129 to 144 ms and 25 to 27% of the stride duration.

The contractile component velocities during the stride (Fig. 4.5) were generally unaffected by changes in \(v_{max}\), although velocities tended to be higher as \(v_{max}\) increased, particularly in comparison to the slowest simulation \((v_{max} = 4\) \(L_o\) s⁻¹\). The shortening velocities of the plantarflexors (soleus and gastrocnemius) near toe-off increased with each increase in \(v_{max}\), although the muscles were not generating large forces at this time. The discontinuities near 50% of the stride in some panels of Fig. 4.5 (and the other figures of this chapter) were due to the assumption of bilateral symmetry and the assumption that initial contractile component velocities were zero.
According to the force-velocity relationship, since contractile component velocities were similar between $v_{\text{max}}$ conditions (Fig. 4.5), the muscles should have been capable of developing larger forces as $v_{\text{max}}$ increased, since they shortened at rates further from $v_{\text{max}}$. Evidence for this trend is shown for some of the muscles in the force profiles of Fig. 4.6. Average muscle forces during stance, early swing, and late swing (Fig. 4.7) indicate the general patterns in muscle forces as $v_{\text{max}}$ increased, but the following description refers to data in Fig. 4.6. As $v_{\text{max}}$ increased from 4 to 14 $L_o$ s$^{-1}$, the peak force generated by iliopsoas (the primary hip flexor) in early swing increased from 40 to 90% $F_o$. To arrest this motion, the peak force generated by hamstrings (a biarticular hip extensor and knee flexor) immediately prior to foot contact increased from 15 to 25% $F_o$. The peak force in the uniarticular knee flexor (biceps femoris short head) in early swing increased from 25 to 95% $F_o$. In the stance phase, the peak force generated in mid-stance by glutei (primary hip extensor) and vasti (primary knee extensor) increased from 20 to 67% $F_o$ and 37 to 72% $F_o$, respectively. Forces in the ankle muscles and rectus femoris did not show a consistent trend with $v_{\text{max}}$ in stance or swing.

**Shape parameter.** As the shape parameter $a_R$ was increased from 0.1 to 0.3 to 0.5, the sprinting speed increased from 5.60 to 6.75 to 7.17 m s$^{-1}$. Stride lengths at these $a_R$ values were 3.08, 3.39, and 3.60 m, and stride frequencies were 1.82, 1.99, and 1.99 Hz.

Changing $a_R$ did not induce large changes in contractile component velocities (Fig. 4.8), which enabled muscles to generate more force when shortening at these similar velocities (Figs. 4.9 and 4.10). During stance, the peak forces in all nine muscles increased as $a_R$ increased from 0.1 to 0.5. The largest changes were seen in the peak mid-stance forces of glutei (38 to 77% $F_o$) and vasti (25 to 75% $F_o$). In the swing phase, iliopsoas generated
more force throughout swing as $a_R$ increased, and the peak force generated in early swing by biceps femoris increased from 52 to 78% $F_o$. The hamstring force in the second half of swing increased from nearly zero with $a_R = 0.1$ to an average of 15% $F_o$ at $a_R = 0.3$ or 0.5. The peak gastrocnemius force in late swing increased from 36 to 73% $F_o$.

**Eccentric plateau.** The sprinting speeds achieved with the three $C_{ecc}$ values (1.25, 1.45, and 1.65) were 6.23, 6.75, and 6.70 m s$^{-1}$, respectively. The associated stride lengths were 3.17, 3.39, and 3.36 m, and the stride frequencies were 1.96, 1.99, and 1.99 Hz. In other words, weakening the plateau force below its nominal value decreased speed, but strengthening the plateau force did not increase speed.

Like the other sets of simulations, changing $C_{ecc}$ did not have a large affect on the velocities over which the muscle contractile components operated (Fig. 4.11). Muscle force profiles (Fig. 4.12) and average muscle forces during stance and swing (Fig. 4.13) were generally similar for most muscles. With the two stronger $C_{ecc}$ values (1.45 and 1.65), gastrocnemius generated slightly more force during late swing, and iliopsoas generated slightly more force during early swing. Both muscles were acting eccentrically at these times (Fig. 4.11). The peak biceps femoris force in early swing increased as $C_{ecc}$ increased (63 to 97% $F_o$) but the force generated during this time by the other knee flexor (hamstring) decreased (25 to 11% $F_o$). Both muscles were nearly isometric at this time ($\dot{L}_{CC} < 1$).

### 4.4. Discussion

There is ample evidence in the sprinting biomechanics literature that the force-velocity relationship ultimately limits human sprinting speed. This evidence comes from a wide variety of sources, including theory (e.g. Furusawa et al., 1927), experiments (e.g.
Weyand et al., 2000), computer simulations (e.g. Lee & Piazza, 2009), and conclusions from review articles (van Ingen Schenau et al., 1994). Most of these studies have offered a general conclusion without indicating how specifically the force-velocity relationship limits sprinting speed. In this study, we sought to clarify this issue by examining how specific parameters that define the force-velocity relationship affect sprinting speed. Confidence in the accuracy of the simulations and the ability to generalize their results to human sprinting is gained from comparisons with the experimental data (Figs. 4.2 and 4.3): the nominal simulation sprinted at a realistic speed with joint angles, GRF, and muscle excitations that generally resembled those of sprinting humans.

We found that all three parameters ($v_{max}$, $a_R$, and $C_{ecc}$) increased the maximum sprinting speed to some extent when they were increased from the smallest to largest values identified in the literature. The speed range of the simulations (5.2-7.1 m s$^{-1}$) was similar to the speed range of the human subjects in this study (5.6-7.3 m s$^{-1}$), although it is worth noting that the subjects were likely not running at their maximum steady speed due to the short length of the available runway (30 m, with the speed measured at 20 m). As shown in Figure 4.1, each parameter has a different effect on the force-velocity relationship. The maximum shortening velocity $v_{max}$ increases the amount of force that can be produced at fast shortening velocities, the shape parameter $a_R$ increases the amount of force that can be produced at intermediate shortening velocities, and the eccentric plateau $C_{ecc}$ increases the amount of force that can be generated eccentrically. Regardless of which parameter was manipulated, increases in maximum sprinting speed were accomplished mostly by an increase in stride length rather than stride frequency. The mechanism behind this change was that while the muscle contractile component velocities were relatively consistent regardless of parameter
values, they could generate larger forces at these velocities as parameter magnitudes increases (Figs. 4.5-4.13). This finding is consistent with the theory of Weyand et al. (2000) that the force-velocity relationship limits speed by limiting the rate of force development rather than the rate of lower limb motions. However, the effect was seen in both the stance and swing phases, while their theory proposed that speed is limited by the dynamics of stance rather than swing.

The fastest speed (7.13 m s\(^{-1}\)) was attained when \(a_R\) was increased to 0.5 for all muscles. Within the context of the musculoskeletal model, this value corresponds to a fiber type distribution of 100% fast-twitch fibers for all muscles (Eq. 4.1). Similarly, the simulation with \(a_R = 0.1\) corresponds to a distribution of entirely slow-twitch fibers for all muscles. Human lower extremity muscles are of predominantly “slow” or “mixed” fiber compositions, with fast-twitch proportions peaking at 50-60% in the general population (Gollnick et al., 1972). Proportions in non-human mammals are often more extreme, but even the fastest mammal on earth (the African cheetah, who can achieve speeds in excess of 30 m s\(^{-1}\)) does not exceed 60-80% in its large lower limb muscles (Williams et al., 1997). Even with this physiologically unrealistic value for \(a_R\), a realistic sprinting speed for this population of subjects was achieved. The average speed of elite competitive female sprinters, for example, can exceed 9 m s\(^{-1}\) during a 100-m dash, with even faster speeds during the steady speed phase of the sprint. A key component that could explain this difference, and which was untested in this study, is the maximum isometric muscle force \(F_o\). In sensitivity analyses, we have found that the strength of the model needs to be doubled in order to achieve “Olympic” caliber speeds, which does not seem unreasonable. \(F_o\) was not included in this study because it affects not only the force-velocity relationship, but also the
force-length and force-extension relationships. Decoupling $F_o$ from these three relationships leads to numerical problems, as Hill-based muscle model algorithms typically use the force output from the force-length and/or force-extension relationships to determine the contractile component velocity.

Sprinting speed increased incrementally as $a_R$ increased ($R^2 = 0.93$). The same was true when $v_{max}$ was increased incrementally ($R^2 = 0.98$), although the fastest sprinting speed achieved by increasing $v_{max}$ (6.89 m s$^{-1}$ when $v_{max} = 14 L_o$ s$^{-1}$) was slower than the fastest speed achieved by increasing $a_R$ (7.13 m s$^{-1}$ when $a_R = 0.5$). This finding is reasonable in the context of how these two variables affect the shape of the force-velocity relationship (Fig. 4.1): $a_R$ has its greatest effect near the intermediate shortening velocities, while $v_{max}$ has its greatest affect near the fastest shortening velocities. Since the contractile component tends to operate in the intermediate range of 2 to 8 $L_o$ s$^{-1}$ during sprinting (Figs. 4.5, 4.7, and 4.9), it follows that $a_R$ would have a greater effect on maximum speed. Like the maximum value for $a_R$, the maximum value of $v_{max} = 14 L_o$ s$^{-1}$ may not be physiologically realistic for all muscles. In the present study, a $v_{max}$ of at least 8 $L_o$ s$^{-1}$ was needed to achieve a sprinting speed within one standard deviation of the mean of the human subjects. Decreasing $v_{max}$ to 6 or 4 $L_o$ s$^{-1}$ greatly slowed the model. Domire and Challis (2010) recently reported a similar result in a simulation of vertical jumping. Most reports of human $v_{max}$ in vivo or in vitro are closer to (or even below) 4 $L_o$ s$^{-1}$ than 12 $L_o$ s$^{-1}$. Although though these estimates are not necessarily error-free, it is paradoxical that seemingly unrealistic values for $v_{max}$ are needed to achieve otherwise realistic simulation results. A possible explanation is the fact that the contractile component of the Hill muscle model is not an anatomical analog to the fibers, fascicles, sarcomeres, or any specific structure of real muscle, since the model is
phenomenological in nature. The lengths of these structures, either in their optimal or resting states, are typically used to scale velocity measurements in real muscle. Mechanistic models based on the crossbridge theory (Huxley, 1957) have more direct anatomical analogs, but are numerically impractical for optimization problems and do not simulate active lengthening as well as Hill-based models (Zahalak, 1981; Cole et al., 1996b). In addition, $v_{\text{max}}$ is notoriously difficult to quantify experimentally since a true zero-force condition is unattainable. $v_{\text{max}}$ is usually estimated by extrapolating force-velocity measurements near the zero-force intercept, but the non-linear relationship between force and velocity makes extrapolation to the intercept very sensitive to experimental noise.

The final tested parameter, the eccentric plateau ($C_{\text{ecc}}$), did not have a large effect on sprinting speed. Decreasing $C_{\text{ecc}}$ by 14% decreased speed by only 8%, while increasing $C_{\text{ecc}}$ by 14% actually decrease speed slightly, by 0.7%. This finding suggests that $C_{\text{ecc}}$ does not play a critical limiting role in human sprinting speed. By examining Figs. 4.5-4.13, it is seen that most of the increases in muscle force as sprinting speed increased occurred when the contractile component was acting concentrically or was nearly isometric, further suggesting that the eccentric plateau force does not critically limit sprinting speed. The maximum tested $C_{\text{ecc}}$ value of 1.65 is likely near the maximum possible force that human muscle can safely generate. Dudley et al. (1990) found that the peak eccentric torque of the quadriceps was 1.4 times the maximum isometric torque, while Pain and Forrester (2009) recently estimated a $C_{\text{ecc}}$ range of 1.30-1.35 for individual knee extensors. Zajac (1989) reported that mammalian tendons can sustain forces up to 1.8 times $F_o$ before rupturing.

Why do alterations in force-velocity characteristics allow runners to attain higher speeds? As mentioned earlier, two different theories on specifically how, where, and when
the force-velocity relationship limits sprinting speed have been proposed: the “energy management” theory of Chapman and Caldwell (1983b), and the “time to generate force” theory of Weyand et al. (2000). The former centers on the swing phase and posits that speed is limited by the rate at which the hip flexors can generate enough energy in the lower limb to rapidly move it forward and complete the swing phase, and by the subsequent inability of the knee flexors to remove this energy in time to position the foot properly for ground contact. The latter theory focuses on the stance phase and states that speed is limited by insufficient ground contact time for generating sufficiently large support forces. These two theories are not necessarily in conflict, but we sought to investigate if one or the other plays a dominant role in human sprinting. It should be noted that the Weyand et al. (2000) theory was posed in a between-subjects context (i.e. seeking to explain why some subjects are faster than others), while the Chapman and Caldwell (1983b) theory was posed in a within-subjects context (i.e. seeking to explain why an individual ultimately cannot run any faster). We found support for both theories in the present study.

The durations of stance and swing did not vary with speed in the sprinting simulations \( p = 0.49; R^2 = 0.14 \), indicating that the lower limbs were not cycled through the swing phase more rapidly when the model sprinted faster. In addition, the peak forces of the major extensor muscles (glutei and vasti) tended to increase as running speed increased (Figs. 4.6, 4.7, 4.9, and 4.10). These muscles are primarily responsible for supporting the body weight during running (Hamner et al., 2010). These results support the theory of Weyand et al. (2000) that sprinting speed is limited by the ability to support the body weight in stance, not by the ability to quickly swing the limb. Hamner and Delp (2010) reported that the plantarflexors (soleus and gastrocnemius) make the greatest contribution to the propulsive
component of the GRF during stance. As speed increases, the stance time decreases and the braking and propelling peaks of the horizontal GRF component increase (Kuitunen et al., 2002). The gastrocnemius force during stance increases as the shape parameter $a_R$ and sprinting speed increased (Figs. 4.9 and 4.10), but otherwise the plantarflexor forces did not show a consistent relationship with increasing speed. This result suggests that the force-velocity relationship has a greater limiting effect in the vertical direction than in the horizontal direction. However, although the GRF is often analyzed as a set of two or three orthogonal forces, we note that it is in reality a single force that can be resolved into any arbitrary number of components in various directions.

The swing phase requires the entire lower limb to clear the ground so that the foot can be positioned in preparation for the next stance phase. Chapman and Caldwell (1983b) described this phase of sprinting as a problem of generating enough kinetic energy within the lower limb to quickly swing the limb, while also removing enough of this energy in order to accurately position the limb for the next stance period. They suggested that the hip flexors (e.g. iliopsoas, rectus femoris), as well as energy transfer from the contralateral limb through the pelvis, were primarily responsible for energy generation, while the knee flexors (e.g. hamstrings, gastrocnemius, various smaller uniarticular muscles) were responsible for energy dissipation. In the present study, increasing speed was usually accompanied by an increase in the force generated by iliopsoas, either in early swing or across the entire swing phase. This increase was usually accompanied by an increase in biceps femoris force in early swing (Fig. 4.7) and an increase in the force and/or eccentric velocity of gastrocnemius or hamstrings in late swing (Figs. 4.5-4.10). Biceps femoris force in early swing serves to flex the knee, which decreases the moment of inertia of the entire limb and allows the hip
acceleration resulting from the iliopsoas force to generate more kinetic energy. Knee flexor forces (via hamstrings and gastrocnemius) in late swing serves to arrest this motion in preparation for foot contact. These findings are consistent with the proposed energy management problem posed by Chapman and Caldwell (1983), and provide evidence at the individual muscle level in support of their theory, which was based on calculations of resultant joint torques. However, since hamstrings and gastrocnemius are lengthening in late swing, the lack of a strong relationship between $C_{ecc}$ and sprinting speed and the insensitivity of late-swing eccentric force and velocity in these muscles when $C_{ecc}$ was increased (Figs. 4.11-4.13) suggests that the ability to generate energy, rather than dissipate it, plays the dominant role in this theory.

In summary, the shape parameter $a_R$ had the greatest effect on maximum speed in forward dynamics simulations of sprinting. The maximum shortening velocity $v_{max}$ had a moderate effect, and the eccentric plateau $C_{ecc}$ had a small effect. These results were due to the contractile component velocities experienced during sprinting, which were moderate rather than high. $a_R$ has its greatest effect on the shape of the force-velocity relationship at moderate velocities and thus had a greater effect on sprinting speed than $v_{max}$, which has its greatest effect at fast velocities. Relatively high values of $v_{max}$ were needed to achieve realistic speeds. The $C_{ecc}$ results suggest that the ability to generate sufficient eccentric forces does not play a major role in limiting sprinting speed. Support was found at the individual muscle level for both the stance and swing phase theories of how the force-velocity relationship limits speed, although the ability of the knee flexors to arrest the motion of the lower limb in late swing did not appear to be a critically limiting factor.
Table 4.1. Muscle model parameters used in the force-velocity relationship derived from the human subjects (Appendix A). $F_o =$ maximum isometric force; $L_o =$ optimal contractile component length; $FT =$ proportion of fast-twitch muscle fibers; $a_R$ and $b_R =$ dimensionless force-velocity constants. $b_R$ was calculated assuming $v_{max} = 12 \frac{L_o}{s}$.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>$F_o$ (N)</th>
<th>$L_o$ (cm)</th>
<th>$FT$</th>
<th>$a_R$</th>
<th>$b_R$ (s$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iliopsoas</td>
<td>2324</td>
<td>12.0</td>
<td>0.52</td>
<td>0.31</td>
<td>3.70</td>
</tr>
<tr>
<td>Glutei</td>
<td>4072</td>
<td>14.0</td>
<td>0.47</td>
<td>0.29</td>
<td>3.46</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>5664</td>
<td>11.0</td>
<td>0.67</td>
<td>0.37</td>
<td>4.42</td>
</tr>
<tr>
<td>Vasti</td>
<td>1008</td>
<td>9.5</td>
<td>0.59</td>
<td>0.34</td>
<td>4.03</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>2680</td>
<td>14.5</td>
<td>0.31</td>
<td>0.22</td>
<td>2.69</td>
</tr>
<tr>
<td>Hamstrings</td>
<td>5888</td>
<td>14.0</td>
<td>0.41</td>
<td>0.26</td>
<td>3.17</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>1464</td>
<td>8.8</td>
<td>0.25</td>
<td>0.20</td>
<td>2.40</td>
</tr>
<tr>
<td>Soleus</td>
<td>3000</td>
<td>4.9</td>
<td>0.19</td>
<td>0.18</td>
<td>2.11</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>2816</td>
<td>7.1</td>
<td>0.50</td>
<td>0.30</td>
<td>3.60</td>
</tr>
</tbody>
</table>
Fig. 4.1. Sensitivity of the muscle force-velocity relationship (van Soest and Bobbert’s (1993) formulation) to incremental adjustments in (a) the maximum shortening velocity (from 4-12 Lo/s), (b) the shape parameter $a$ (from 0.1-0.5), and (c) the eccentric plateau force (from 1.2-1.6). Arrows indicate trend directions for increasing parameter magnitude. Axes are scaled by the optimal contractile component length (Lo) and the maximum isometric force (Fo). The crosshair indicates zero velocity and maximum isometric force.

Figure 4.2. Joint angles and GRF components vs. the stride cycle from the human subjects (dashed lines) and the initial simulation (solid lines). Shaded area is +/- two between-subjects standard deviations around the mean of the human subjects. The stride begins and ends at initial contact of the right foot.
Figure 4.3. Optimized muscle excitations (thin lines), activations (thick lines), and EMG linear envelopes (dashed lines) for the sprinting simulation. ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at heel-strike. Vertical dashed lines indicate toe-off. Linear envelopes were scaled by values from maximum isometric contraction. No EMG data were available for ILP and BF.
Figure 4.4. Speeds (diamonds), stride lengths (triangles), and stride frequencies (squares) of the sprinting simulations as the maximum shortening velocity was increased incrementally from $4-14 \, \text{L}_o \, \text{s}^{-1}$. 
Figure 4.5. Profiles of contractile component velocity (units of $L_o s^{-1}$) vs. the stride cycle for six maximum shortening velocities ($v_{max} = 4, 6, 8, 10, 12, \text{ and } 14 \, L_o s^{-1}$). ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at initial contact of the right foot. Vertical dashed lines indicate toe-off. Negative/positive velocities are lengthening/shortening.
Figure 4.6. Profiles of muscle forces vs. the stride cycle for six maximum shortening velocities ($v_{\text{max}} = 4, 6, 8, 10, 12, \text{and } 14 \ L_0 \ s^{-1}$). ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at initial contact of the right foot. Vertical dashed lines indicate toe-off. The y-axis is scaled to the maximum isometric force ($F_o$).
Figure 4.7. Individual muscle forces (scaled to $F_o$) at six different maximum shortening velocities ($v_{\text{max}}$, in $L_o \text{s}^{-1}$), averaged over (a) the stance phase, (b) the first half of swing, and (c) the second half of swing. Within each muscle (horizontal axis), $v_{\text{max}}$ increases from left to right.
Figure 4.8. Profiles of contractile component velocity (units of $L_o \text{s}^{-1}$) vs. the stride cycle for three shape parameter values ($a_R = 0.1, 0.3, \text{and} 0.5$). ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at initial contact of the right foot. Vertical dashed lines indicate toe-off. Negative/positive velocities are lengthening/shortening.
Figure 4.9. Profiles of muscle forces vs. the stride cycle for three shape parameter values ($\alpha_R = 0.1, 0.3, \text{ and } 0.5$). ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at initial contact of the right foot. Vertical dashed lines indicate toe-off. The y-axis is scaled to the maximum isometric force ($F_o$).
Figure 4.10. Individual muscle forces (scaled to $F_o$) at three different shape parameter values ($a_R$), averaged over (a) the stance phase, (b) the first half of swing, and (c) the second half of swing. Within each muscle (horizontal axis), $a_R$ increases from left to right.
Figure 4.11. Profiles of contractile component velocity (units of $L_o \, s^{-1}$) vs. the stride cycle for three eccentric plateau values ($C_{ecc} = 1.25, 1.45,$ and $1.65$). ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at initial contact of the right foot. Vertical dashed lines indicate toe-off. Negative/positive velocities are lengthening/shortening.
Figure 4.12. Profiles of muscle forces vs. the stride cycle for three eccentric plateau values ($C_{ecc} = 1.25, 1.45$, and 1.65). ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at initial contact of the right foot. Vertical dashed lines indicate toe-off. The y-axis is scaled to the maximum isometric force ($F_o$).
Figure 4.13. Individual muscle forces (scaled to $F_o$) at three different eccentric plateau values ($C_{ecc}$), averaged over (a) the stance phase, (b) the first half of swing, and (c) the second half of swing. Within each muscle (horizontal axis), $C_{ecc}$ increases from left to right.
CHAPTER 5
VIRTUAL AGING OF THE MUSCULAR SYSTEM AND ITS EFFECTS ON RUNNING BIOMECHANICS

5.1. Introduction

Running is one of the most popular forms of exercise among the general population. The high rate of energy expenditure and resulting cardiovascular adaptations are beneficial for metabolic health (Williams, 1997), and the high mechanical loads experienced by the lower extremities can be beneficial for skeletal health (Woo et al., 1981) without increasing the risk of osteoarthritis (Wilick & Hansen, 2010). With recent emphasis on the importance of exercise for maintaining metabolic and musculoskeletal health over the lifespan (Nelson et al., 2007), running is becoming a more popular activity for older adults (Jokl et al., 2004), with “older” typically defined as ages 65+.

Recent studies have identified biomechanical distinctions between the running gaits of young and older adults (Bus, 2003; Cavagna et al., 2008a; Fukuchi & Duarte, 2008). When matched for speed, older runners tend contact the ground with a more flexed knee, flex and extend the knee through a smaller range of motion, and experience greater vertical impact peaks and loading rates (Bus, 2003). They also use shorter strides, greater stride rates to run at a given speed (Cavagna et al., 2008a; Fukuchi & Duarte, 2008) and perform less external work on the center of mass per stride (Cavagna et al., 2008a). Older runners also experience different injury patterns than young runners, in that they are injured more frequently and sustain more injuries to soft tissues such as the calves and hamstrings.
Resistance training increases muscle mass and strength even in very old (age 80+) individuals (Aagaard et al., 2010), but older adults do not appear to be selectively weaker in the calves and hamstrings compared to other muscle groups. It is unknown how the loading environments of these muscles compare between young and older running gaits, or how the strength of these muscles affects their loading during running.

It has been suggested that these differences are linked to age-related changes in the mechanical properties of skeletal muscle (Bus, 2003; Cavagna et al., 2008a). Cross-sectional studies of muscular properties have consistently found that muscles of older adults are weaker, slower, and differ in stiffness compared to those of young adults (Narici et al., 2008). Causal links between altered muscular properties and age-related differences in running mechanics and injury patterns would be beneficial for recommending appropriate exercise programs for older adults to obtain the health benefits of running while minimizing injury risk, but these relationships are difficult to establish with experiments on live human subjects. Longitudinal studies of aging, while possessing considerably more inferential power than cross-sectional studies, are impractical due to the restrictively long time investment required. Even if such studies could be performed, some variables of interest (mechanical parameters and internal loading at the individual muscle level) cannot be measured in live human subjects. Computer simulations can be useful in these situations due to their predictive capabilities and the ability to access variables that cannot easily be measured in humans. In particular, performance-based simulations, which maximize the quality of the simulated movement according to a specified optimality criterion without tracking experimental data (Chapter 3), can be used to predict the biomechanical movement response to adjustments in model characteristics, such as muscular properties (Chapter 4).
Therefore, the purpose of this study was to determine the effect of “virtual” aging of the muscular system on the mechanics and energetics of a computer simulation of human running. We hypothesized that adjusting the muscular properties to be representative of older adults would result in increased knee flexion at heel-strike, reduced knee angle range of motion, and increased stride frequency / decreased stride length. Since running injuries arise most directly from the loading (stress and strain) of susceptible musculoskeletal structures (Hreljac, 2004), we hypothesized that aging the model would increase the stress and strain of hamstrings, gastrocnemius, and soleus, and that increasing the maximum isometric strength of these muscles (i.e. a simulated strength training intervention) would reduce their loading.

5.2. Methods

Forward dynamics simulations of running were generated using the mathematical modeling and optimization framework described in detail in Chapter 3. Briefly, the model consisted of 11 rigid segments (trunk, thighs, legs, feet, toes) linked at eight ideal pin joints (hips, knees, ankles, metatarsophalanges) and confined to movement in the sagittal plane. Joint ranges of motion were restricted by passive restoring torques (Riener & Edrich, 1999). Eighteen Hill-based muscle models (nine per each lower limb; iliopsoas, glutei, vasti, biceps femoris (short head), tibialis anterior, soleus, rectus femoris, hamstrings, gastrocnemius) actuated the joints (Appendix A). Each muscle model had a contractile component (CC) in series with an elastic component (SEC). Metabolic energy expenditure was calculated according to Umberger et al. (2003). Viscoelastic frictional elements on the plantar surfaces of the feet and toes generated the model’s ground reaction forces. A vertical sinusoidal force on the shoulder represented arm swing kinetics.
The computer model was parameterized to represent the anthropometry and strength characteristics of a group of 12 young adult females (mean±sd age = 27±6 years). The model had a mass of 61.0 kg and a standing height of 1.66 m. Experimental kinematic, ground reaction force (GRF), electromyographic (EMG), and metabolic data were collected from the subjects as they ran at a self-selected speed (3.80±0.51 m s$^{-1}$). These data were used to generate a data tracking simulation (e.g. Miller & Hamill, 2009) and for subsequent validation purposes. The EMG data were processed into a binary “on/off” timing signal as described in Chapter 3.

5.2.1. Young Simulation

To generate the initial “young” performance-based simulation of running, the muscle model excitation signals that maximized an objective function were found using simulated annealing (Higginson et al., 2005). The simulation began at heel-strike of the right foot and ended at heel-strike of the left foot. A full stride was reconstructed from this single step by assuming bilateral symmetry (Anderson & Pandy, 2001a). A data tracking solution that replicated the mean experimental joint angles and ground reaction forces was used as an initial guess. Parameters defining the arm swing kinetics, the initial kinematic state, the stiffness of the metatarsophalangeal joints, and the stride duration were also allowed to vary within reasonable bounds. The objective function was:

$$J = OC - \left( 0.01\varepsilon_\theta + 0.0001\varepsilon_\omega + 0.001\varepsilon_{pas} \right)$$

(Eq. 5.1)

where $OC$ is the optimality criterion and the other right-hand side terms are penalty terms. $\varepsilon_\theta$ and $\varepsilon_\omega$ were the sums of the squared differences between the initial and final segment angular positions and velocities, respectively, and encouraged periodic kinematic states. $\varepsilon_{pas}$
was the sum of the integrals of the squared passive joint moments, and discouraged joint hyperextension. The weighting coefficients on the penalty terms were adjusted in preliminary simulations and set to the smallest values that produced nearly periodic strides. The optimality criterion was:

$$OC = -\frac{1}{t_f} \sum_{i=1}^{m} \left( \int_0^{t_f} A_i^2 dt \right)$$  

(Eq. 5.2)

where $A_i(t)$ is the time-varying activation of the $i^{th}$ muscle in an $m$-muscle system, and $t_f$ is the stride duration. This criterion is based on the rationale that by using the unweighted muscle activations (all varied nondimensionally on [0,1]), exhaustion of any single muscle and a consequential fatigue-induced change in movement are avoided, regardless of the size or strength of the muscle. It is sometimes referred to as “minimum fatigue” (Ackermann & van den Bogert, 2010) and generated the most realistic performance-based simulations of running compared to other candidate criteria in Chapter 3.

### 5.2.2. Age-related Adjustments in Muscle Model Parameters

To simulate the aging of the muscular system, a variety of changes in muscle model parameters were imposed. The variables selected for change and the magnitude of change were determined by a review of literature. Values for the muscle model parameters referred to in this section are found in Appendix A.

**Muscle strength and fiber type.** Compared to young adults, healthy older adults are weaker in isometric strength, primarily due to a loss of muscle mass and selective atrophy of fast-twitch muscle fibers (Lexell, 1995). The decrement in strength varies depending on the muscle group considered (e.g. Hortobagy et al., 1995; Pousson et al., 2001; Narici et al., 2005) but is reported to be about 30% on average (Raj et al., 2010). The decrement in fast-
twitch muscle fibers also varies depending on the muscle group (e.g. Proctor et al., 1995; Klein et al., 2003) but is also about 30% on average. Based on these findings, values for the maximum isometric force ($F_o$) and the proportion of fast-twitch fibers ($FT$) were both reduced by 30% for all muscles. In the muscle model, $F_o$ is the product of the muscle’s physiological cross-sectional area (PCSA; Haxton, 1944) and the specific tension, which was set at 40 N cm$^{-2}$ for all muscles in the young model. Older muscles have both smaller PCSAs and weaker specific tensions than young muscles (Larsson et al., 1997; Yu et al., 2007), although the decrement in PCSA is comparatively larger and there is equivocal evidence on differences in specific tension between young and older women (Akima et al., 2001; Yu et al., 2007). Based on these findings, we reduced $F_o$ by 30% by reducing PCSA by 20% and specific tension by 12.5%. This adjustment also reduced muscle mass by 20%.

**CC force-velocity relationship.** In the present muscle model, reducing $FT$ also reduced the amount of force that muscles can produce at moderate shortening velocities, which is consistent with experiments (e.g. Hortobagyi et al., 1995; Pousson et al., 2001). There is also evidence that the maximum shortening velocity ($v_{max}$) is slower in older adults (e.g. Larsson et al., 1979; Narici et al., 2005) by about 20% on average (Raj et al., 2010). We therefore reduced $v_{max}$ from 12.0 to 9.6 optimal contractile components lengths per second for the “older” simulation.

**SEC force-extension relationship.** There are some equivocal findings in the literature regarding differences in series elastic compliance with respect to age. However, most studies have reported that the absolute stiffness (slope of the force-extension curve at an absolute force level) is reduced with aging (Magnusson et al., 2008). Due to the nonlinear shape of the series elastic component’s force-extension curve (Caldwell, 1995), this
adaptation was accomplished by reducing $F_0$ as mentioned previously. The SEC strain when the muscle generated a force of $F_0$ was set to 4% of the unloaded SEC length in both the young and older models. Since $F_0$ was reduced by 30% in the older model, the older SECs were more compliant at an absolute force level.

**CC force-length relationship.** There is no convincing evidence that the active force-length relationship of muscle depends on age (van Schaik et al., 1994; Winegard et al., 1997). This relationship was therefore unchanged in the “older” simulation.

**Passive force-length relationship.** The relative amount of passive tissue in muscles is greater in older adults (Kent-Braun et al., 2000), which may increase the passive stiffness of older muscles. This theory is consistently supported in animal models, where parallel elastic stiffness can be measured directly (e.g. Kovanen, 1989), but the in vivo evidence in older adult humans is equivocal (Gajdosik, 1999; Porter et al., 1997; Valour & Pousson, 2003; Ochala et al., 2004). However, the finding of a reduced range of motion at the hip in older adults has been consistent (James & Parker, 1989; Nonaka et al., 2002; Macedo & Magee, 2009). The passive torque-angle function at the hip (Riener & Edrich, 1999) was modified to reduce the flexion and extension ranges of motion by $8^\circ$ and $13^\circ$, respectively (Nonaka et al., 2002). The ankle and knee functions were unchanged.

**Activation dynamics.** There is evidence that the time courses of muscle activation and deactivation are both slowed with aging, although the evidence is mostly drawn from animal models and is more convincing for deactivation dynamics (Hunter et al., 1999; Margreth et al., 1999; Plant & Lynch, 2002). In the present muscle model, reducing $FT$ decreased the time constants for both activation and deactivation in the excitation-activation relationship (He et al., 1991).
5.2.3. Older Simulations

The process of generating the older simulations was identical to that of the young simulation described in Section 5.2.1, except the muscle model parameters were adjusted as described in Section 5.2.2. The optimal solution from the young simulation was used as an initial guess for the optimization. An extra term was added to the objective function (Eq. 5.1) to encourage the older simulation to run at the same speed as the young simulation:

\[ J = OC - \left( 0.01\epsilon_\theta + 0.0001\epsilon_\omega + 0.001\epsilon_{pas} + 10\epsilon_{speed} \right) \]  

(Eq. 5.3)

\[ \epsilon_{speed} = (v_{old} - v_{young})^2 \]  

(Eq. 5.4)

where \( v_{young} \) and \( v_{old} \) are the average horizontal velocities of the center of mass in the young and old simulations, respectively. To examine the effects of an increase in muscle strength such as might be expected from a resistance training program, the older simulation was re-optimized with \( F_o \) returned to 100% of the young value for hamstrings, gastrocnemius, and soleus by returning their PCSA and specific tension to the young values. Other parameters were retained at their older values.

5.2.4. Analysis

The output simulation variables for comparison were those that have distinguished between young and older runners in the experimental literature (Bus, 2003; Cavagna et al., 2008a; Fukuchi & Duarte, 2008): stride length and frequency, knee flexion angle at heel-strike, knee flexion range of motion, vertical GRF impact peak, and the external work performed on the center of mass. External power was calculated as the dot product between the GRF vector and the velocity vector of the center of mass, and external work was calculated by integrating the external power over the stride time (Cavagna, 1975). Gross
metabolic power was calculated as the sum of the individual muscle metabolic rates (Umberger et al., 2003), plus the energy rates for quiet stance and the arm swing force (Chapter 3). The cost of transport was the ratio of metabolic power and running speed.

Running injuries can be attributed to the internal loading environment of the musculoskeletal system (Hreljac, 2004), which includes the stresses and strains experienced by muscles and tendons. To assess muscular injury potential, particularly in the hamstrings and plantarflexors which are injured more frequently in older runners (McKean et al., 2006), the peak stress of the contractile component (peak force divided by physiological cross-sectional area) and the peak strain of the series elastic component (peak extension divided by unloaded length) were calculated. The knee joint contact force ($F_c$) was calculated from the internal force balance equation in the tibia’s reference frame (Morrison, 1970):

$$F_R = F_m + F_c + F_l$$

(Eq. 5.5)

by subtracting the total muscle force vector ($F_m$) from the resultant joint force vector ($F_R$). $F_m$ was the sum of the muscle force vectors for vasti, rectus femoris, hamstrings, biceps femoris, and gastrocnemius. The ligament force ($F_l$) was assumed zero because the knee did not approach the extremes of its ranges of motion in mid-stance when $F_c$ is large.

5.3. Results

5.3.1. Young Simulation

The young simulation ran at 3.64 m s$^{-1}$ with a stride length of 2.43 m and a stride frequency of 1.50 Hz. The metabolic power and cost of transport were 16.2 W kg$^{-1}$ and 4.44 J m$^{-1}$ kg$^{-1}$, respectively. All of these values were within 5% of the mean values from the human subjects. The simulation’s hip, knee, and ankle angles and GRF were on average
within two between-subjects standard deviations of the experimental means (Fig. 5.1). The optimized muscle excitations in general agreed with the EMG on/off timing (Fig. 5.2).

5.3.2. Virtual Aging

The older simulation ran at nearly the same speed as the young simulation (3.67 m s\(^{-1}\); 0.8% faster) with a 7% higher stride frequency (1.60 Hz) and a 6% shorter stride length (2.29 m). The metabolic power (16.5 W kg\(^{-1}\)) was 2% higher than the young simulation, and cost of transport (4.49 J m\(^{-1}\) kg\(^{-1}\)) was 1% higher.

The joint angles and GRF during the stride are compared between the young and older simulations in Fig. 5.3. The older simulation flexed its knee by an additional 5.6° at heel-strike (young -12.5°, older -18.1°). The knee range of motion decreased by 14.2° in stance (young 33.4°, older 19.2°) and 28.8° in swing (young 87.0°, older 58.2°). The magnitude of the vertical GRF impact peak increased from 2.5 to 3.2 times body weight, and the vertical GRF loading rate increased from 75.3 to 102.8 body weights per second. The older simulation performed 22% less total external work to move the center of mass forward (young 1.31 J m\(^{-1}\) kg\(^{-1}\), older 1.03 J m\(^{-1}\) kg\(^{-1}\)) even though it ran at nearly the same speed. This change was due primarily to a 23% reduction in the vertical center of mass excursion.

Changes in the peak loading of the muscles with aging are summarized in Fig. 5.4. The loading hypotheses were supported for hamstrings and soleus, but gastrocnemius loading was relatively similar regardless of age. In the older simulation, peak stress of the contractile component increased by 32% in hamstrings (young 14.1 N cm\(^{-2}\), older 18.6 N cm\(^{-2}\)) and 56% in soleus (young 31.7 N cm\(^{-2}\), older 49.2 N cm\(^{-2}\)) but only 4% in gastrocnemius (young 28.1 N cm\(^{-2}\), older 29.2 N cm\(^{-2}\)). The peak strain of the series elastic component increased by 12%
in hamstrings (2.8% young, 3.2% older), 12% in soleus (3.8% young, 4.2% older), and 1% in gastrocnemius (3.6% young, 3.7% older). The peak stress of vasti was reduced by 32% in the older simulation (28.9 N cm\(^{-2}\) young, 19.8 N cm\(^{-2}\) older). Consequently, the peak knee joint contact force was reduced by 40% from 12.7 times body weight in the young simulation to 7.6 times in the older simulation. Peak stresses also increased in hamstrings and gastrocnemius, but these changes were not large enough to increase the older simulation’s knee joint contact force in light of the large reduction in peak vasti force and stress.

5.3.3. Virtual Strength Training

After strengthening hamstrings, soleus, and gastrocnemius to their young maximum isometric forces, the speed, stride length and frequency, and knee angle were all within 3% of the initial older simulation. The cost of transport was 4.1 J m\(^{-1}\) kg\(^{-1}\), a 9% decrease from the initial older simulation. The peak muscle stresses and strains in the older simulations with the weaker and stronger hamstrings, soleus, and gastrocnemius are compared in Fig. 5.5. The peak stresses of all three strengthened muscles were reduced, by 16% in hamstrings (18.6 to 15.6 N cm\(^{-2}\)), 21% in soleus (49.3 to 39.0 N cm\(^{-2}\)), and 41% in gastrocnemius (29.3 to 17.3 N cm\(^{-2}\)). The peak strains of these muscles were reduced by 6, 6, and 10%, respectively. The peak stresses of vasti and rectus femoris increased by 20%, which resulted in a 16% increase in the peak knee joint contact force (8.8 bodyweights). The magnitude of the knee joint contact force was still 31% smaller than the young simulation. Peak stresses of other muscles in the strengthened older simulation were similar to the initial older simulation (5% decrease on average).
5.4. Discussion

When studying the aging of muscles, research questions are often of the form, “How do muscular properties change with advancing age, and how do these changes affect performance?” One of the great difficulties in answering these questions is the effort required to observe changes in individuals’ muscular properties as they age from young to older adults. The span of time between these life stages is typically at least 30 years, a prohibitively long period for performing longitudinal studies. Consequently, longitudinal studies of aging in adulthood are quite rare, and much of our knowledge of age-related changes in muscle is drawn from cross-sectional studies (e.g. Karamanidis & Arampatzis, 2006). It can be difficult to draw longitudinal inferences from cross-sectional data due to a litany of potential confounding factors (e.g. activity level and fitness, genetic predisposition, health and injury/disease/medication history, body size). In additional, cross-sectional and longitudinal analyses do not necessarily tell the same story regarding aging and muscle quality (Metter et al., 1999).

The simulation framework presented in this study offers an opportunity to address this issue by performing “virtual” longitudinal studies. The model can be adjusted to represent the aging process, and used to predict the resulting effects on the movement performance. Since the researcher has complete control over what is changed and controlled in the aging process, the framework also permits researchers to draw direct causal inferences between changes in muscular properties and the characteristics of the model’s movement. Since the age-induced changes of the model’s muscular properties were derived from cross-sectional studies, we cannot be certain that they reflect the magnitude of longitudinal changes. However, the simulation approach provides a potential framework for testing the
effects of incremental or isolated changes in muscular properties on movement performance. For example, we found that aging the muscular system increased the loading of hamstrings and the plantarflexors, and reduced the peak knee joint contact force by 40%. These directional changes in loading magnitudes between the young and older simulations correspond to locations where older runners are injured more frequently (hamstrings and plantarflexors) or less frequently (knee). Strengthening the older simulation’s hamstrings and plantarflexors reduced the loading of these muscles such that the peak stresses of hamstrings and soleus were about 50% closer the young values, and the peak gastrocnemius stress was even lower than the young value. The knee joint contact force was still 30% below that of the young simulation. While it remains to be seen which specific muscles and muscular properties explain other age-related changes in running mechanics, such as stride length and mechanical work, the results demonstrate the potential for using the simulation framework to test hypotheses related to aging, muscular properties, human performance, and injury etiology and prevention.

Confidence in generalizing the simulation results to real human running is gained by favorable comparisons with the experimental data (see Figs. 5.1 and 5.2, as well as Chapters 3 and 4). In addition, the changes in running mechanics and energetics induced by virtual aging of the muscular system were remarkably similar to differences observed between young and older runners, and in several cases nearly mirrored these differences on a quantitative basis. Bus (2003) reported that older runners had a more flexed knee at heel-strike by 5.6° and used a 0.1-Hz higher stride frequency, while the simulation increased its knee flexion angle at heel-strike by 5.6° and its stride frequency by 0.1 Hz when its muscles were aged. Changes in the simulation’s knee range of motion during stance (14.2°) and
vertical GRF loading rate (27.5 body weights per second) were also similar to differences (10.0° and 22.0 body weights per second) documented by Bus (2003). Cavagna et al. (2008a) reported the external mechanical work performed on the center of mass averaged 1.2 and 0.9 J m\(^{-1}\) kg\(^{-1}\) for young and older runners, respectively. In the present work, the simulation performed 1.3 J m\(^{-1}\) kg\(^{-1}\) of external work when young and 1.0 J m\(^{-1}\) kg\(^{-1}\) when old. Aging the model increased its vertical GRF impact peak by 0.7 body weights. This change exceeds the difference documented by Bus (2003) of 0.2 body weights, although their subjects ran at a slower speed (~3.35 vs. ~3.65 m s\(^{-1}\)) and were not subject to the rigid body assumption that elevates the contributions of non-muscular forces (Neptune et al., 2004) to the impact force.

Between the young and older simulations, the only difference in the model’s parameterization was the muscle model parameter values. Other than requiring the older simulation to run at the same speed as the young simulation, no changes were made to the objective function, optimality criterion, or optimization framework. The age-induced changes in running mechanics and energetics were therefore directly attributed to changes in muscular mechanical properties. These results support the previous hypothesis that differences in running mechanics between young and older adults are due to differences in muscle mechanical properties (Bus, 2003; Cavagna et al., 2008a). The simulations can be viewed as a virtual longitudinal study of aging and resistance training of an individual, and were not subject to many of the potential confounding factors of a cross-sectional study (e.g. genetic variance). Even so, the simulation results qualitatively (and many times quantitatively) replicated the findings of cross-sectional comparisons between young and older runners. This result suggest that age-related cross-sectional differences in muscular
properties and running mechanics may generalize to longitudinal conclusions, but this suggestion is made cautiously as it has not been investigated experimentally.

The knee flexion angle of the young and older simulations is deserving of further analysis. When the simulation was aged from young to older, the knee flexion angle at heel-strike increased, as did the vertical GRF impact peak (Fig. 5.1). The metabolic cost remained nearly the same. These findings are consistent with experiments that compared these variables between young and older runners (Allen et al., 1985; Bus, 2003; Cavagna et al., 2008a) but are in conflict with studies that manipulated the knee angle at heel-strike. A more flexed knee greatly increased the metabolic cost of running in McMahon et al. (1987) and reduced the impact peak in Derrick (2004). We note that the conditional differences in knee flexion in those studies were greater than the age-induced change in this study (e.g. 5.6° at heel-strike). In addition, the relatively narrow range of knee motion during stance in the older simulation (Fig. 5.3) indicates a “stiffer” knee according to the popular mass-spring model of running (Cavagna et al., 1977), which results in greater vertical GRF. It is also possible that older runners respond differently than younger runners to experimentally-induced knee angle adjustments, but this theory has not been tested.

Epidemiological studies have reported that older runners sustain more injuries to the hamstrings, calf muscles, and Achilles tendon, while young runners incur more injuries to the knee joint. These locations corresponded to locations where aging changed the magnitude of internal loading in the simulation. Virtual aging resulted in increased stress and strain of the hamstrings and plantarflexors and a reduction in the magnitude of the knee joint contact force. These changes may explain why older adults are more prone to hamstring and calf injuries, but less prone to knee injuries. When the older simulation was repeated with the
maximum isometric strengths of these muscles restored to their young values, their loading variables were reduced (Fig. 5.5). The knee joint contact load increased but was maintained at a force well below the young simulation. These results suggest that older runners could benefit from a strength-training program targeting the hamstrings and calves, without compromising the safety of the knee joint. While general aging is inevitable and unavoidable, strength training acts as a “fountain of youth” for the muscular system and can reverse (Reeves et al., 2006) or at least slow (Faulker et al., 2007) many of changes in muscular properties that result from aging and/or disuse, even in very old individuals (Caserotti et al., 2008). The older simulation altered its muscle coordination at the knee such that the knee joint contact force was reduced, but the change in knee joint load was not reflected in the vertical GRF impact peak, which increased with aging. This finding supports the theory of Bus (2003) that the running gait of older adult may serve to protect the knee joints, as well as the conclusion of Miller and Hamill (2009) that changes in internal loading should not be inferred from external loading.

In conclusion, aging the muscular system of a computer simulation of human running induced changes in mechanics and energetics consistent with those documented between young and older runners. The results provide a framework for testing hypotheses on longitudinal relationships between muscular properties and human movement performance and suggest direct links between muscular properties, aging, and running biomechanics. Differences in muscle and knee joint loading were consistent with injury patterns in young and older runners. Strengthening the hamstrings and plantarflexors of the older simulation reduced the loading variables of these muscles and suggests older runners may benefit by increasing the strength of these muscles.
Figure 5.1. Joint angles and GRF components vs. the stride cycle from the means of the human subjects (dashed lines) and the young performance-based simulation (solid lines). Shaded area is \( \pm \) two between-subjects standard deviation around the mean of the human subjects. The stride begins and ends at heel-strike.
Figure 5.2. Optimized muscle excitations vs. the stride cycle for the young. ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius. The stride begins and ends at heel-strike. Vertical dashed lines indicate toe-off. Horizontal bars indicate the “on” time of the EMG data. No EMG data were available for ILP and BF.
Figure 5.3. Joint angles and GRF components vs. the stride cycle from young (solid) and older (dashed) performance-based simulations. The stride begins and ends at heel-strike. Vertical lines indicate toe-off for the young (solid) and older (dashed) simulations.
Figure 5.4. (a) Peak contractile component stresses (peak muscle force divided by PCSA) and (b) peak series elastic component strains (relative to unloaded length) for each muscle model in the young (empty bars) and older (filled bars) simulations. ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius.
Figure 5.5. (a) Peak contractile component stresses (peak muscle force divided by PCSA) and (b) peak series elastic component strains (relative to unloaded length) for each muscle model in the initial older simulations (dark bars) and with muscle strength restored for hamstrings, soleus, and gastrocnemius (light bars). ILP = iliopsoas; GLU = glutei; VAS = vasti; BF = biceps femoris (short head); TA = tibialis anterior; SOL = soleus; RF = rectus femoris; HAM = hamstrings; GAS = gastrocnemius.
CHAPTER 6
CONCLUSION

6.1. Summary

This dissertation is titled “Optimal Control of Human Running”. The meaning of “human running” is likely obvious to most readers, but the meaning of “optimal control” may not be. Optimal control is a mathematical theory used to derive control policies for dynamical systems. The premise is to find a set of control variables (e.g. muscle excitations) that optimizes (minimizes or maximizes) the value of an objective function. This value depends on the system’s state variables (e.g. joint angles and velocities), which are functions of the control variables. Optimal control has been a major component of physics and mathematics for centuries, but has only been used in human movement science for the last 40 years (Chow & Jacobson, 1971; Ghosh & Boykin, 1976). In biomechanics and motor control, the central question addressed by optimal control is:

Between initial state $A$ and final state $B$, how should the musculoskeletal system be controlled in order to move in an optimal way?

An answer to this question requires a definition of what it means to be optimal. In other words, why do humans run the way they do, and what quantity does the nervous system prioritize when activating muscles during running?

The work was motivated by evidence that human running has characteristics of a constrained optimization problem, where the quality of the movement is maximized in
accordance with some unknown optimality criterion (Cavanagh & Williams, 1982; Hamill et al., 1995; Gutmann et al., 2006). The goal of Chapter 3 was to investigate what this criterion might be by generating forward dynamics simulations that minimized several different candidate criteria. Simulations of this type are known as performance-based simulations. The results were compared to data from experienced human runners, who were presumably minimizing the true unknown criterion as they ran. The next two chapters demonstrated the utility and predictive nature of performance-based simulations by addressing two topics that are difficult to study in live human subjects: the sensitivity of maximum sprinting speed to the force-velocity relationship (Chapter 4), and the relationship between aging, muscular properties, and running biomechanics (Chapter 5). The results and conclusions of each study were addressed in detail in their respective chapters, and are summarized here:

- **Chapter 3:** Although all three simulations qualitatively resembled human running, minimizing muscle activations generated the most realistic simulation in comparison to experimental data. Minimum activation avoids fatiguing any muscle regardless of its size or strength, and appears to be an important control policy for human running, perhaps more so than minimizing the rate of metabolic energy consumption.

- **Chapter 4:** Of the three parameters that characterize the muscle force-velocity relationship, maximum sprinting speed was most sensitive to the “shape parameter” $a$, which defines the contour of the concentric force-velocity hyperbola. This result was due to enhanced force production capabilities at moderate shortening velocities, where muscle contractile components tended to operate during sprinting.
• **Chapter 5:** Aging the model’s muscular system shifted its running mechanics towards the characteristic gait of older adults. The simulation approach thus provides a framework for testing hypotheses on muscular properties and human performance. Changes in internal loading between the young and older simulations reflected differences in injury locations between younger and older adults. Strengthening injury-prone muscles in the older simulation reduced the loading of these locations. Older runners may benefit by strengthening specific muscle groups to avoid injuries.

6.2. Revisiting the Hypothesis

Chapter 1 of this document presented seven hypotheses that were tested in the three studies. These hypotheses are revisited here. In the first study (Optimality criteria for performance-based simulations of human running; Chapter 3), Hypothesis 1.1 stated that minimizing the cost of transport would generate the most realistic performance-based simulation of running. This hypothesis was not supported. Minimizing the sum of the squared muscle activation integrals generated a more realistic simulation. Hypothesis 1.2 stated that the model would be dynamically stable in response to small perturbations to its initial conditions. This hypothesis was supported when “stability” was defined from a theoretical mechanics perspective (see Appendix B). However, since the model’s neural control scheme lacked any peripheral feedback, it could not perform multiple strides.

In the second study (Sensitivity of maximum sprinting speed to characteristic parameters of the muscle force-velocity relationship; Chapter 4), Hypothesis 2.1 stated that the maximum shortening velocity \( v_{\text{max}} \), the eccentric plateau force \( C_{\text{ecc}} \), or the concentric
shape parameter would increase the maximum sprinting speed. This hypothesis was supported, although the three variables had different levels of effect. Maximum speed was most sensitive to the value of $a_R$, and the fastest speed was achieved when $a_R$ was set at its maximum possible value. Hypothesis 2.2 (maximum sprinting speed would be most sensitive to $v_{max}$) was therefore not supported. The muscle contractile components shortened at moderate velocities, $a_R$ has its greatest effect, for most of the stride and generally did not approach the extreme concentric and eccentric velocities where $v_{max}$ and $C_{ecc}$ have their greatest effects.

In the third study (Virtual aging of the muscular system and its effects on running biomechanics; Chapter 5), Hypothesis 3.1 stated that adjusting the muscle model parameters to represent the aging process would induce changes in running biomechanics characteristic of older adults. Hypothesis 3.2 stated that these adjustments would result in increased loading of the plantarflexors and hamstrings. Hypothesis 3.3 stated that strengthening the maximum isometric force of the plantarflexors and hamstrings would reduce their loading in the “older” running simulation. These hypotheses were all supported.

6.3. General Conclusions

The introduction distinguished between two types of forward dynamics simulations: the data-tracking approach, and the performance-based approach. Recall that the data-tracking approach optimizes the control variables to minimize the “tracking error” with an experimental data set so that the model output matches the experimental data as close as possible. In contrast, the performance-based approach maximizes the achievement of a movement goal defined by an optimality criterion and does not use experimental data as an
explicit tracking template. Both approaches technically use optimal control theory, although optimal control is more often referred to in the context of performance-based work. The performance-based approach has been used frequently to study walking (e.g. Chow & Jacobson, 1971; Anderson & Pandy, 2001a; Sellers et al., 2003; Ackermann & van den Bogert, 2010; Umberger, 2010) but has only rarely been applied to running. The author is aware of only two studies that generated performance-based simulations of running (Hase & Yokoi, 2002; Sellers et al., 2010) and one that generated simulations of sprinting (Sellers & Manning, 2007). None of these studies compared their state and control variables to data from human runners, and they all assumed the appropriateness of a single optimality criterion without evaluating others. These studies therefore did not directly address whether the optimal control approach is valid for studying human running. Establishing this validity was an important contribution of the present work.

There are two major points of concern relating to the level of trust that can be placed in the present results. First, the output of a model is influenced, sometimes to a large extent, by the values assigned to its parameters (Caldwell, 1995; Scovil & Ronsky, 2006; Redl et al., 2007; Domire & Challis, 2010). This concern is particularly important in optimization studies, where it is possible to “over-fit” the model by allowing too much freedom in parameter assignments in order to track a target data set. Second, model-based predictions of variables that cannot be measured in vivo, such as muscle forces, are difficult to validate since “gold standard” in vivo data are not readily obtainable. However, for the data that were available, most of the whole-body parameters (speed, stride length and frequency, metabolic energy expenditure) matched the means of the human subjects to within a few percentage points, and most of the simulated joint angles and GRF fell within two standard deviations of
the human runners’ mean data. Comparisons between the magnitudes of the optimized muscle excitations and the experimentally collected electromyograms are difficult due to a variety of issues (Kamen & Caldwell, 1996), but the timing of these data were similar in most cases. While there are admittedly many different sets of excitations and muscle forces that could accurately simulate one or two of these variables, there are likely very few that could accurately simulate all of them. Based on these comparisons, we conclude that the performance-based approach to forward dynamics can generate realistic simulations of human running, depending on the choice of optimality criterion.

The real strength of the performance-based approach lies not in its ability to accurately reproduce experimental data without explicitly tracking it, but in what can be done once this ability is established. Skeletal muscle is an extraordinary material whose mechanics and energetics are at best impractical, and at worst impossible, to observe during full-body human movement with current imaging technologies. This case is unfortunate, as much of our ability to move depends on the abilities of our muscles to generate forces and perform work on the skeleton. The latter two studies of this dissertation (Chapters 4 and 5) provide examples of how simulations can be used to investigate relationships between the properties of the muscular system, the resulting mechanics and energetics of muscles during movement, and the movement of the skeleton that results from those muscular actions. This framework has great potential for addressing questions that cannot be answered purely with experiments on human runners. Forward dynamics modeling and simulation in general, and performance-based simulations specifically, have historically not been widely used in running biomechanics research. Experimental work and the inverse dynamics model (Elftman, 1939, 1940) dominate the field. Modeling studies are often fairly criticized for not
including an experimental component, but the author is unaware of any experimental study that has been criticized for not including a modeling component. Every study on running biomechanics obviously does not need to include a forward dynamics model, but researchers should not shy away from questions that can’t be answered with in vivo experimentation. Simulations provide a useful alternative in these cases and will almost always compliment a well-designed experiment.

6.4. Future Work

Suggestions for future work fall into two categories: work with the model in its present state, and work with a model adjusted to remove some of the limitations of the present model. In its present state, potential questions that could be addressed include: (1) What is the shape of the energy-speed relationship in human running? (2) How do adjustments in the mechanical properties of specific muscles influence sprinting ability? (3) How do specific age-related changes in muscle properties affect running performance when they are adjusted in isolation? Each of these questions was considered for inclusion in this dissertation, but was ultimately not included due to time constraints.

Regarding modifications to the model, a specific issue is the generation of simulated GRF. The accuracy of this variable is notoriously poor in forward dynamics simulations of locomotion, particularly during the initial impact phase. The acceptance of forward dynamics simulations as a valid and useful tool would be enhanced by improvements in the accuracy of simulated GRF. In the present work, the tracking simulations had very realistic GRF, while the performance-based simulations were less realistic. This result suggests that realistic GRF are within the capabilities of even this relatively simple 2D model. The
difficulty issue is to get the model to generate these realistic forces without explicitly telling it to do so, either by adjustments of the objective function or additions to the model. The rigid body assumption likely contributes to errors in the dynamics of the impact phase, although we did not find the addition of the Gruber et al. (1998) wobbling mass model to improve the GRF in Chapter 3. A variety of ground contact models have been documented in the literature, with varying success in generating GRF during simulations of walking and running (Gerritsen et al., 1995; Neptune et al., 2000; Anderson & Pandy, 1999, 2001a), but no study has compared the performance of various ground contact models within the same musculoskeletal model.

The model was used to simulate running in the present work, but is capable of generating a wide range of movements as long as an appropriate optimality criterion can be defined. Forward dynamics simulations have for some time been championed as a predictive tool for optimizing outcomes in sports training (e.g. Hatze, 1983) and orthopedic surgery (e.g. Delp et al., 1990) on a subject-specific basis. It is the author’s view that no current model is sufficiently detailed to safely make clinical decisions on an individual, and that such a reality, while possible, remains a distant goal. A more realistic short-term goal is to use simulations to contribute to the knowledge base of a coach, athlete, or surgeon by providing them with a new source of information (e.g. what does the model say will happen if we strengthen these muscles, or transfer this tendon to this location?). A more detailed model will likely be needed to make such suggestions with confidence. Potential additions include (1) a three-dimensional musculoskeletal system, (2) a more detailed arm and upper body model, and (3) additional muscles and subject-specific musculoskeletal geometry. Depending on the application, additional modifications will be needed on a case-by-case
basis. For example, a simulation optimizing the design of a total knee replacement for performing a particular activity would require a more detailed model of the knee anatomy.
APPENDIX A

MUSCLE MODEL ALGORITHM AND PARAMETERS

A.1. Introduction

The muscle model was a two-component Hill-based model (Hill, 1938) with a contractile component (CC) in series with an elastic component (SEC). The model was based on the work of Caldwell (1995) and Umberger et al. (2003). A two- rather than three-component model was used because it allows parallel elasticity to be implemented at the level of the joints, where passive torque-angle relationships have been widely studied (e.g. Riener & Edrich, 1999). This appendix describes the muscle model algorithm and also presents the method by which the muscle-specific parameters were determined.

A.2. Algorithm

Each muscle had two state variables: the CC length $L_{CC}$ and the activation $A$. The goals at each time step were to calculate the time derivatives of these variables so that they could be updated by the integrator for the next time step, and to calculate the current muscle force. The total muscle length was $L_m = L_{CC} + L_{SEC}$, where $L_{SEC}$ is the SEC length. $L_m$ was a known function of the skeleton’s kinematic state. $L_{SEC}$ was therefore calculated as $L_{SEC} = L_m - L_{CC}$. The force in the SEC, $F_{SEC}$, was then calculated from the SEC force-extension relationship:

$$F_{SEC} = F_o C_{SEC} \left( \exp \left( K_{SEC} \left( \frac{L_{SEC}}{L_u} - 1 \right) \right) - 1 \right)$$

(Eq. A.1)
where $F_o$ is the maximum isometric force, $C_{SEC}$ is the SEC scaling coefficient, $K_{SEC}$ is the SEC stiffness, and $L_u$ is the unloaded SEC length. $C_{SEC} = 0.0258$ and $K_{SEC} = 92.08$ were defined for every muscle such that $F_{SEC} = F_o$ when $L_{SEC} = 1.04L_u$ (Ettema & Huijing, 1989; Caldwell, 1995). $F_o$ and $L_u$ were defined on a muscle-specific basis (Section A.3).

Since the CC and SEC were in series, the CC force $F_{CC}$ was equal to $F_{SEC}$. Knowing $F_{CC}$ and $L_{CC}$, the CC velocity $\dot{L}_{CC}$ was calculated. First, the position of the CC on its force-length relationship, $FL$, was calculated (Woittiez et al., 1983):

$$FL = -\frac{1}{W^2}\left(\frac{L_{CC}}{L_o} - 1\right)^2 + 1 \quad \text{(Eq. A.2)}$$

where $W$ is the width of the force-length parabola and $L_o$ is the optimal CC length (i.e. the apex of the force-length parabola). Both parameters were defined on a muscle-specific basis.

The CC force-velocity relationship was used to calculate $\dot{L}_{CC}$. There were three possible conditions at each time step: isometric, concentric, or eccentric. The isometric condition ($\dot{L}_{CC} = 0$) occurred when $F_{CC}$ was equal to the maximum isometric force, after considering the current length and activation ($F_{CC} = F_o \cdot FL \cdot A$). The concentric condition ($F_{CC} < F_o \cdot FL \cdot A$), has a hyperbolic force-velocity relationship (van Soest & Bobbert, 1993):

$$\dot{L}_{CC} = -L_o \left(\frac{b_R (FL + a_R)}{F_{CC} F_o^{-1} A^{-1} + a_R} - b_R\right) \quad \text{(Eq. A.3)}$$

where $a_R$ and $b_R$ are Hill’s dynamic constants that describe the shape of the hyperbola (Hill, 1938). $a_R$ is dimensionless while $b_R$ has units of s^{-1}. Both were defined as functions of the muscle fiber type ratio. The ratio $b_R / a_R$ is the maximum shortening velocity $v_{max}$.

For the remaining eccentric condition, the CC force approached an asymptotic value as the speed of lengthening increased (van Soest & Bobbert, 1993):
\[ \dot{L}_{CC} = -L_0 \left( \frac{C_1}{F_{CC} F_o^{-1} A^{-1} + C_2} - C_3 \right) \]  
(Eq. A.4)

\[ C_2 = -F L \cdot C_{ecc} \]  
(Eq. A.5)

\[ C_1 = \frac{b_R (FL + C_2)^2}{C_{slope} (FL + a_R)} \]  
(Eq. A.6)

\[ C_3 = \frac{C_1}{FL + C_2} \]  
(Eq. A.7)

where \( C_{ecc} \) is the asymptotic force in multiples of \( F_o \) and \( C_{slope} \) defines the slope of the force-velocity relationship at \( \dot{L}_{CC} = 0 \). \( C_{ecc} = 1.45 \) for all muscles, which was the mean value from the review by Zajac (1989). \( C_{slope} = 2.0 \) for all muscles, which made the intersecting slopes of the concentric and eccentric portions of the force-velocity relationship approximately equal. To avoid division by zero in the force-velocity equations, \( F_{CC} \) had a maximum of \( F_o \cdot F L \cdot A \cdot C_{ecc} - 0.0001 \) N.

The time derivative of activation, \( \dot{A} \), was calculated from the current activation \( A \) and the current excitation \( E \) using a first-order ordinary differential equation first presented by He et al. (1991):

\[ \dot{A} = (\beta E + \beta_2)(E - A) \]  
(Eq. A.8)

\[ \beta_2 = \tau_{deact}^{-1} \]  
(Eq. A.9)

\[ \beta_1 = \tau_{act}^{-1} - \beta_2 \]  
(Eq. A.10)

where \( \tau_{act} \) and \( \tau_{deact} \) were time constants for activation and de-activation, respectively. Both parameters were functions of the muscle fiber type ratio (Section A.3). \( A \) had a minimum value of 0.001 (0.1% of maximum activation) to avoid division by zero in the force-velocity equations.
A.3. Parameters

Each muscle model required values for eight parameters: the maximum isometric force $F_o$, the optimal CC length $L_o$, the unloaded SEC length $L_u$, the force-length parabola width $W$, the dynamic constants $a_R$ and $b_R$, and the activation / de-activation constants $\tau_{act}$ and $\tau_{deact}$. This set was reduced to six parameters by defining a new parameter, $FT$, which was the muscle fiber type ratio (specifically, the proportion of fast-twitch muscle fibers):

\[
a_R = 0.1 + 0.4FT \quad \text{(Eq. A.11)}
\]
\[
\tau_{act} = 0.04 - 0.025FT \quad \text{(Eq. A.12)}
\]
\[
\tau_{deact} = 0.0475 - 0.03FT \quad \text{(Eq. A.13)}
\]

With $a_R$ known, $b_R = V_{\text{max}} \cdot a_R$ was then calculated assuming $V_{\text{max}} = 12 \, L_o \, s^{-1}$ for all muscles, which is consistent with the estimate by de Ruiter et al. (2000) for human muscle.

The five remaining parameters ($F_o$, $L_o$, $L_u$, $W$, $FT$) were determined using an optimization procedure. Subjects performed maximum isovelocity contractions of hip flexion, hip extension, knee flexion, knee extension, ankle plantarflexion, and ankle dorsiflexion on a dynamometer (System 3, Biodex, Shirley, NY, USA). Each contraction was repeated at three concentric velocities (-20, -90, and -150 deg s$^{-1}$) and one eccentric velocity (+150 deg s$^{-1}$). The resulting torque-time profiles were averaged over subjects for each contraction. These contractions were then simulated using the computer model. All agonist muscles were assumed to be 100% active while all antagonists were 10% active. All other muscles were inactive. The five parameters were optimized simultaneously for each muscle to minimize the sum of the mean square errors between the simulated and experimental joint torques. Parameters were bounded to be within 50% of those presented by Umberger et al. (2006).
sectional area (PCSA) was optimized, then multiplied by an assumed specific tension of 40 N cm$^{-2}$, which is within the range of estimates for human muscle in vivo (Fukunaga et al., 1996; Erskine et al., 2009; O’Brien et al., 2010). Muscle mass, required for the energetics model, was calculated as the product of PCSA, $L_o$, and the muscle density, which was assumed to be 1.06 g cm$^{-3}$ (Mendez & Keys, 1960). The average RMS error between the simulated and measured joint torques was 19 Nm or 8% of the maximum joint torque. The muscle model parameters are listed in Tables A.1 and A.2.
Table A.1. Optimized muscle model parameters. PCSA = physiological cross-sectional area; \( L_o \) = optimal contractile component length; \( L_u \) = unloaded series elastic component length; \( W \) = width of force-length parabola; \( FT \) = proportion of fast-twitch muscle fibers.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>PCSA (cm(^2))</th>
<th>( L_o ) (cm)</th>
<th>( L_u ) (cm)</th>
<th>( W ) (% ( L_o ))</th>
<th>( FT ) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iliopsoas</td>
<td>58.1</td>
<td>12.0</td>
<td>8.0</td>
<td>65</td>
<td>52</td>
</tr>
<tr>
<td>Glutei</td>
<td>101.8</td>
<td>14.0</td>
<td>12.5</td>
<td>60</td>
<td>47</td>
</tr>
<tr>
<td>Vasti</td>
<td>141.6</td>
<td>9.5</td>
<td>13.0</td>
<td>85</td>
<td>59</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>25.2</td>
<td>14.5</td>
<td>9.0</td>
<td>55</td>
<td>31</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>67.0</td>
<td>8.8</td>
<td>20.5</td>
<td>51</td>
<td>25</td>
</tr>
<tr>
<td>Soleus</td>
<td>147.2</td>
<td>4.9</td>
<td>23.3</td>
<td>88</td>
<td>19</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>36.6</td>
<td>11.0</td>
<td>29.0</td>
<td>110</td>
<td>67</td>
</tr>
<tr>
<td>Hamstrings</td>
<td>75.0</td>
<td>14.0</td>
<td>31.0</td>
<td>92</td>
<td>41</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>70.4</td>
<td>7.1</td>
<td>39.2</td>
<td>88</td>
<td>50</td>
</tr>
</tbody>
</table>

Table A.2. Calculated muscle model parameters. \( F_o \) = maximum isometric force; \( a_R \) = force-velocity shape parameter; \( b_R \) = force-velocity intercept parameter; \( \tau_{act} \) = activation time constant; \( \tau_{deact} \) = deactivation time constant.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>( F_o ) (N)</th>
<th>( a_R )</th>
<th>( b_R ) (s(^{-1}))</th>
<th>( \tau_{act} ) (ms)</th>
<th>( \tau_{deact} ) (ms)</th>
<th>( m ) (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iliopsoas</td>
<td>2324</td>
<td>0.31</td>
<td>3.70</td>
<td>27.0</td>
<td>31.9</td>
<td>0.74</td>
</tr>
<tr>
<td>Glutei</td>
<td>4072</td>
<td>0.29</td>
<td>3.46</td>
<td>28.3</td>
<td>33.4</td>
<td>1.51</td>
</tr>
<tr>
<td>Vasti</td>
<td>5664</td>
<td>0.34</td>
<td>4.03</td>
<td>25.3</td>
<td>29.8</td>
<td>1.42</td>
</tr>
<tr>
<td>Biceps femoris</td>
<td>1008</td>
<td>0.22</td>
<td>2.69</td>
<td>32.3</td>
<td>38.2</td>
<td>0.39</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>2680</td>
<td>0.20</td>
<td>2.40</td>
<td>33.8</td>
<td>40.0</td>
<td>0.63</td>
</tr>
<tr>
<td>Soleus</td>
<td>5888</td>
<td>0.18</td>
<td>2.11</td>
<td>35.3</td>
<td>41.8</td>
<td>0.76</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>1464</td>
<td>0.37</td>
<td>4.42</td>
<td>23.3</td>
<td>27.4</td>
<td>0.43</td>
</tr>
<tr>
<td>Hamstrings</td>
<td>3000</td>
<td>0.26</td>
<td>3.17</td>
<td>29.8</td>
<td>35.2</td>
<td>1.11</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>2816</td>
<td>0.30</td>
<td>3.60</td>
<td>27.5</td>
<td>32.5</td>
<td>0.53</td>
</tr>
</tbody>
</table>
APPENDIX B

STABILITY ANALYSIS

B.1. Introduction

This chapter describes the musculoskeletal model from Chapter 3 in terms of its state space and assesses its stability from a nonlinear dynamics perspective. The term “stability” refers to the response of the simulation within its state space to random perturbations. Dynamic stability is a signature of normal human locomotion (e.g. Hurmuzlu & Basdogan, 1994; Jindrich & Qiao, 2009), and our purpose was to determine if the model also possessed this feature.

B.2. State Space

The human neuro-musculo-skeletal system has thousands of degrees of freedom among its many joints, muscles, and motor units. The present model greatly simplified the musculoskeletal system for tractability and ease of interpretation. Gross neural input to the muscles was included, but the dynamics of the nervous system were not coupled to those of the muscular and skeletal systems and the control scheme was entirely feedforward.

The musculoskeletal model was a system of 58 coupled ordinary differential equations (state equations) to which a set of control variables were input. The equations were solved numerically for the 58 state variables. The state equations in vector form were:

\[ \dot{\theta} = f_1(\theta, \dot{\theta}, L_{cc}, A) \]  
(\text{Eq. B.1})

\[ \dot{L}_{cc} = f_2(L, A) \]  
(\text{Eq. B.2})
\[ \dot{A} = f_3(A, E) \]  

(Eq. B.3)

where Eq. B.1 is the skeletal equations of motion, Eq. B.2 is the muscle contractile dynamics, and Eq. B.3 is the muscle activation dynamics. \( \theta \) and \( \dot{\theta} \) are vectors of 11 generalized coordinates and speeds, \( L_{cc} \) is the vector of 18 muscles model contractile component lengths, and \( A \) and \( E \) are vectors of 18 muscle model activations and excitations. Overhead dots indicate derivatives with respect to time. \( \theta, \dot{\theta}, L_{cc}, \) and \( A \) comprised the 58 state variables while \( E \) was a control variable along with the initial kinematic state and several other variables (see Chapter 3). Simulations were performed by solving Eqs. B.1-B.3 for the state variables, given values for the control variables (see Chapters 3-5). In the terminology of nonlinear dynamics, the “state” is the set of current state variable values, and the “state space” is the set of all possible states.

**B.3. Calculation of Orbital Stability Metric**

Stability was assessed using two tools from nonlinear dynamics: Floquet theory (Floquet, 1883) and Poincaré return maps (Poincaré, 1890). The method was based on the work of Hurmuzlu and Basdogan (1994) and provides an assessment of “orbital dynamic stability”, which quantifies a system’s tendency to return to a stable orbit from one cycle to the next when it encounters a small perturbation. The analysis was performed on the center of mass (CoM) vertical position because the time history of this variable is sensitive to all of the state variables.

Three data sets were used: (1) 163 steps from a human subject running on a treadmill; (2) 163 cycles of a “stable orbit”, i.e. a series of steps where each step was point-for-point identical to every other step, and (3) a set of 50 one-step simulations where some control
variables were perturbed. Steps were assessed rather than strides because the human vertical CoM position is essentially bilaterally symmetric for normal running, and because the simulation does not maintain its periodicity when perturbed. The human data were collected from a female subject with body mass (63.2 kg) and preferred running speed (3.55 m s\(^{-1}\)) that were similar to the model. The subject ran on an instrumented treadmill that sampled ground reaction forces (GRF) at 2400 Hz (Athletic Republic, Fargo, ND, USA). The vertical GRF component was divided by body mass and gravitational acceleration was subtracted to calculate the CoM vertical acceleration. CoM vertical position was then calculated by double-integrating the CoM vertical acceleration with respect to time. The stable orbit data set was constructed by taking the mean CoM vertical position time series from the human subject’s data, de-trending it, then concatenating the de-trended step 162 times.

To generate a data set of perturbed steps, the MinAct solution from Chapter 3 was used as a starting point. The initial kinematic state (angular positions and velocities) was perturbed 49 times within a range of +/- one between-subjects standard deviation using Fortran’s intrinsic “rand” function. All other control variables were held constant. Each time, the state equations were solved with the perturbed initial kinematic state, and the CoM vertical position was output in 1-ms intervals.

The next step was to construct first return maps for each data set from the CoM vertical positions of all steps:

\[ y_{i+1,n} = f_{map}(y_{i,n}) \]  

(Eq. B.4)

where \( y_{i,n} \) is CoM vertical position at time \( n \) within the \( i^{th} \) step. Figure B.1 shows the return map from the human subject’s data at heel-strike \( (n = 1) \). Next, the Jacobian matrix \( J \) of Eq.
B.4 was calculated by “linearizing” the return map about a theoretical equilibrium point \( y_{eq} \), which represents a possible stable orbit:

\[
y_{eq} = f_{map}(y_{eq})
\]  
(Eq. B.5)

What this step means in practice is that \( y_{eq} \) is subtracted from the input and output of Eq. B.4, and the function \( f_{map} \) is assumed to be a linear mapping between \( y_{i,n} \) and \( y_{i+1,n} \):

\[
y_{i+1,n} - y_{eq} = J_n \cdot (y_{i,n} - y_{eq}) \Rightarrow J_n = \frac{y_{i+1,n} - y_{eq}}{y_{i,n} - y_{eq}}
\]  
(Eq. B.6)

\( y_{eq} \) was taken to be the mean CoM vertical position at the appropriate time. The eigenvalues of \( J_n \) define the stability of the system. Specifically, each eigenvalue is referred to as a “Floquet multiplier”. In order for the system to be orbitally stable in response to the perturbations it encounters, the magnitudes of all Floquet multipliers must be below 1.0 (Poincaré, 1890). The index of orbital stability is therefore the Floquet multiplier with the largest magnitude, i.e. the maximum Floquet multiplier \( \lambda_{\text{max}} \).

B.4. Results

The CoM vertical positions from the human subject and the simulations are presented for comparison in Fig. B.2. Recall that the mean curve from the human subject (Fig. B.2) was the basis for the stable orbit data set. The mean perturbed CoM profile from all strides of the simulations was similar to the MinAct solution’s unperturbed CoM profile (RMSE = 1.0 cm), indicating that the perturbations did not induce a systematic shift in the CoM height. All three data sets were orbitally stable across the entire step as indicated by the maximum Floquet multipliers in Fig. B.3. For the stable orbit, \( \lambda_{\text{max}} \) was zero across the entire step. For the human subject, \( \lambda_{\text{max}} \) averaged 0.86 and the largest \( \lambda_{\text{max}} \) (0.90) occurred at 100% of the
step, in mid-swing. For the simulations, $\lambda_{\text{max}}$ averaged 0.19 and the largest $\lambda_{\text{max}}$ (0.27) occurred at 22% of the step, in mid-stance. Orbital stability of running has not been widely studied, but the results are consistent with the presence of orbital stability in human walking (Hurmu"{z}lu & Basdogan, 1994; Dingwell & Kang, 2007).

B.5. Discussion

In summary of the stability analysis, both the human runner and the perturbed simulation where stable according to Floquet theory, with the maximum Floquet multiplier $\lambda_{\text{max}} < 1.0$ for the full step duration. The simulation had smaller $\lambda_{\text{max}}$ across the step duration compared to the human runner, and the theoretical stable orbit had a constant value of $\lambda_{\text{max}} = 0$. To interpret these results, recall that the maximum Floquet multiplier is a “yes/no” criterion of the system’s ability to return to a stable orbit in response to small perturbations. $\lambda_{\text{max}} < 1.0$ indicates a stable system, but a smaller $\lambda_{\text{max}}$ does not necessarily indicate a more stable system. Different conclusions can be reached depending on the definitions of stability and instability, which are well defined mathematically but can be difficult to reconcile with human movement.

For example, $\lambda_{\text{max}}$ was zero for the stable orbit in Fig. B.2 because the system experienced no perturbations, not because it is extremely robust to perturbations. However, $\lambda_{\text{max}} = 1.0$ indicate the presence of a center manifold (Moehlis et al., 2006), which means that the state space behavior likely contains bifurcations. The theory and mathematics of these topics are complex, but they essentially mean that when the system with $\lambda_{\text{max}} = 1.0$ is perturbed, it may return to a different orbit in the state space depending on the type and intensity of the perturbation. Human locomotion is extremely flexible and versatile, and
certainly contains many of these features. For example, when walking speed is increased above a certain threshold, humans transition to a run rather than losing their locomotor ability. Less severe perturbations can be handled by adjustments for example in speed and cadence. These features may explain why $\lambda_{\text{max}}$ were closer to 1.0 for the human runner, but closer to zero for the simulation model, which lacks many of these features due to its greatly reduced degrees of freedom and lack of peripheral feedback in its control scheme.

It should also be remembered that the orbital dynamic stability indicated by $\lambda_{\text{max}}$ is only one type of stability analysis. Other measures of stability such as the variance of relative phase (Haken et al., 1985; Lamoth et al., 2002) and the Lyapunov exponent (Dingwell et al., 2001; Bruijn et al., 2009) are often used in nonlinear dynamics. Conclusions on stability from relative phase variance are only appropriate under a specific set of conditions that are generally not found in human running. Lyapunov exponents are calculated from large, continuous data sets of many cycles, which cannot be generated using the present model due to its lack of coupled neural dynamics and peripheral feedback. An enhanced version of the model that included these features was able to perform several consecutive strides, but required a tremendous amount of CPU time and was deemed outside the scope of the present work.
Figure B.1. First return map of vertical center of mass position ($y$) at heel-strike for the human subject (closed circles) and the simulations (open circles).

Figure B.2. Vertical center of mass positions vs. the % of the step duration for the human subject (left) and the simulation (right). Solid lines are mean profiles. The dashed line is the unperturbed profile from the MinAct simulation.
Figure B.3. Maximum Floquet multipliers ($\lambda_{\text{max}}$) vs. the step duration for (a) the human subject (solid line), (b) the stable orbit (dashed), and (c) the simulation (dash-dotted).
APPENDIX C

THE METABOLIC COST OF THE SWING PHASE

C.1. Introduction

There are conflicting views on whether the metabolic cost of the swing phase in running is small (Taylor et al., 1974, 1980), moderate (Marsh et al., 2004; Modica & Kram, 2005), or large (Hill, 1950; van Ingen Schenau et al., 1997) compared to the stance phase. Most of the evidence has some from comparative studies of quadrupeds (e.g. Taylor et al., 1974), theoretical arguments (e.g. van Ingen Schenau et al., 1997), or creative experimental designs (e.g. Modica & Kram, 2005), which all provided indirect evidence on the relative costs of stance and swing in human running. More direct evidence for bipedal running has come from measurements of blood flow to individual muscles in guinea fowl (Marsh et al., 2004), but these measurements are highly invasive and cannot be performed in humans.

Therefore, the purpose of the study was to compare the metabolic costs of the stance and swing phases in human running using a computer simulation approach. We hypothesized that stance would account for the larger proportion of the total metabolic energy consumed by muscles during the stride cycle.

C.2. Methods

The MinCoT, MinAct, and MinStress simulations from Chapter 3 were selected for further analysis. The gross metabolic power of each muscle was calculated at 1-ms intervals using the model of muscle energy expenditure (Umberger et al., 2003). The metabolic
powers were integrated over the stride duration and summed over all muscles to calculate the total metabolic energy consumed by muscles. Stance and swing phase portions were identified for each muscle using the vertical ground reaction force (VGRF) as an index for foot-ground contact (VGRF > 0 = stance; VGRF ≤ 0 = swing).

C.3. Results

The swing phase accounted for 49% of the total metabolic energy consumed by muscles in the MinCoT simulation, and 48% in the MinAct and MinStress simulations. The stance and swing phase costs are broken down into individual muscles in Tables C.1-C.3. Iliopsoas, biceps femoris, tibialis anterior, and rectus femoris consumed most of their energy (70-95%) during swing. Soleus and gastrocnemius consumed most of their energy (68-86%) during stance. Glutei also consumed most of its energy during stance (56-75%). The other muscles were less dramatically aligned with one phase or the other. Vasti consumed 39% of its energy during swing for the MinCoT simulation, 51% for the MinAct simulation, and 57% for the MinStress simulation. Hamstrings consumed 53% energy during swing for the MinCoT simulation, 49% for the MinStress simulation, and 40% for the MinAct simulation.

Although no muscles crossed the metatarsophalangeal (MTP) joint in the present model, the model still had some degree of active control over this joint since its stiffness was a control variable. Despite its small size, the MTP joint accounted for up to 6% of the total positive mechanical joint work in the simulations. If we assume that the MTP joint’s positive joint mechanical work was generated by active muscular forces with an efficiency of 30±10% (the mean±SD of the other muscles), the range of proportional swing phase costs becomes 38-43%. 

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C.4. Discussion

In support of the hypothesis, we found that the swing phase accounted for under half of the metabolic energy consumed by muscles during running. The estimated range was 38-49%, depending on assumptions about the origin of the mechanical work performed by the MTP joint and the duration of the stance phase. The results agree qualitatively with conclusions that the swing phase accounts for a moderate portion of the metabolic cost of running (Marsh et al., 2004; Modica & Kram, 2005), although our model-based estimate is greater than the estimates of those studies. Marsh et al. (2004) estimated 26% in running guinea fowl from measurements of muscle blood flow, and Modica and Kram (2005) estimated 20% in running humans whose swing phase was assisted by an external towing force. The present results do not agree with arguments that the swing phase comprises the vast majority of the metabolic cost of running (Hill, 1950; van Ingen Schenau et al., 1997).

The differences between the present estimate and the more conservative estimates of Marsh et al. (2004) and Modica and Kram (2005) could be due to differences in the lower limb musculature of humans and guinea fowl, the function of the swing assist device used by Modica and Kram (2005), and the design of the present model. The musculature of the guinea fowl hindlimb has one set of muscles that are active only during stance, and another unique set of muscles that are active only during swing. One moderately sized muscle is active during both stance and swing. The relative cost of the swing phase (26%) was insensitive to the gait mode (walking or running) and the speed of running. Based on these features, it is possible that guinea fowl have evolved to have an especially economical swing phase. Humans could certainly have a similar adaptation. However, the large muscles of the human lower limb are active during both stance and swing (Cappellini et al., 2006;
Gazendam & Hof, 2007). Relationships between gait mode, speed, and swing phase cost have not been investigated in humans. Such an adaptation would be particularly advantageous for predator avoidance in an animal like the guinea fowl, which are poor flyers and cannot use their forelimbs for defense and weaponry like humans can.

Modica and Kram (2005) build an external swing assist (ESA) system that used elastic cords to pull the foot forward at toe-off and during swing. They calculated the swing phase cost as the reduction in the net cost of transport with ESA forces from 0-4% bodyweight. It is not clear what portion of the swing phase cost was offset by the ESA system. They found a strong linear relationship ($R^2 = 0.98$) between the reduction in the cost of transport and the ESA force, but did not test forces above 4% bodyweight. In addition, the ESA system did not replace the muscular effort needed to maintain the positions of the knee and ankle joints or to arrest the forward motion of the lower limb in late swing. Based on these limitations, the authors may have underestimated the relative cost of the swing phase. It was also not clear how the ESA system affected the stance phase cost.

The simulation’s control scheme placed no restrictions on the permitted values of the muscle excitation parameters, which can potentially overestimate the swing phase cost if muscles that are active primarily during stance never “turn off” completely during swing. Umberger (2010) used a control scheme with block-shaped excitations that forced the muscle excitations to be zero for part of the stride cycle, and estimated a relative swing phase cost during normal walking of 29% using a musculoskeletal model similar to the present one. The “unrestricted” scheme of the present simulations was used because it generated a better data tracking simulation (see Chapter 3) than a block scheme with an enforced zero-excitation period for each muscle. It is not clear which scheme is more appropriate for
generating performance-based simulations of human running, although electromyographic signals from the human lower limb are generally non-zero throughout the stride cycle (Cappellini et al., 2006; Gazendam & Hof, 2007). The simplicity of the model’s muscular system may have also limited its ability to perform the swing phase with a low cost. For example, the hamstrings are comprised of three muscles but were modeled as a single muscle.

The stance and swing phase costs were likely affected by the duration of the stance phase in the simulations, which was shorter (31% on average) than the human subjects (38% on average). A short stance phase may bias the stride cycle towards energy consumption in the swing phase. This effect could be tested by generating an additional simulation with a penalty term that enforced a more realistic stance phase duration. Initial efforts in this direction were unsuccessful in generating a realistic running gait, although the added penalty term (check for a non-zero GRF prior to 38% of the stride duration) was likely too simple.

In summary, the swing phase accounted for at least 38% (and up to 49%) of the metabolic energy consumed by muscles in the running simulations. Due to the limitations of the model, this result should not be interpreted as the exact cost of swinging the lower limb in human running, as there are features of the model’s design and control scheme that could cause an overestimation of the swing phase cost. However, experimental studies that identified smaller values may have underestimated the swing phase cost with their limitations. We suggest the interpretation that the swing phase accounts for a moderate portion of the metabolic cost of human running, above 20% but under 50%. Theoretical arguments that swing accounts for the great majority of running’s metabolic cost are not supported by the present simulation study or by previous experimental studies.
APPENDIX D
USER’S GUIDE

D.1. Introduction

This appendix presents a guide to using the model described in Ch. 3 to generate simulations of movement. The Fortran code and the SPAN optimization algorithm (Higginson et al., 2005) used in the present work are available on the author’s SimTK project repository (http://www.simtk.org/svn/runmod2D). The code as provided is set up generically for generating simulations of human movement in two dimensions (sagittal plane). The author primarily used the model to simulate running and sprinting, but has also generated simulations of walking, jumping, landing, and quiet stance.

This guide assumes that the user is familiar with the Fortran programming language and basic concepts such as variable types, memory allocation, common blocks, and creating and calling subroutines. For model development, the author recommends beginning with a user-friendly programming environment (e.g. MATLAB), then translating the working algorithms to a more efficient programming language (e.g. Fortran, C++).

D.2. Description of the Code

The code is named “Runner2D_SimTK.f”. It consists of 3,579 lines organized into 10 subroutines: Runner2D, EQNS1, IO, KUTTA, SOLVE, PasMod, GroundMod, MusLengths, MusMomArms, and MusMod. Runner2D, EQNS1, IO, KUTTA, and SOLVE were initially generated using Autolev 4.1, a commercial software program for
symbolic dynamics, then modified by the author. The remaining subroutines (\texttt{PasMod}, \texttt{GroundMod}, \texttt{MusLengths}, \texttt{MusMomArms}, and \texttt{MusMod}) were created by the author. The creators and supporters of Autolev (OnLine Dynamics, Sunnyvale, CA, USA) are out of business as of December 31, 2010. A similar product named Motion Genesis is presently available (http://www.motiongenesis.com).

D.3. Description of Subroutines

Each subroutine begins with a block of variable declarations that pre-allocate their memory locations as required in FORTRAN. There are 10 \texttt{COMMON} blocks that appear in most subroutines:

1. \texttt{COMMON/CONSTNTS} contains variables with constant values that are specified by the user in \texttt{Runner2D}.
2. \texttt{COMMON/SPECIFIED} contains time-dependent variables whose values are calculated in other subroutines.
3. \texttt{COMMON/VARIABLES} contains the model’s state variables.
4. \texttt{COMMON/ALGBRAIC} contains variables that are algebraic functions of the state variables.
5. \texttt{COMMON/MISCELLNS} contains miscellaneous variables that are used in several subroutines.
6. \texttt{COMMON/CONTROLS} contains the model’s control variables.
7. \texttt{COMMON/MUSKINEM} contains muscle kinematics variables (lengths, velocities, and moment arms).
8. \texttt{COMMON/MUSENERG} contains muscle metabolic energy rates.
9. **COMMON/MUSFORCE** contains the muscle forces.

10. **COMMON/TRACKDAT** contains variables that were needed for the objective functions of the simulations.

The remaining variable definition blocks are specific to the subroutine that they are located in. Users are free to add variables to the any block (or create new blocks), but should avoid removing variables from the blocks unless desired modifications to the model necessitate this. New variables in any subroutine should not be given the same name as any variable that appears in a COMMON block.

### D.3.1. Runner2D Subroutine

Runner2D begins by opening the output files that are written to disk when it is called (line 55). Next, some parameters of the model are specified (line 78): total mass ($BM$), gravitational acceleration ($G$), segment lengths ($L^*$), segment masses ($M^*$), segment mass center locations ($P^*$), and segment mass moments of inertia ($I^*$). * indicates that the variable name is followed by letters indicating the segment (hat = head/arms/trunk, hereafter “trunk”; t = thigh; c = calf/leg/shank; f = foot; e = toes) and the side of the body (r = right; l = left). If the user enters the total mass and segment lengths, the code is set up to calculate the remaining parameter values based on the “female” equations of de Leva (1996). These equations will not necessarily be appropriate for a specific individual or for other populations. When specifying the gravitational acceleration, only the magnitude should be entered (do not include the minus sign indicating direction).

Beginning on line 116, initial values for the state variables are specified. With its present set-up, the state variables include the 11 generalized coordinates ($Q_1$-$Q_{11}$), 11 generalized
speeds (Q1p-Q11p), 18 muscle model activations (A1-A18), and 18 muscle model contractile component (CC) lengths (L1-L18). For the generalized coordinates:

- Q1 = horizontal position of the hip joint
- Q2 = vertical position of the hip joint
- Q3 = angular position of the trunk segment
- Q4, Q5, Q6, Q7 = angular positions of the right hip, knee, ankle, and MTP joints
- Q8, Q9, Q10, Q11 = angular positions of the left hip, knee, ankle, and MTP joints

The generalized speeds follow this same sequence. Linear positions should be specified in meters, angular positions in degrees, and speeds in time units of seconds. For the activations and CC lengths, numbers 1-9 indicate the iliopsoas, glutei, vasti, biceps femoris, tibialis anterior, soleus, rectus femoris, hamstrings, and gastrocnemius muscles for the right limb. Numbers 10-18 indicate the left limb muscles in this same order. It is left to the user to determine appropriate initial values for these variables based on the application.

On line 176, the user specifies parameters related to the time of the simulation and the “integrator” algorithm that solves its state equations. TINITIAL and TFINAL are the times at which the simulation starts and stops. The duration of the simulation (within the simulation world, not the real world) is TFINAL – TINITIAL. TINITIAL is usually zero. INTEGSTP is the maximum time step size of the integrator. TINITIAL, TFINAL, and INTEGSTP should be specified in seconds. PRINTINT is the interval (in multiples of INTEGSTP) at which the program writes data to the output files. ABSERR and RELERR specify the tolerances for absolute and relative errors in the integrator. When both tolerances are met, the integrator moves on to the next time step. The Autolev guidelines for specifying the error tolerances are:
1. **ABSERR** should be set to $10^{-8} \cdot x_{\text{small}}$, where $x_{\text{small}}$ is the expected smallest maximum absolute value of the variables being integrated.

2. **RELERR** should be set equal to $10^{-d}$, where $d$ is the desired number of significant digits in the integrator output.

In testing the model, the author found no differences in solutions generated using $10^{-4}$, $10^{-5}$, or $10^{-6}$ for both tolerances, and used $10^{-5}$ for both tolerances. Setting the tolerances too lax (say $10^{-1}$) will lead to inaccurate numerical solutions. Setting them too strict (say $10^{-20}$) will cause the integrator to fail.

Starting at line 278, the user should enter variables that are to be stored at each time step for later use. For example, at the end of the simulation, the user will often want to know the time histories of the joint angles and angular velocities. These are not the variables that are written to the output files (that part comes later in the **IO** subroutine). Rather, these are the variables that are used in calculating an objective function score for an optimization. $ss$ is the integer “counting” variable for this purpose. Its value increases by one every time the code writes data to the output files. The memory space for the storage arrays needs to be defined in the declaration blocks at the beginning of the **Runner2D** subroutine. Many of them are defined already at line 44. At line 282, the data-writing subroutine **IO** is called. If the user does not want to write data files for whatever reason, this line should be commented out. The subroutine ends with some output file headings and error and notification messages.

Note that two **STOP** commands from the Autolev-generated code are commented out and replaced with **RETURN** commands at lines 370 and 374. These adjustments are necessary if the code is used within an optimization algorithm, where **Runner2D** will be called over and over again. **STOP** will cause the code to hang after the first call.
Aside from specifying parameters, initial values, and storage variables, the user should not change the code in Runner2D. It is important for stepping forward in time and calling the integrator subroutine with a specific set of options and input variables.

D.3.2. EQNS1 Subroutine

The EQNS1 subroutine contains the equations of motion of the model (i.e. the skeletal dynamics). The actual equations of motion are found on lines 520-1350. The user should not change any code within this subroutine, with one exception. Starting at line 468, values for the “specified” variables (COMMON/SPECIFIED block) are calculated. If the user creates any new custom subroutines, they should be called here. If the user creates any new specified variables, they should be calculated here. Existing subroutines can also be removed here by commenting out their CALL statements. Lines 474-481 sum the active and passive joint torques calculated in the MusMod and PasMod subroutines to calculate the resultant joint torques (Tkr for right knee torque, Tml for left MTP torque, etc.).

D.3.3. IO Subroutine

The IO subroutine writes data for the current time step to the output files. It is called at every time step. To create a new output file, first open it and name it in the Runner2D subroutine (line 55), then add a new block of code for this file onto the end of the “Output files added later” section (line 1692). The user will also need to pass the new variables into the IO subroutine using a COMMON block.

The large chunk of code before the output files (lines 1458-1666) calculates some variables that are written to the output files, but were not already calculated in Runner2D.
D.3.4. KUTTA Subroutine

The KUTTA subroutine is the “integrator” that numerically solves the state equations for the state variables. A user’s guide is provided in the subroutine’s preamble (lines 1728-1784). The Kutta-Merson algorithm is used to solve the equations. It is a five-stage numerical method with fourth-order accuracy and variable time stepping. See Fox (1962) for more details. The user should not modify the code here.

D.3.5. SOLVE Subroutine

The SOLVE subroutine solves the matrix equation $Ax = B$ for the vector $x$ using LU decomposition and back substitution. It is called by EQNS1 to solve the state equations for skeletal motion, which are of this form. The user should not modify the code here.

D.3.6. PasMod Subroutine

The PasMod subroutine calculates the passive portions of the joint moments based on the angular positions and velocities of the joints. The output variables are named PAS_XY, where X indicates the joint (H = hip; K = knee, A = ankle; M = MTP) and Y indicates the side of the body (R = right; L = left).

D.3.7. GroundMod Subroutine

The GroundMod subroutine calculates the forces in the ground contact elements underneath the feet based on their linear positions and velocities. GCx1-6 and GCy1-6 are the horizontal and vertical positions of the six elements (1-3 for the elements below the heel, MTP joint, and toe of the right foot, 4-6 for the left foot). GCxv1-6 and GCyv1-6 are the
horizontal and vertical velocities. The output variables are the vertical (\( F_{v1-6} \)) and horizontal (\( F_{h1-6} \)) ground contact forces.

**D.3.8. MusLengths Subroutine**

The **MusLengths** subroutine calculates the origin-to-insertion lengths of the muscles from the current joint angular positions. The output variable is the 18-element vector \( \text{Lm} \), which contains all muscle lengths in the same order as the muscle model state variables (1-9 for the right side muscles, 10-18 for the left side muscles). Joint angles are entered in radians and muscle lengths are calculated in meters.

**D.3.9. MusMomArms Subroutine**

The **MuMomArms** subroutine calculates the moment arm lengths of the muscles to each joint from the current joint angular positions. If a muscle does not span a joint, it is assigned a moment arm of zero. The output variable is the 6x18-element matrix \( \text{Rm} \), which contains all the moment arm lengths. The rows denote the joint (1 = right hip; 2 = right knee; 3 = right ankle; 4 = left hip; 5 = left knee; 6 = left ankle) and the columns denote the muscle, in the same order as the muscle model state variables. Joint angles are entered in radians and moment arm lengths are calculated in meters.

**D.3.10. MusMod Subroutine**

The **MusMod** subroutine calculates rate of change in activation (\( d\text{ACT} \)) and CC length (\( d\text{Lcc} \)) so that the state variables for activation and CC length can be updated for the next time step. It also calculates the muscle forces and the active joint torques that they
generate about the skeleton. MusMod is the most complicated of the user-made subroutines. The first part of the code defines a number of muscle-specific parameters. These parameters are summarized in Table D.1. Next, the current values of the muscle model state variables are copied into two vectors: \texttt{ACT} for the activations and \texttt{Lcc} for the CC lengths (line 2893). The \texttt{MusLengths} and \texttt{MusMomArms} subroutines are then called to obtain values for \texttt{Lm} and \texttt{Rm}.

The main loop for the muscle model algorithm begins on line 2938. The algorithm is calculated for one muscle at a time. For this reason it is important that all vectors storing muscle-related variables and parameters are ordered identically. The first task is to define the excitation level (\texttt{EXC}) for the muscle. This is left as an exercise for the user, as the most appropriate scheme will vary depending on the movement simulated. At line 2941, the user specifies a minimum muscle activation level (\texttt{ACT}), which is needed to avoid zero divides in the state equations. Lines 2946-3001 calculate the state variable derivatives for activation (\texttt{dACT}) and CC length (\texttt{dLcc}) so that the integrator can update the state variables for the next time step. Lines 3003-3061 implement the muscle energy expenditure model of Umberger et al. (2003). \texttt{TER(i)} is the total energy rate (W kg$^{-1}$) for muscle \texttt{i}. \texttt{TER\_sum} is the total energy rate summed over all muscles. The user should adjust the variable \texttt{Sa} on line 3021 depending on the dominant metabolic pathway of the simulated movement (1.5 for aerobic metabolism; 1.0 for anaerobic metabolism).

The last three sections of the MusMod subroutine pass the state variable derivatives to the integrator (lines 3063-3099), adjust the ankle muscle moment arms depending on the current ankle muscle forces (lines 3101-3107; Maganaris et al., 1998, 1999), and calculate the active joint torques from the muscle forces and moment arms (lines 3109-3124).
Table D.1. Parameters of the MusMod subroutine: notation within the code, symbol from Appendix B, and description. CC = contractile component; SEC = series elastic component.

<table>
<thead>
<tr>
<th>Code</th>
<th>Symbol</th>
<th>Description (units)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FT</td>
<td>( FT )</td>
<td>Proportion of fast-twitch muscle fibers (--)</td>
</tr>
<tr>
<td>PCSA</td>
<td>( PCSA )</td>
<td>Physiological cross-sectional area (cm(^2))</td>
</tr>
<tr>
<td>Lo</td>
<td>( L_o )</td>
<td>Optimal CC length (m)</td>
</tr>
<tr>
<td>Lu</td>
<td>( L_u )</td>
<td>Unloaded SEC length (m)</td>
</tr>
<tr>
<td>W</td>
<td>( W )</td>
<td>CC force-length parabola width (multiples of ( L_o ))</td>
</tr>
<tr>
<td>aR</td>
<td>( a_R )</td>
<td>CC force-velocity shape parameter</td>
</tr>
<tr>
<td>bR</td>
<td>( b_R )</td>
<td>CC force-velocity intercept parameter</td>
</tr>
<tr>
<td>Vmax</td>
<td>( v_{max} )</td>
<td>CC maximum shortening velocity (multiples of ( L_o ) per second)</td>
</tr>
<tr>
<td>Ta</td>
<td>( \tau_{act} )</td>
<td>Activation time constant (s)</td>
</tr>
<tr>
<td>Ta</td>
<td>( \tau_{deact} )</td>
<td>Deactivation time constant (s)</td>
</tr>
<tr>
<td>Cecc</td>
<td>( C_{ecc} )</td>
<td>Eccentric plateau (multiples of ( F_o ))</td>
</tr>
<tr>
<td>m</td>
<td>--</td>
<td>Muscle mass (kg)</td>
</tr>
<tr>
<td>Csec</td>
<td>( C_{SEC} )</td>
<td>SEC scaling coefficient (N)</td>
</tr>
<tr>
<td>Ksec</td>
<td>( K_{SEC} )</td>
<td>SEC stiffness (--)</td>
</tr>
<tr>
<td>STEN</td>
<td>--</td>
<td>Specific tension (N cm(^2))</td>
</tr>
<tr>
<td>Fo</td>
<td>( F_o )</td>
<td>Maximum isometric muscle force (N)</td>
</tr>
</tbody>
</table>
APPENDIX E

SIMULATED ANNEALING NOMENCLATURE

This appendix explains the nomenclature of the simulated annealing algorithm used in all optimization problems in this work. The algorithm (Higginson et al., 2005) based on the original work of Goffe et al. (1994). The important parameters are:

- **N** The number of control variables
- **NS** Number of cycles temperature
- **NT** Number of function evaluations per cycles
- **T** Initial temperature
- **RT** Rate of temperature reduction

Although the Goffe et al. (1994) algorithm is loosely based on the thermodynamics of quenching from materials science, the “temperature” of simulated annealing is not exactly a thermodynamic temperature. When temperature is high, the algorithm searches stochastically and has a high probability of jumping to a neighbor solution even if it has a worse objective function score than the current solution. As the temperature decreases, the algorithm focuses on promising areas located in the solution domain. At very low temperatures, the algorithm is essentially deterministic. Temperatures that are “high” and “low” will vary from one problem to another. In general the initial temperature (T) should be high enough that step lengths (VM) cover the entire upper and lower bounds of most control variables for at least the first temperature.

A total of N*NS*NT function evaluations are performed at each temperature. After reaching this total, the temperature is reduced to a new value of RT*T. If the initial
temperature is $T$, after $N \times NS \times NT$ function evaluations, the temperature becomes $T \times RT$. After another $N \times NS \times NT$ evaluations, the temperature becomes $T \times RT^2$, then $T \times RT^3$, etc. This process repeats until the termination criterion is met or until the user terminates the optimization. The parameter NS retains some of the algorithm’s stochastic nature even when the temperature is low. At each temperature, after $N \times NS$ function evaluations the step lengths are adjusted so that half of all function evaluations are accepted regardless of whether they lie uphill or downhill from the current solution.
BIBLIOGRAPHY


Miller RH, Umberger BR, and Caldwell GE (2010). Theoretical limitations to maximum sprinting speed imposed by muscle mechanical properties. *American Society of Biomechanics Young Scientist Predoctoral Award Lecture*, Providence, RI, USA.


Nigg BM and Bobbert MF (1990). On the potential of various approaches in load analysis to reduce the frequency of sports injuries. *Journal of Biomechanics, 23-S1*, 3-12.


Ramsey RW and Street SF (1940). The isometric length-tension diagram of isolated skeletal muscle fibers of the frog. *Journal of Cellular Physiology*, 15, 11-34.


