Sprint Exercise Performance: Does Metabolic Power Matter?

Matthew W. Bundle and Peter G. Weyand

1Biomechanics Laboratory, Department of Health and Human Performance, University of Montana, Missoula, MT; and 2Locomotor Performance Laboratory, Department of Applied Physiology and Wellness, Southern Methodist University, Dallas, TX.

BUNDLE, M.W. and P.G. WEYAND. Sprint exercise performance: does metabolic power matter? Exerc. Sport Sci. Rev., Vol. 40, No. 3, pp. 174–182, 2012. Prevailing physiological paradigms explain both sprint and endurance exercise performance in terms of the availability of metabolic energy. However, for all-out efforts of 60 s or less, the prevailing view is no longer viable. Contemporary evidence indicates that sprinting performance is determined by musculoskeletal force application, with a duration dependency explained by the intrinsically rapid rates at which skeletal muscle fatigues in vivo. Key Words: force application, skeletal muscle, in vivo fatigue, locomotion, biomechanics

INTRODUCTION: TIME-DEPENDENT ENGINES

Imagine purchasing a new sports car and taking it to an empty highway for a performance test. With nothing but open road ahead, you put the gas pedal all the way to the floor. In a matter of seconds, the transmission shifts, the engine revs, and the vehicle accelerates to attain a maximum velocity of 200 km h\(^{-1}\). However, as you settle in at full throttle with the expectation of sustained speed, the engine suddenly begins to lose power. The power losses are rapid at first but become more gradual over time. Eventually, your new engine provides only enough power output to sustain a relatively slow velocity between 50 and 100 km h\(^{-1}\).

As odd as the preceding scenario seems in the context of a man-made engine, from a mechanical and temporal standpoint, this is precisely how the muscular engines of humans and other animals perform. Although natural and manufactured engines can be similar in relying on chemical energy to generate force and power, their performance-duration relationships are strikingly dissimilar. Thus, we quite naturally expect automobile and other man-made engines to perform without fatiguing, but the biologically equivalent prospects of an elite human sprinter finishing a mile run in just over 2 min or a cheetah galloping through 26 miles in less than half an hour do not seem remotely possible. Yet the respective top speeds of these two athletes, if sustained, would permit these performances.

Instead, personal experience and observation lead us to expect rapid decrements in performance capabilities any time the duration of a maximal physical effort becomes more prolonged. The duration dependency of performance that has been well characterized for humans and other animals appears in general form in Figure 1. As illustrated by the negative exponential nature of the relationship, the greatest decrements in performance occur across those efforts that span the briefest durations. For example, the decrements that occur as effort durations extend from 2 to 30 s are much larger than those that occur with duration increases from 30 to 60 s which exceed those that take place from 60 to 120 s, etc. This pattern of exponential decrease continues until durations extend to between 5 and 10 min, where performance falls to the levels that can be well sustained by the body’s renewable aerobic sources of energy (13). Thereafter, performance decrements become relatively small, even as durations extend to several hours.

Here, we consider the mechanical and metabolic factors responsible for the duration dependency of biological engines. We start with the mechanics that directly determine performance during burst sprint activities of a few seconds or less and then consider how these requirements change as sprint durations increase. We also evaluate the prevailing view that the duration dependency universally observed for biological engines results from the availability of chemical energy to provide fuel to the active muscles. Although this view has been largely unchallenged in the last half century, reconsideration is warranted given that more extensive evidence now available.

Address for correspondence: Peter G. Weyand, Ph.D., Locomotor Performance Laboratory, 5538 Dyer Street, Suite 105, Department of Applied Physiology and Wellness, Southern Methodist University, Dallas, TX 75206 (E-mail: pweyand@smu.edu).

Accepted for publication: March 27, 2012.

Associate Editor: Daniel P. Ferris, Ph.D.
Our analysis draws largely on the all-out running speeds and cycling power outputs of humans because of the extent and quality of the data available and because the mechanical and metabolic contrasts between these two exercises provide informative scientific insights. Although we rely heavily on the experimental evidence from these two modes of human sprinting, we expect our conclusions to generalize to nonhuman species and any activity that engages a large fraction of the body’s musculature for basic biological reasons. The structural and functional properties of the musculoskeletal system are largely invariant across species (11,26) as are the pathways of chemical energy provision within skeletal muscle (15). Our analysis focuses on all-out efforts in the nonsustainable range of durations of up to roughly 5 min, with a particular emphasis on those efforts lasting less than a single minute. We have focus on the duration range in which performance decrements are greatest to provide the most rigorous evaluation of existing scientific ideas.

**THE MECHANICAL BASIS OF SPRINTING PERFORMANCE: EXTERNAL FORCE APPLICATION**

In the simplest terms, performance can be analyzed by considering either the input or, the output from, the skeletal muscles that serve as biological engines: the input being the chemical energy that fuels muscular contraction and the output being the force or mechanical power that the contractions produce. Generally, performance in both sprint and endurance events has been causally attributed to the chemical energy available: 1) how well metabolic models explain endurance performance (8,13,22) and 2) the absence of data to refute them.

We have opted to deviate from the classical approach by focusing on the mechanical output of the musculoskeletal system that can be measured rather than the chemical energy input that cannot. The understanding of the relationships between force, motion, and performance provided by classical Newtonian mechanics support the viability of this approach. From the respective whole-body mechanical entities provided by the external forces applied, either running speed or cycling power output, simple quantitative performance relationships have been put forth (5,34,35):

$$\text{Spd} = \frac{F_p}{F_{Wb}} \cdot L_s \cdot \text{Freq}_{\text{aer}}$$

(1)

$$P = F_p \cdot L_{ds} \cdot \text{Freq}_{\text{aer}}$$

(2)

where \(\text{Spd} \) and \(P\) represent running speed and cycling mechanical power output, \(F_p/F_{Wb}\), and \(F_p\) represent the external applied forces, for running, the stance-averaged vertical ground force as a multiple of the body’s weight, and for cycling, the average down-stroke pedal force. The length terms, \(L_s\), and \(L_{ds}\) represent the forward distance through which the body travels while the foot is in contact with the ground during running and the distance through which the pedal force is applied during each cycling down-stroke (i.e., one half of the pedal circumference). Finally, the frequency terms, \(\text{Freq}_{\text{aer}}\) and \(\text{Freq}_{\text{aer}}\), are the inverse of the step and down-stroke times, where the former is defined as the sum of one contact and one aerial period.

These force-performance relationships have several features that should be noted. The running equation does not include the horizontal component of the ground reaction force because these forces are relatively small and contribute limitedly to the magnitude of the total ground reaction force during constant-speed sprint running without wind resistance (34). Our cycling equation does not include the condition-specific factors that introduce variability into the overground power-speed relationship. In addition, the forces determining performance are mass-specific for the exercise that is weight-bearing (equation 1) and absolute for the exercise (equation 2) that is not.

The general relationship between the external forces the skeleton applies to the environment, and the level of performance attained is illustrated in Figure 2A, 2D. The forces appearing in the figure represent those typical of athletic subjects tested in the two primary modes of sprinting examined here. In both cases, the threshold separating sustainable and nonsustainable forces occurs at the minimum level of mechanical performance that can be supported by the maximum rates of aerobic metabolism. Note that although the
maximum rates of aerobic metabolism available to support external force application in these two exercises is virtually the same (6,33), the relative forces, timing of force application, corresponding rates of energy utilization, and the relative intensities attained are not (Fig. 2B, C, E, F). During running, the aerobic power of well-trained subjects typically can sustain ground forces that are twice the body’s weight and 75% to 85% of those applied during a top speed sprint. In contrast, the pedal forces that can be sustained by the same level of aerobic power during cycling are only one fourth of the body’s weight and only 25% to 35% of those applied during a burst cycle sprint when peak power output is achieved.

However, in both exercises, the force-performance relationships presented in Figure 2 are reasonably linear across the full range of endurance and sprint exercise intensities. These close force-performance relationships result from limited variation in the length and frequency terms in our respective force-performance equations. Specifically, running contact lengths are a narrow function of leg lengths and exhibit little variation as runners increase from their intermediate to top sprinting speeds (34,35). Cycling down-stroke lengths are fixed mechanically by crank dimensions that are largely standardized across different bikes and riders. Similarly, the respective stride and pedal frequencies that maximize burst sprint performances exhibit modest variation between individuals during running (35) and almost no variation during cycling (19). Thus, for both exercises, differences in sprinting performance are predominantly a function of the magnitude of the external forces applied because length and frequency variation is limited.

In addition to the deterministic relationship between external force application and performance, external forces also appear to be reasonably representative of the extensor muscle forces required. The data currently available from techniques that estimate the minimum net extensor forces acting across the joints of the limb suggest that the relationship between

Figure 2. The direct relationship typically observed between the external force applied and level of performance attained for cycling (A) data from (33) and running (D) data from (6,35). Consecutive periods of force application by opposite limbs occur in immediate succession during cycling (B) but are separated by aerial periods without force application during running (E) data from (34)). Hypothesized rates of chemical energy utilization by the contractile elements of the extensor muscles activated during sprint cycling (C) and running (F) are presented schematically as square waves for simplicity. The horizontal bars above the force waveforms in (B) and (E) identify the durations of pedal revolutions or stride times and the periods of external force application. The ground and pedal forces illustrated correspond to the \( F_g \) and \( F_p \) terms in equations 1 and 2, respectively standardized to body weight.
the external forces applied and the net muscle forces generated across the joints most relevant for performance is relatively constant. During running, the ankle and knee extensors generate forces that are roughly two to three times greater than the ground forces applied (3). During cycling, the knee and hip extensor forces are roughly three times greater than the pedal forces applied (3,12). Accordingly, within each mode, the external forces applied during sprinting appear to be consistently related to the corresponding muscle forces regardless of the intensity or duration of the effort.

NEURAL CONTROL AND MAINTENANCE OF FORCE APPLICATION

The recognition that sprinting performance and its duration dependency are set directly by the external forces applied to the environment begs two questions of immediate relevance. First, what determines the maximum external forces that the musculoskeletal system can apply during brief, all-out, burst-style sprints? And, second, why do the external forces applied become progressively smaller as the duration of sprinting increases even though the effort being put forth is maximal? In the first case, understanding the factors determining the maximal dynamic limb extensor forces that can be applied, during sprint running particularly, remains to be established (34). In the second case, insight into the mechanisms of force impairment can be gleaned from the patterns of neuromuscular activation observed during all-out sprint trials.

Selecting and maintaining the external forces needed for a sprint trial of any given intensity requires a fairly precise mechanism for controlling muscle force generation. Force outputs are regulated primarily by the number of motor units and, therefore, muscle fibers activated and secondarily by frequency modulation within the activated units (4,28). Experimentally, the levels of neuromuscular activation resulting from both neural control mechanisms can be assessed by surface electromyography (EMG) to measure the electrical activity resulting from membrane depolarization of the activated muscle fibers. For both static and dynamic contractions requiring similar limb positioning and relative shortening velocities, external force application is related directly to the rectified and integrated EMG (iEMG) signal (4). As can be seen in Figure 3A, for the vastus lateralis muscle that extends the knee, the iEMG-external force relationship is linear over a sixfold range of pedal forces from 100 to 600 N at a pedal cadence of 100 rpm when fatigue is not present.

With this relationship in place, we next considered the neuromuscular activity-external force relationship across the full time course of all-out sprint trials of different intensities. For both sprint cycling and running, trials to failure were administered at constant intensities to hold the external and joint extensor forces required relatively constant. Three sprint cycling trials (Fig. 3B) were administered with pedal forces exceeding 300 N and, therefore, in the nonsustainable force range for this individual, with the fourth being administered at a sustainable force level of 100 N. The sprint running trial in Figure 3 was administered at a nonsustainable treadmill running speed of 7.3 m s⁻¹. For all three of the sprint cycling trials in Figure 3B, the iEMG activity of the vastus lateralis muscle continuously increased to maintain the constant pedal forces the trial required. Similarly, during the sprint running trial, the iEMG activity of the extensor muscles, which were monitored continuously, increased throughout the 47-s trial.
illustrated while the ground forces remained relatively constant (Fig. 3C, D). In contrast, in the nonsprint cycling trial that required relatively low sustainable pedal forces, the iEMG activity remained essentially constant during the course of the trial.

The EMG data provide several conclusions regarding the maintenance of the external forces applied during all-out sprinting. First, for all the sprint cycling and running trials examined, the levels of neuromuscular activation needed to maintain a constant external force increased continuously from the outset to the conclusion of the trial. Second, the rates of increase in the compensatory neuromuscular activity observed were typically more rapid for the briefest trials requiring the greatest forces. Third, no increases in neuromuscular activity were observed when the force required was sufficiently small to be fully supported by aerobic metabolism. And, fourth, the levels of neuromuscular activation at the point of trial and force failure were lower for the longer duration sprint trials that required lesser forces.

These relatively simple neuromuscular experiments indicate that all-out sprinting performances are highly duration dependent because of the rapidity of musculoskeletal fatigue in vivo during dynamic exercise requiring large force outputs. The timing and intensity-dependent nature of the force impairment observed complements the functional understanding of muscle force production at both the systemic and cellular levels. In the former case, fatigue, as indicated by compensatory neuromuscular activity, more rapidly occurs in those sprints that require greater external forces that activate and rely on faster, more fatigable, muscle fibers (1,4,28). In the latter case, the virtually instantaneous and intensity-dependent nature of the fatigue observed is consistent with a cellular-level force-impairment mechanism that is believed to be brought about by the metabolic by-products of the cross-bridge cycle itself (1). In addition, the lower iEMG values generally observed at the failure point for longer versus shorter trials raise the possibility that maximum levels of neuromuscular activation may be systematically reduced as the duration of all-out sprint trials is increased.

With a mechanistic explanation for the progressive impairment of musculoskeletal forces identified, we next investigated whether the duration dependency of sprinting performance might somehow be linked to the mechanics of external force application.

**FROM PERFORMANCE VARIABILITY TO A FORCE MODEL FOR SPRINTING**

From the outset of our own experimental efforts, we used a design strategy of altering three independent variables to maximize the sprint performance variation observed. First, we recruited individuals with large differences in their sprint performance capabilities (5,30,35). Second, we administered all-out sprint trials across a broad range of durations from 2 to 300 s during which we knew a priori that performance levels would vary considerably (5,6,30,33). Third, we compared performances across different modes of sprint locomotion (33). A representative sample of the performance variability observed is illustrated for two runners and two cyclists in Figure 4A.

Our initial objective was to standardize the variation attributable to the first of our independent variables: individual performance differences. After compiling a sizable data set for sprint running, we found that individual differences could be standardized using a simple two-step process (6,30,33). Step 1 is quantifying the upper and lower intensity limits that bracket the range of sprinting performances for each individual: 1) the maximum burst sprint of 2 s or less (Spd_{burst} for running; P_{burst} for cycling) and 2) the minimum intensity that elicits the maximum rate of aerobic metabolism (Spd_{aer} for running; P_{aer} for cycling). The difference between these upper
and lower limits represents the full range of nonaerobic and therefore nonsustainable speeds ($Spd_{nonsust}$) or power outputs ($P_{nonsust}$) possible for sprint efforts. Step 2 is standardizing sprinting speeds or power outputs by expressing the performance achieved as a fraction of the individual’s nonsustainable speed or power reserve (e.g., $Spd_{nonsust} = 1.0$, $Spd_{nonsust} = 0.0$, etc.; see Fig. 4B).

In comparative terms, the fraction of an individual’s nonsustainable speed or power is the sprinting equivalent of expressing endurance exercise intensities as a fraction of an individual’s maximum aerobic power or $\dot{V}O_2_{max}$. Because endurance efforts predominantly rely on aerobic or sustainable sources of metabolic power, relative intensities need only be referenced to one variable: the minimum intensity eliciting the maximum aerobic power of the performer (6,30). In contrast, sprinting efforts rely on both sustainable and nonsustainable sources of metabolic power and, therefore, need to be referenced to two variables: both the burst and aerobic maximum of the performer. Here, our primary impetus for developing an index of relative sprint exercise intensities was the potential for predicting all-out sprint trial durations.

Our expression of relative sprinting intensities did, in fact, lead us to a direct means by which to quantify the variability introduced from our second independent variable: sprint trial duration. This is illustrated in the first two panels appearing in Figure 4. When the absolute sprinting performances in Figure 4A were expressed as fractions of the nonsustainable speed or power of the individual performers, their relative sprinting intensities fell in essentially the same duration-dependent manner within the respective modes (Fig. 4B). Thus, the relationship between trial duration and relative sprinting intensity can be described using single mode-specific exponents ($k_{run}$ or $k_{cycle}$) that provide the respective curves:

Running: $Spd_{f-NS}(t) = e^{-k_{run} \cdot t}$

Cycling: $P_f-NS(t) = e^{-k_{cycle} \cdot t}$

where $Spd_{f-NS}(t)$ and $P_f-NS(t)$, respectively, represent the fraction of the nonsustainable speed and power that can be maintained for an all-out sprint trial of duration $t$, $e$ is the base of the natural logarithm, and $k_{run}$ and $k_{cycle}$ are the exponents that describe the duration-dependent decrements in relative sprinting intensities within each mode of exercise. Validations of our model using hundreds of running trials administered to both sprint and endurance athletes, over a broad range of durations and in both field and laboratory settings, have predicted the performances observed to within 2% to 4% on average (6,30).

The performance variability introduced by the third variable, mode of exercise, was perhaps the most difficult to quantify as there is no standardized approach for equating absolute running speeds and cycling power outputs. Moreover, even after standardization of the different absolute speed and power output values to relative sprint intensities, large between-mode differences in the relative intensity-duration relationship were present (Fig. 4B). Relative sprint cycling performances fell more sharply in relation to trial duration than relative sprint running performances did, as reflected in the twofold difference in exponential values (33), providing the best empirical fits to the respective data ($k_{run} = 0.013 s^{-1}$; $k_{cycle} = 0.026 s^{-1}$).

We attribute this twofold, between-mode difference to a corresponding difference in the fractional duration of external force application in the different modes of sprint exercise. Each pedal revolution involves consecutive periods of limb-pedal force application by the right and left legs that occur in virtually immediate succession. In contrast, consecutive periods of limb-ground force application during each running stride are separated by aerial periods of equivalent duration during which no ground force is applied (Fig. 2B, E). Accordingly, the fraction of the total sprint time that involves external force application by a single limb to the pedal or ground, i.e., the duty factor (DF), is two times greater for cycling than sprint running ($DF_{run} = 0.24; DF_{cycle} = 0.50)$.

When duration-dependent decrements in relative sprint cycling and running performance are expressed in terms of the time of external force application only (trial time $\times DF$), rather than the total sprinting time, decrements in the two modes conform to a common relationship (Fig. 4C).

This third model element quantitatively links the duration dependency of performance expressed in equations 3 and 4 to the mechanics of external force application originally introduced in equations 1 and 2. Given the limited variation of the length and frequency terms in equations 1 and 2 previously noted, the performance-duration relationship predominantly reflects a duration dependency in the maximal forces the musculoskeletal system can produce and apply externally (33). The existence of an apparently common relationship between relative sprinting intensities and the duration of external force application across two mechanically distinct modes of sprint exercise has several basic implications. First, at the whole-body level, that fraction of the sprint running speed or cycling power output provided by nonsustainable anaerobic sources of chemical energy has a discrete duration dependency dictated by the cumulative duration of external force application. Second, the duration dependency observed results from a rapid progressive impairment of muscular force resulting from a reliance on anaerobic sources of chemical energy to fuel the contractions dictated by the mechanics the exercise requires. This demand-driven, fatigue-based explanation is fully consistent with numerous observations: the virtually immediate and progressive fatigue evidenced in our EMG data (5), rates of fatigue that are intensity dependent, the more rapid time course of fatigue in cycle versus run trials of similar duration (Fig. 4B), and muscle force impairment at the cellular level resulting from the metabolic by-products of a reliance on anaerobic metabolism to fuel the contractile activity supporting external force application (1,5,33).

Although aspects of our sprinting performance model remain a work in progress, our design strategies and force application framework have provided empirical, predictive, and testable outcomes that have not come forth from the energy supply-limit models. These include quantification of relative sprinting intensities, identification of a common duration dependency of relative sprinting performances, linking the duration dependency of performance to external force application,
and the identification of a force impairment explanation for the duration dependency of sprinting performance that can be tested at the tissue and cellular levels.

**METABOLIC ENERGY RELEASE DURING SPRINTING: DRIVEN BY DEMAND OR LIMITED BY SUPPLY?**

The tradition of conceptualizing all-out locomotor performance as a metabolic energy input and, therefore, supply-limited endeavor originated nearly a century ago with the work of independent contemporary scholars, A.V. Hill (10,11) and R. Margaria (16–18). Hill (10,11) related approximations of the metabolic energy available to record performance data from a variety of species and modes of human locomotion. Margaria et al. (16,17,18) attempted to quantify the maximum rates of chemical energy release during all-out runs of different durations via direct experimentation. Although both investigators provided the foundations for energy supply-limit modeling that continues to this day (Hill: 13, 14, 22, 27, 29; Margaria: 8, 9, 23), they reached opposite conclusions regarding sprint performance limitations. Margaria et al. (16,17) were sufficiently convinced of an energy supply limit, even for burst-type sprints as short as 2 to 3 s, that they introduced the term “anaerobic muscular power” to describe them. They further proposed that sprint performances measured in mechanical units should be expressed in metabolic terms (17). In contrast, in his original 1925 work on performance limits, Hill stated, “It is obvious that we cannot pursue our (metabolic energy supply) argument to times below about 50 s,” because these performances are limited by factors “mechanical and nervous” (10). Nonetheless, nearly a century after Hill published his conclusion, the supply-limit models he inspired continue to be applied (14,22,27,29) to the very sprint performances that he recognized they could not explain.

The numerous energy supply-limit models that have come forth since Hill and Margaria differ in their specific features but share a common characteristic: none have been validated empirically because the data required to do so are not available. In the continued absence of valid whole-body anaerobic energy release measurements, these models have been formulated with largely uncertain and widely varying assumptions (2) regarding the quantities of the anaerobic and aerobic energy available, their respective release rates, and the efficiency with which chemical energy is converted into speed, power, and force. Thus, the close fits that these models can provide to performance data are achieved by incorporating assumptions that have unknown or poor (29) validity, as aptly noted (2,27). Critical consideration of the explanations these models offer for sprinting performance is overdue, particularly given the performance-duration patterns that are now available for well-controlled sprint trials of very brief durations.

In their original view of burst and brief sprint exercise performance, Margaria et al. (16,17) estimated that chemical energy resupply to the contractile machinery could operate at maximal power for durations of 5 to 6 s. Hence, these investigators also believed that maximal sprinting intensities could be maintained for durations of 5 to 6 s before further increments in duration and slowing rates of energy resupply would compromise performance. Yet, the data now available demonstrate that performance decrements begin to follow a negative exponential pattern that occurs either instantaneously at the outset of exercise or within the first 2 to 3 s (5,6,33). Thus, in contrast to the anaerobic muscular power limitation proposed by Margaria, the greatest decrements in sprinting performance occur precisely during those very brief durations during which 1) the rates of anaerobic energy resupply to the contractile machinery are most rapid and 2) intracellular stores of chemical energy are greatest.

A second difficulty with the energy supply-limit models is mechanistic inconsistency with energetic measurements at the cellular level. One of the most widely noted features of muscle cell metabolism is the relative constancy of intracellular concentrations of the adenosine triphosphate (ATP) molecule that serves as the immediate source of chemical energy to the contractile proteins. This well-regulated maintenance, even during the most intense contractile periods, is attributable to the rapid one-step creatine phosphokinase reaction that resupplies ATP. Accordingly, this near-equilibrium reaction is widely regarded as a temporal buffer that safeguards intracellular energy stores (15). Indeed, measurements made possible within living skeletal muscle by nuclear magnetic resonance spin technology indicate that the phosphocreatine reaction is capable of resynthesizing ATP several times more rapidly than the contractile proteins within the muscle cells can use it (21). Thus, the rate-limiting step in the release of chemical energy at the cellular level has been conclusively shown to be the contractile event that uses the energy and not the metabolic pathways that resupply it.

Beyond the mechanistic inability to explain whole-body performance patterns and cellular-level energetic data during intense periods of contractile activity, energy supply-limit models also imply or predict that 1) sprinting performance should be impaired when the total metabolic power available is reduced and 2) sprinting performance should be largely unaffected by interventions that alter the mechanics of sprint exercise. Neither expectation has been borne out by the whole-body data that are now available. In the first case, hypoxic conditions have been used to reduce the availability of metabolic energy from aerobic metabolism during brief all-out sprint efforts. In our running experiments (32), we found little difference between normoxic and hypoxic sprint performances lasting 60 s or less, despite aerobic contributions that were reduced by as much as 25% in the hypoxic condition. In the second case, mechanical interventions that prolong the lengths of external force application in accordance with our original force-performance equations (equations 1 and 2) have consistently enhanced sprinting performance. These include elliptical pedal orbits that increase single-leg cycling power outputs (20) by prolonging the down-stroke portion of the pedal stroke (equation 2); artificially compliant, lightweight, double-lower limb prostheses that enhance running speeds (31) by prolonging contact lengths; reducing limb repositioning times and elevating stride frequencies (equation 1); and hinged-blade ice skates that increase speed by prolonging the duration of the push-off portion of the skating stance phase (7).
CONCLUSIONS: DOES METABOLIC POWER MATTER FOR SPRINTING?

The conclusion that sprinting is not energy supply-limited as traditionally conceived (8,9,14,16–18,22,23,27,29) prompts the general question of the functional role metabolism does play and the specific question posed in our title: does metabolic power matter for sprinting performance? For burst-type sprints that last only a few seconds, a wealth of data spanning multiple levels of biological organization is fully consistent in indicating that the availability of metabolic power neither determines nor directly limits performance. These burst sprints predominantly reflect musculoskeletal function and not the “anaerobic muscular power” of Margaria et al. (16,17) or the many anaerobic fitness parameters that evolved subsequently. Nonetheless, metabolic power does assume progressively greater functional relevance as the duration of all-out sprinting extends from a few seconds to a few minutes, but in this case also, not in keeping with the traditional conceptualization. The predictive success of our force application model, both within and across modes, indicates that as efforts extend from a few seconds to a few minutes, the fractional reliance on anaerobic metabolism progressively impairs whole-body musculoskeletal performance and does so with a rapid and remarkably consistent time course. In this respect, the sprint portion of the performance duration curve predominantly represents, not a limit on the rates of energy resupply, but the progressive impairment of skeletal muscle force production that results from a reliance on anaerobic metabolism to fuel intense sequential contractions (Fig. 1).

Thus, the duration dependence of the performances of elite human sprinters, cheetahs, and other vertebrate animals that rely on skeletal muscle is attributable to the provision of chemical energy from both sustainable and nonsustainable sources in their natural engines. In contrast to synthetic engines that can convert chemical energy into force and mechanical power with relatively constant efficiency and without fatiguing, skeletal muscle has an intrinsic duration dependence directly linked to that proportion of the muscular force derived from the nonsustainable, anaerobic sources. From a design standpoint, these nonsustainable energy sources markedly enhance the range of musculoskeletal performances possible, but do so only transiently because the additional mechanical function provided is compromised so rapidly.

In closing, we offer three basic conclusions regarding sprint exercise performance and a biological contrast they reveal. First, the view that brief all-out exercise performance is directly limited by rates of chemical energy provision to the contractile machinery in skeletal muscle is no longer supportable. Second, the metabolic energy released during sprinting is demand-driven and not supply-limited. Third, sprint exercise performance is determined by the application of musculoskeletal forces with a duration dependency dictated by how rapidly these forces are compromised by rates of fatigue in vivo.

Finally, we note that the relationship between exercise mechanics, metabolism, and performance differs fundamentally between sprint and endurance exercise. Although a common relationship has traditionally been assumed to generalize across a broad duration continuum of sprint and endurance efforts, contemporary evidence indicates otherwise. For endurance events, the metabolic energy available via sustainable, aerobic sources of metabolism predominantly determines performance by setting the intensity of the musculoskeletal mechanics that can be sustained throughout the effort. For sprint efforts, precisely the opposite is true: the intensity of the mechanical activity that the musculoskeletal system can transiently achieve determines the quantities of metabolic energy released and the level of performance attained.

Acknowledgments

Because of manuscript length and reference restrictions, the literature on neuromuscular fatigue and critical power could not be incorporated and acknowledged to the extent warranted by existing scholarship in these areas. This work was made possible by award DAMD 17-02-2-0053 from the U.S. Army Medical Research and Materiel Command and the Telemedicine and Advanced Technology Research Center to P.G.W. The body of scholarly work presented would not have been possible without the support and guidance of Reed W. Hoyt and the late C. Richard Taylor. The authors declare no conflicts of interest.

References


34. Weiland PG, Sandell RF, Prime DN, Bundle MW. The biological limits to running speed are imposed from the ground up. J. Appl. Physiol. 2010; 108:950–61.