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A metabolic basis for impaired muscle force production and neuromuscular compensation during sprint cycling

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Bundle, Matthew W., Carrie L. Ernst, Matthew J. Bellizzi, Seth Wright, and Peter G. Weyand. A metabolic basis for impaired muscle force production and neuromuscular compensation during sprint cycling. *Am J Physiol Regul Integr Comp Physiol* 291: R1457–R1464, 2006. First published July 13, 2006; doi:10.1152/ajpregu.00108.2006.—For both different individuals and modes of locomotion, the external forces determining all-out sprinting performances fall predictably with effort duration from the burst maximums attained for 3 s to those that can be supported aerobically as trial durations extend to roughly 300 s. The common time course of this relationship suggests a metabolic basis for the decrements in the force applied to the environment. However, the mechanical and neuromuscular responses to impaired force production (i.e., muscle fatigue) are generally considered in relation to fractions of the maximum force available, or the maximum voluntary contraction (MVC). We hypothesized that these duration-dependent decrements in external force application result from a reliance on anaerobic metabolism for force production rather than the absolute force produced. We tested this idea by examining neuromuscular activity during two modes of sprint cycling with similar external force requirements but differing aerobic and anaerobic contributions to force production: one- and two-legged cycling. In agreement with previous studies, we found greater peak per leg aerobic metabolic rates [59% (±6 SD)] and pedal forces at $\dot{V}O_{2\,peak}$ [30% (±9)] during one- vs. two-legged cycling. We also determined downstroke pedal forces and neuromuscular activity by surface electromyography during 15 to 19 all-out constant load sprints lasting from 12 to 400 s for both modes of cycling. In support of our hypothesis, we found that the greater reliance on anaerobic metabolism for force production induced compensatory muscle recruitment at lower pedal forces during two- vs. one-legged sprint cycling. We conclude that impaired muscle force production and compensatory neuromuscular activity during sprinting are triggered by a reliance on anaerobic metabolism for force production.

motor control; aerobic and anaerobic contributions; performance

HUMAN LOCOMOTOR PERFORMANCE depends directly upon the forces that the musculoskeletal system can generate and transmit to the environment. For example, runners and cyclists modulate their speed and power output primarily by altering the respective forces they apply to the ground (61) or pedals (29). The maximum external forces applied at top running speed or at peak cycling power output can be supported by skeletal muscle for only 3 s or less (41, 61). As the duration of all-out efforts become more prolonged, the external forces that the musculoskeletal system can provide become progressively reduced. As a result, performances decline from the 3-s maximum to a nearly sustainable level as the duration of all-out efforts extends to 300 s (10, 17, 30). The reductions in force production responsible for the well-characterized performance-duration relationship have been considered by some investigators to be a whole-body manifestation of muscle fatigue (9, 62). Given the rapidity with which the duration-dependent decrements in force production occur, shorter-duration, all-out efforts provide a potentially powerful approach for examining the time course of muscle fatigue in vivo.

In contrast to the consistent decrements in all-out locomotor performance observed in humans and other species for efforts of increasing duration (17, 30), the extent and time course of fatigue measured from isolated muscle preparations are variable and appear to depend on the stimulation protocol (4, 11, 16, 42). Although the time courses can vary considerably, in vitro and in vivo decrements in force production may have a common metabolic basis. Isolated muscle fibers contracting at low frequencies in oxygenated solutions (31, 42) and in vivo muscle active at levels below the maximum rate of aerobic metabolism (13) show little indication of decrements in force production over time. In contrast, muscle fibers contracting in anoxic solutions (31, 38) and individuals relying heavily on anaerobic pathways of ATP resynthesis (60) experience impaired force production after relatively few contractions. The mechanism responsible for the rapid decrements in force has been studied extensively but remains incompletely understood (14, 18, 25, 49).

Regardless of the mechanism, during prolonged in vivo contractions at elevated fractions of the maximum force available, skeletal muscle fatigues rapidly. To maintain a constant external force output, the nervous system can compensate by either increasing the rate of stimulation or recruiting additional muscle fibers (24). The progressive increase in neuromuscular activity characteristic of high-force, intermittent contractions (33, 45) generally occurs via motor unit recruitment (43, 55). In contrast, at lower force levels, those equal to 30% or less of the maximum voluntary contraction (MVC), intermittent contractions can be sustained with steady levels of neuromuscular activation and do not appear to require compensatory muscle recruitment (21, 40).

Although the mechanical and neuromuscular responses to fatiguing contractions are generally considered with respect to the fraction of the MVC, the mechanism triggering compensatory recruitment may be metabolic. Our recent work with short-duration, all-out running and cycling efforts (10, 58, 60)

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suggests that duration-dependent decrements in sprinting performance may be determined by the metabolic pathways supporting force production regardless of the absolute level of force generated. Accordingly, applying pedal forces greater than the maximums that can be supported by aerobic metabolism results in compensatory muscle recruitment to maintain performance (20, 45), whereas lower pedal forces are maintained with constant neuromuscular activity (45).

Here, we employed an experimental tool that allowed us to alter the aerobic and anaerobic contributions to the external forces produced during all-out sprint locomotion. Specifically, we took advantage of the greater pedal forces that could be supported aerobically during one- vs. two-legged cycling to evaluate whether compensatory recruitment would be triggered at the same absolute pedal force or by a reliance on anaerobic metabolism for force production. We knew from previous work that the timing of pedal force application (54) and muscle activation patterns (36) are similar during one- and two-legged cycling, but that the maximum pedal forces that can be supported by aerobic metabolism are not. On a per leg basis, rates of oxygen uptake during one-legged cycling have been found to be at least 40% greater than during two-legged cycling (15); thus, allowing greater muscle and pedal forces to be supported aerobically in the one-legged mode.

We hypothesized that the duration-dependent decrements in external force production that occur during all-out sprint cycling result from impaired muscle force production and that the impairment has a metabolic basis. We specifically predicted that a reliance on anaerobic metabolism for force production during locomotion compromises muscular force production, thereby triggering compensatory recruitment during all-out efforts. In accordance with the greater pedal forces that can be supported by the maximum rates of aerobic metabolism in one- vs. two-legged cycling, we expected the onset of compensatory neuromuscular activity to occur at a greater pedal force threshold in the one-legged mode. Conversely, for all-out sprint efforts requiring equivalent pedal forces, we anticipated that compensatory neuromuscular activity would be greater during two- vs. one-legged cycling.

MATERIALS AND METHODS

Subjects. Six males, 22–36 years of age [mass, 75.2 kg (±7.2 SD)]; height, 179.5 cm (±2.6) volunteered to participate in this study and provided their written informed consent in accordance with the protocol reviewed and approved by the Institutional Review Board of Harvard University. All of the subjects were moderately to highly fit. Five of the six subjects were actively engaged in endurance training at the time of the study; two subjects were trained cyclists, two were former competitive runners, one was a cross-country skier, and one was active but did not exercise regularly.

Testing protocol. Subjects completed a minimum of eight testing sessions, which were separated by at least 48 h of rest. In the first two sessions, subjects underwent progressive discontinuous tests to determine $\dot{V}O_{2\ peak}$ and their steady-state rates of oxygen uptake at a series of different work rates with one- and two-legs, respectively. In sessions three through eight, subjects performed one- or two-legged all-out cycling bouts at work rates that elicited failure between 15 and 400 s. Subjects performed five sprints with one-leg during three of the sessions and five sprints with two-legs during the other three sessions. Within sessions, subjects were instructed to take as much rest between sprints as they deemed necessary to be fully recovered but were required to take a minimum of 10 min. Subjects alternated one- and two-legged conditions between successive test sessions.

Mechanical power and pedal forces. All tests were completed on a stationary, friction-braked cycle ergometer (Monark Ergomedic 818 E) equipped with drop bars, a racing saddle, and pedal straps. Each ergometer trial was conducted at a constant cadence of 100 rpm. This ensured that differences in power output were due to differences in pedal force, rather than differences in both force and velocity.

One-legged tests were performed using the dominant leg with subjects sitting in the same position as for the two-legged tests with the inactive leg supported by a stool adjacent to the vacant pedal. During the one-legged tests, the vacant pedal was weighted with a 9.4-kg counterweight to facilitate a smooth pedaling motion. For the sprint trials, the ergometer was loaded over a 2-s period by gradually releasing a hand-held spring clamp to apply the desired tension to the flywheel. Timing of the sprint duration began immediately after the load had been applied. Subject feedback for pedal cadence was provided with a tachometer equipped with video display and a metronome that beat 100 times per minute. The tachometer measured the revolutions from reflective tape on the flywheel, which triggered a photocell (Banner Engineering, Valu-Beam SM912LV) mounted at the base of the ergometer. Pulses from the photocell were digitized (National Instruments model NB-MIO-16H) and recorded in custom-designed software (National Instruments, LabView 4.0). The software converted flywheel revolutions to pedal revolutions using the ergometer gear ratio (52:14).

During short-duration high-intensity efforts, measurements of mechanical power from friction-braked cycle ergometers underestimate the power supplied by the subject if the inertial work done to accelerate flywheel is omitted (37, 44). We accounted for this by using a post hoc correction modeled after Morin and Belli (44) that incorporates the flywheel velocity. For the one-legged trials, we also added the work done to accelerate the counterweight (radius = 0.175 m). The influence of these corrections is greatest for trials of very short duration. During the shortest sprints completed by the subjects, the corrected power output was 4.4% (±1.6) (range: 7.3 to 2.4) greater than that to overcome friction alone.

We calculated the average force applied to the pedals per revolution (F_{avg}) from power output in accordance with

$$F_{avg} = \frac{\text{Power Output}}{\text{Radius} \cdot 2\pi(\text{Freq})} \quad (1)$$

where radius was the length of the crank arm (0.175 m) and Freq was the measured mean cadence in revolutions per second. Because the pattern of force application during two-legged cycling results in one of the two legs always being in a downstroke, $F_{avg} = F_{ds}$, where F_{ds} is the average force applied to the pedals during the downstroke period. However, for one-legged cycling, force can only be applied during the downstroke period of the active leg, and since revolutions with constant angular velocity have equal downstroke and upstroke periods, the F_{ds} at a given power output for one-legged cycling is

$$F_{ds} = 2 \cdot F_{avg} \quad (2)$$

This method provides the time-averaged force applied during each downstroke. Because our measures of electromographic activity are also downstroke averages, this approach allows the most direct comparison between pedal force application and neuromuscular activity. The average pedal force during downstroke, F_{ds}, is referred to as pedal force throughout the manuscript, and these data are presented exclusively on a per-leg basis.

Maximal and submaximal rates of oxygen uptake ($\dot{V}O_{2\ peak}$, $\dot{V}O_2$ ml, O_2 kg/min). One- and two-legged $\dot{V}O_{2\ peak}$ values were determined from a progressive, discontinuous cycling test to failure. The peak rate of O_2 uptake for each condition was the highest single-minute $\dot{V}O_2$ value obtained during the progressive, discontinuous cycling test. The test consisted of a minimum of seven 5-min constant load, work bouts

on the ergometer, separated by at least 5 min of rest. Work rates were increased 10 to 30 W per workload in accordance with the estimated fitness level of the subject. The test was initiated at a power output estimated to be 30–40% of one- or two-legged $\dot{V}O_{2\,peak}$ and was terminated when the subject was unable to complete a 5-min bout. Expired air was collected during the last 2 min of each 5-min bout using Douglas bags. A sample of air from each bag was analyzed for O_2 (Applied Electrochemistry SA-3), and CO_2 (Applied Electrochemistry CD-3A) fractions after calibration with gas of known concentrations. Gas volumes were determined with a dry gas meter (Parkinson-Cowan CD4) and digital thermometer (Wescor TH-65 TC). Rates of oxygen uptake were determined from O_2 and CO_2 fractions, and the expired volumes in accordance with Consolazio et al. (12).

Sprint cycling. Sprint tests for one- and two-legged cycling were constant load trials administered at intensities between 100% and 300% of the respective $\dot{V}O_{2\,peak}$. Subjects completed 15–19 all-out trials for each cycling condition at pedal forces that were chosen to elicit failure in 15 to 400 s. Five sprints were performed per session; subjects were allowed the time necessary between sprints to fully recover. This was generally 20 min after long sprints (>180 s) and a minimum of 10 min after shorter sprint tests. Tests were initiated with a 2-s unloaded acceleration followed by the application of the frictional load. Tests were terminated when the subject could not maintain the desired cadence of 100 rpm for 5 s. Subjects were instructed to increase their cadence and allowed 3 s to do so at least once before the investigator stopped the test.

Pedal force provided anaerobically. The force supported by anaerobic metabolism was defined as the difference between the pedal force during each sprint trial (F_{ds}) and the minimum pedal force required to elicit $\dot{V}O_{2\,peak}$ i.e., F_{aer} ($F_{ds} - F_{aer}$). Anaerobic pedal force (F_{an}) values should be regarded as lower limits because they assume the maximum pedal forces that can be supported aerobically in the respective modes are available to subjects throughout the one- and two-legged sprint trials.

In accordance with prior practice (10, 58, 60), we have quantified the mechanical performance supported by anaerobic metabolism to avoid relying on uncertain estimates of the quantities of anaerobic energy released. This approach offers two advantages. First, the mechanical end products of anaerobic metabolism can be accurately measured while the anaerobic energy released cannot be (6, 7, 48). Second, the mechanical measures provide accurate predictions of individual performance capabilities (10, 58, 60) that are not available from the quantitative estimates of the anaerobic energy released during these efforts.

Electromyography. Bipolar surface electromyography (EMG) electrodes were placed on the skin of the dominant leg overlying the muscle belly of the distal portion of vastus lateralis with an interelectrode distance of 10–14 cm. The reference electrode was placed on the anterior medial aspect of the tibialis. Repeatable electrode placement was achieved by marking the location of each electrode during the first sprint session with indelible ink. Subjects were instructed to reapply the ink as necessary throughout their participation in the study. To reduce electrical impedance, the skin was lightly abraded and cleaned with rubbing alcohol before electrode placement. The surface EMG signals were amplified (1,000–5,000 times) and filtered (100–3,000 Hz half-amplitude band pass and 60 Hz notch filter) using a Grass P511 preamplifier. The analog output from the preamplifier was recorded throughout the duration of each one- and two-legged sprinting bout by a computer using the previously described A/D board with a sampling frequency of 6,000 Hz.

The recorded EMG data were rectified and integrated per contraction (iEMG). Because of the considerable variation in iEMG values that occur from one contraction to the next, we determined the rate of increase in iEMG over the course of the trial by comparing the average of the first five contractile bursts after the load was applied to the average of the last five bursts recorded during each all-out trial. Because we were interested in the increase in iEMG over the course of the all-out sprint trials rather than the magnitude of the EMG signal, we normalized the start and end iEMG averages to the five contraction average from the start of each trial. We deemed all-out trials of less than 12 s to be too short to obtain consistent within-trial increases in iEMG using our averaging technique.

Data analysis and statistics. For the purpose of comparing condition means for one- and two-legged cycling at common standardized durations, we interpolated the mechanical performance data to durations of 15, 20, 30, 45, 60, 75, 90, 120, 180, 240, and 300 (58). For each subject and condition, the measures of power output and F_{ds} were fit with an iterative procedure in accordance with the general equations provided in Weyand et al. (59).

One- and two-legged means for $\dot{V}O_{2\,peak}$, $\dot{V}O_{2\,peak}$/leg, power output, power output/leg, and F_{ds} at the aerobic limits (F_{aer}) were compared using a paired *t*-test ($\alpha < 0.05$).

RESULTS

Pedal forces at $\dot{V}O_{2\,peak}$. The mean absolute $\dot{V}O_{2\,peak}$ values measured during cycling with one and two legs were 44.4 (±5.6) and 55.9 (±8.1) ml O_2 kg/min, respectively, with corresponding power outputs of 196 (±32) and 305 (±54) W, respectively. These power outputs were achieved with the significantly greater mean pedal forces (F_{aer}) of 214 (±34) N during one-legged cycling compared with 166 (±30) N for two-legged cycling (Fig. 1). When expressed on a per-leg basis, the aerobic limits and power outputs were significantly greater for one-legged cycling. Per-leg values of $\dot{V}O_{2\,peak}$ and power output for two-legged cycling were 27.9 (±4.0) ml O_2 kg/min and 152 (±27) W. For efforts below 90% of $\dot{V}O_{2\,peak}$, the relationship between metabolic rate and power output was linear for both one- ($\dot{V}O_2 = 0.20 \cdot$power output + 6.9) and two-legged ($\dot{V}O_2 = 0.15 \cdot$power output + 10.9) cycling.

Pedal forces during sprint cycling. The power output per leg and pedal forces (F_{ds}) during the one- and two-legged all-out sprinting bouts decreased with a time course similar to that measured previously for two-legged cycle ergometry (60). The decrements in mechanical performance for a typical subject as the duration of all-out sprinting became more prolonged are illustrated in Fig. 2.

For sprints of equal durations, F_{ds} was greater during one- than two-legged cycling. The power outputs for one-legged

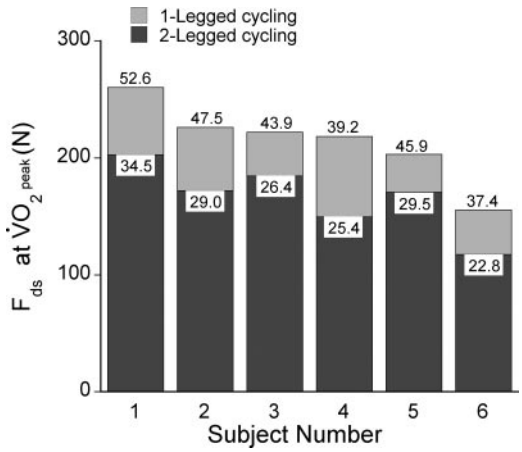

Fig. 1. Numeric values above (one leg) and within the bars (two legs) represent peak rates of oxygen uptake (ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) per leg for each subject. The minimum pedal force (F_{ds}) eliciting $\dot{V}O_{2\,peak}$ was 30 (9.4)% greater during one- vs. two-legged cycling at a cadence of 100 rpm.

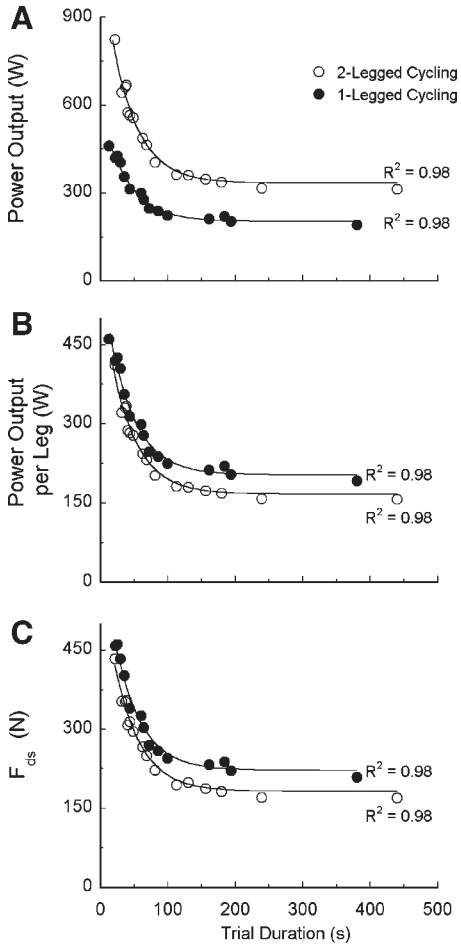

Fig. 2. The decrements in total mechanical power (*A*), per leg mechanical power (*B*) and pedal force, F_{ds} (*C*), vs. the duration of all-out constant load sprints for a representative subject during one- and two-legged cycling.

sprints longer than 120 s required pedal forces that were 24 (±6)% greater than those of a two-legged sprint of equal duration. For the shortest sprints (i.e., 15 s) the difference in F_{ds} between the two conditions was 16 (10)%.

As anticipated for both one- and two-legged sprints, our estimates of the minimum contribution to force production from anaerobic energy sources decreased with increments in all-out trial duration (Fig. 3). The anaerobic contribution to pedal force for efforts of 15 s was 57 (±7) and 61 (±5)% of the total during the one- and two-legged conditions, respectively. For longer efforts, 300 s, the anaerobic contribution to force production was only 3 (±6) and 7 (±6)% of the total force requirements for the respective conditions.

Table 1. *Trial durations and F_{an} for one- and two-legged sprints at the same pedal forces*

Pedal Force F_{ds}, N	Sprint Duration, s		Anaerobic Force, N		Percent Difference	
	One Leg	Two Legs	One Leg	Two Legs	Duration, %	F_{an}, %
225	241	80	11	59	201	−81
250	106	63	36	84	68	−57
275	80	52	61	109	54	−44
300	64	43	86	134	49	−36
400	33	20	186	234	65	−21

Sprint durations, anaerobic forces (F_{an}), and the respective percent differences [(1L-2L)/2L] for one- and two-legged sprint trial performed at equal pedal force (F_{ds}).

When considered for trials of equal F_{ds}, two-legged sprints relied more heavily on anaerobic metabolism for force production than one-legged trials (Table 1). For those one- and two-legged trials conducted at equal pedal forces, the proportion of F_{ds} generated anaerobically during the least and most forceful trials was 22 (±6) % and 11 (4)% greater, respectively, for two- vs. one-legged cycling.

EMG. EMG activity measured per contraction increased throughout the duration of each all-out trial (Fig. 4). Typically, we observed higher rates of iEMG increase (Δ iEMG/Δ time) during shorter vs. longer trials (Fig. 5). When the rate of increase in iEMG was regressed against F_{ds}, the correlation coefficients of these regressions were high; average R^2 value for one-legged cycling was 0.91 and that for two-legged cycling was 0.93. Slopes for the one- and two-legged conditions varied between individuals but were essentially parallel for five of the six subjects (Fig. 6). The x-intercept of this relationship, the largest F_{ds} with no increase in iEMG, occurred at a greater F_{ds} during one- vs. two-legged cycling for all six subjects.

DISCUSSION

Our results support the hypothesis that the impairment of muscle force production during sprint locomotion has a metabolic basis. As expected, our one- vs. two-legged comparison provided per leg differences in peak rates of aerobic metabolism and pedal forces at $\dot{V}O_{2\,peak}$ of 59 (±6) and 30 (±9)%,

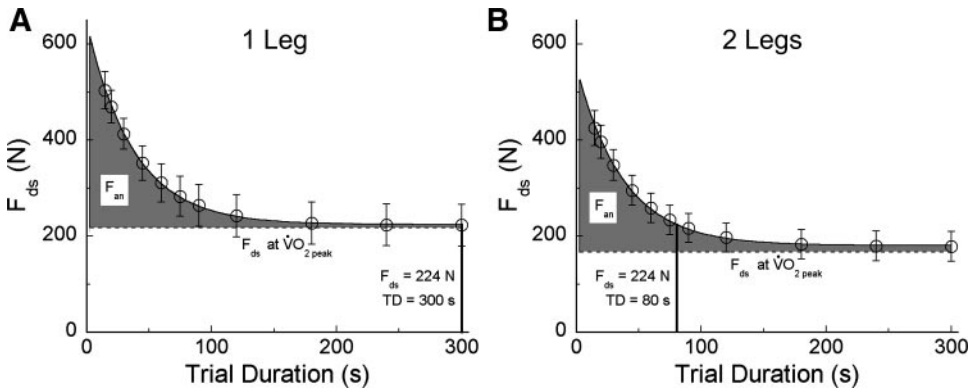

Fig. 3. The decrements in pedal force (F_{ds}) vs. the duration of all-out one- (*A*) and two-legged (*B*) sprint trials. Shaded areas represent the minimum contribution to force production provided by anerobic metabolism (F_{an}). The thick vertical line in each panel denotes sprints of equal pedal force (F_{ds} = 224 N). The greater maximum aerobic forces during one-legged cycling reduce the reliance on anaerobic metabolism, allowing the required pedal force to be sustained nearly four times as long.

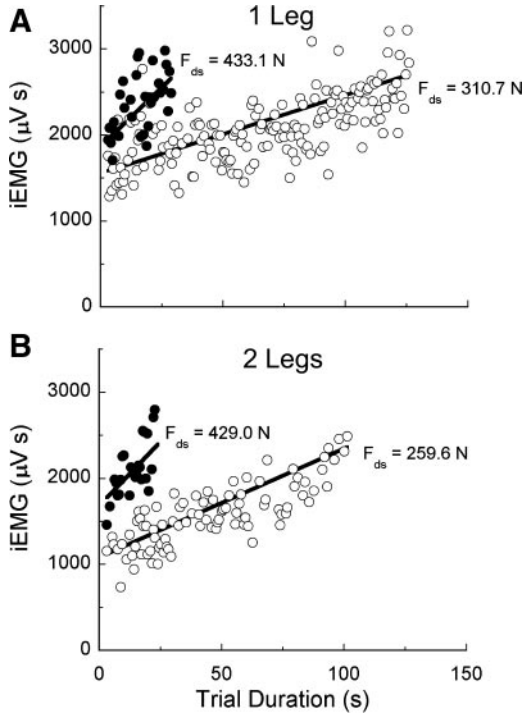

Fig. 4. Contraction-by-contraction rectified and integrated EMG (iEMG) values increased throughout the duration of an all-out sprint trial for both one- (*A*) and two-legs (*B*). The solid line joins the means of the first five contractions to that of the final five contractions for each of the sprints shown. Rates of iEMG increase were greater for trials with larger pedal force (F_{ds}) requirements and greater for two- vs. one-legged cycling at similar F_{ds}.

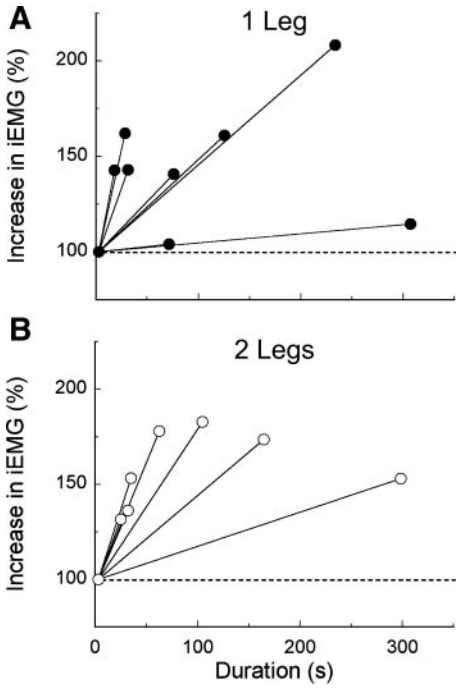

Fig. 5. Rates of iEMG increase from the first five contractions to the last five contractions during the even-numbered, all-out trials completed by subject #1 during one- (*A*) and two-legged (*B*) cycling. Rate of iEMG increase (slope of the line) were generally greater for shorter compared with longer trials.

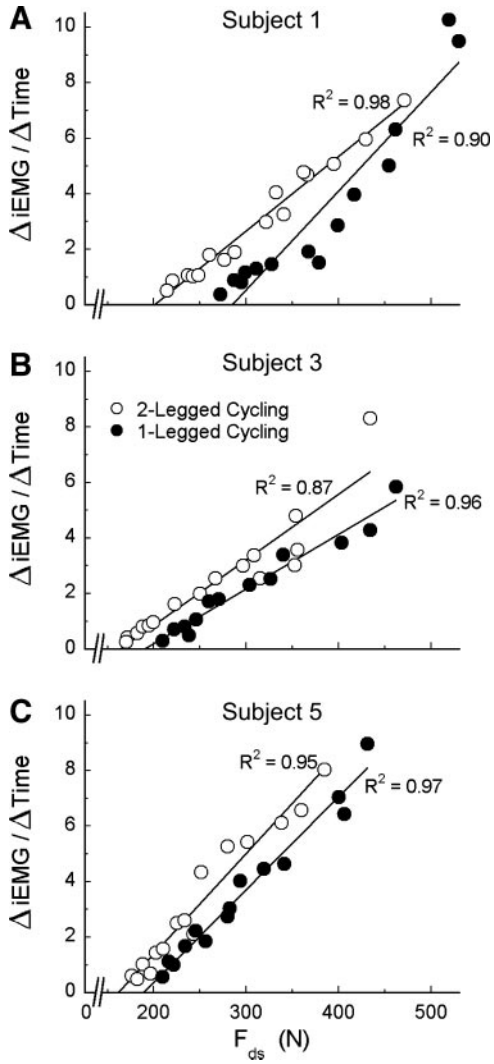

Fig. 6. Rates of iEMG increase ($\Delta\%\ s^{-1}$) in relation to the pedal forces (F_{ds}) at which one- (*A*) and two-legged (*B*) sprint trials were conducted by the odd numbered subjects. At the same F_{ds}, rates of iEMG increase were greater for the mode during which a greater proportion of the force required was provided anaerobically (i.e., two- vs. one-leg condition) (*C*).

respectively. Additionally, during both one- and two-legged constant-load sprints performed at pedal forces greater than those provided aerobically, we observed the progressive increases in neuromuscular activation that were also expected. The latter result suggests that compensatory motor unit recruitment occurred throughout each trial to provide the muscle force necessary to maintain a constant pedal force. Because of the lesser pedal forces supported aerobically during two-legged cycling, the onset of compensatory muscle recruitment occurred at lower pedal force thresholds in this mode (Fig. 6). Similarly, at equivalent pedal forces, the rates of increase in compensatory neuromuscular activity were greater during two- vs. one-legged sprint cycling. We attribute these between-mode differences in the rates at which muscle force is impaired (i.e., muscle fatigue), and neuromuscular compensation occurs to the greater reliance on anaerobic pathways of ATP resynthesis for force production during two- vs. one-legged cycling.

Muscle recruitment. Our measures of muscle recruitment were determined from positive increments in the integrated surface electromyogram. Recordings from surface EMG are primarily influenced by the volume of active muscle adjacent to the electrodes and the discharge rate of the motor neurons that innervate the active fibers. Distinguishing between these components from surface EMG is not possible, and caution has long been urged when inferring motor control strategies from these measures (34, 39). However, because of the difficulties of identifying individual motor unit action potentials during strong dynamic contractions, very little experimental evidence exists to address which motor control strategy is used (19). During isometric contractions of unfatigued limb muscles, force production is modulated by recruitment for outputs of up to 85% of the maximum level (24), and the available data suggest that the neuromuscular strategy employed during concentric contractions conforms to the same trend (19).

During fatiguing submaximal contractions, measures of EMG are known to increase throughout the effort (8, 23, 33). This response has been attributed to motor unit recruitment because motor neuron firing rates during these contractions have generally decreased or exhibited little net change (1, 8, 27). In the absence of direct measurements, we have assumed that potential changes in firing rates during fatiguing concentric contractions are similar to those that have been measured during equivalent isometric contractions. Given this assumption and the observation that the measured force outputs from the shortest trials were 83 (±2)% of the predicted maximums for sprint cycling (60), we infer that the measured increase in EMG (Fig. 4) is attributable primarily to motor unit recruitment. Regardless of whether the increase in EMG is achieved through rate coding, motor unit recruitment, or both, our primary finding would be unaffected; a reliance on anaerobic metabolism for force production during sprint efforts compromises muscular force production and triggers compensatory increases in neuromuscular activity.

For each subject, the onset of compensatory neuromuscular activity occurred at greater pedal forces during one- vs. two-legged cycling. Could the muscle force thresholds at which compensatory neuromuscular activity occurred have been the same even though pedal force thresholds were different? Two observations suggest this was not the case. First, within both individual muscles and muscle groups, both oxygen uptake and maximum aerobic force production are consistently greater when the exercise engages a lesser total mass of muscle (5, 15, 50, 53). Previous results from the many one- vs. two-legged cycle ergometry and knee-extension studies indicate greater aerobic force production occurs via higher rates of oxygen uptake in the working muscle (35, 51), rather than through the recruitment of synergist muscles. Supporting this, during one-legged knee-extensions neuromuscular activity has been found to be absent in synergistic muscle groups at work rates up to the aerobic maximum (5). Second, although we cannot exclude the possibility that synergistic muscles proximal to the knee might have been more active during one- vs. two-legged cycling, their anatomy would likely preclude meaningful contributions to the muscle forces required to extend the knee during downstroke. These observations indicate that the pedal force thresholds at which compensatory neuromuscular activity occurred were representative of differences in the quadriceps muscle forces required under the two conditions.

Force decrements and impaired muscle force production. During one- and two-legged all-out cycling trials, the pedal forces (F_{ds}) maintained for the shortest vs. longest sprints differed by 2.3 (±0.4) fold. For trials lasting between 15 and 300 s, this decrement corresponded to pedal force differences of 250 (±43) N. The rapid time course for the decrements in external force output supports the idea that the impairment of force production characteristic of muscle fatigue commences at the onset of activity (9). Our results here and elsewhere (60) suggest that duration-dependent decrements in force production are determined by the cumulative duration of the contractions involved and not by limitations in the rates at which ATP can be resynthesized from the metabolic pathways available (47, 52).

Much of the current understanding for the cellular basis of muscle fatigue stems from the rigorous work of Westerblad and colleagues (56, 57) and Allen and colleagues (3, 4). These authors have postulated that the fatiguing mechanisms they propose to be active in vitro can qualitatively account for whole-body performance loss (57). Several quantitative differences between the extent and time course of in vitro vs. in vivo rates of fatigue suggest that current cellular explanations may not generalize to whole-body performances. First, the initial loss of force that Allen and Westerblad (4) attribute to an accumulation of inorganic phosphate may be much less pronounced at physiological temperatures (14, 18). Second, the precipitation of calcium (Ca^{2+}) and inorganic phosphate (P_i) within the sarcoplasmic reticulum depends on a sequence of processes that occur with slow time courses (4, 22, 26). Therefore, this mechanism is unlikely to contribute to the impairment of force production during all-out efforts of durations less than 60 to 120 s (4). In contrast, 70% of the duration-dependent decrements we report here occur within 60 s. Third, there is no in vivo analog to the prolonged period of constant force generation between periods of force loss (3, 57) and because this period of stasis is not always observed in isolated muscle, whether this result is a product of stimulation protocol is unclear (e.g., 11, 32).

Although our results do not identify a cellular mechanism responsible for the impairment of force production during intense sequential contractions, they do provide direct experimental evidence that the onset of fatigue in vivo has a metabolic basis. Contractile inhibition by the metabolic byproducts generated through a reliance on anaerobic energy for ATP resynthesis has long been considered the most likely mechanism responsible for the decrements in force production that occur during fatigue (28, 31). However, experimental perturbations and tests at physiological temperatures have yet to definitively identify a mechanism (14, 18, 49). The results we present here indicate that a dependence on nonoxidative pathways of ATP resynthesis impairs muscle force production during locomotion.

Regardless of the mechanism, the metabolic basis for muscle force impairment and compensatory neuromuscular activity that we report here for one- and two-legged sprint cycling seems likely to be general (10, 58, 60) and to operate similarly in both more and less fit subjects. We expect that the individual force thresholds at which these phenomena are triggered will simply vary directly with the aerobic power of the individual.

Numerous investigators have suggested that there may be no single mechanism responsible for muscle fatigue. During com-

plex tasks (34, 46) and longer-duration efforts (25, 52), the mechanisms inducing failure may differ from those we describe here. However, we believe the mechanism of muscle fatigue that we identify here explains why the duration-dependent decrements in force production that occur during sprint locomotion can be described so accurately in metabolic terms (2, 10, 58, 60). During these and similar dynamic efforts, we suggest that common mechanisms of muscle fatigue are likely present at cellular, tissue, and systemic levels, although the specific mechanisms remain to be firmly established.

We conclude that impaired muscular force production and compensatory neuromuscular activity during sprint locomotion are triggered by a reliance on anaerobic metabolism for force production.

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