High-speed running performance is largely unaffected by hypoxic reductions in aerobic power

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Weyand, Peter G., Cherie S. Lee, Ricardo Martinez-Ruiz, Matthew W. Bundle, Matthew J. Bellizzi, and Seth Wright. High-speed running performance is largely unaffected by hypoxic reductions in aerobic power. *J. Appl. Physiol.* 86(6): 2059–2064, 1999.—We tested the importance of aerobic metabolism to human running speed directly by altering inspired oxygen concentrations and comparing the maximal speeds attained at different rates of oxygen uptake. Under both normoxic (20.93% O_2) and hypoxic (13.00% O_2) conditions, four fit adult men completed 15 all-out sprints lasting from 15 to 180 s as well as progressive, discontinuous treadmill tests to determine maximal oxygen uptake and the metabolic cost of steady-state running. Maximal aerobic power was lower by 30% (1.00 ± 0.15 vs. 0.77 ± 0.12 ml $O_2 \cdot kg^{-1} \cdot s^{-1}$) and sprinting rates of oxygen uptake by 12–25% under hypoxic vs. normoxic conditions while the metabolic cost of submaximal running was the same. Despite reductions in the aerobic energy available for sprinting under hypoxic conditions, our subjects were able to run just as fast for sprints of up to 60 s and nearly as fast for sprints of up to 120 s. This was possible because rates of anaerobic energy release, estimated from oxygen deficits, increased by as much as 18%, and thus compensated for the reductions in aerobic power. We conclude that maximal metabolic power outputs during sprinting are not limited by rates of anaerobic metabolism and that human speed is largely independent of aerobic power during all-out runs of 60 s or less.

sprinting; locomotion; oxygen deficit; anaerobic metabolism

THE SUSTAINABLE RUNNING SPEEDS of humans and other terrestrial mammals depend on the rate at which oxygen can be taken up from the environment to provide metabolic energy (4, 20). Because oxygen provides nearly all the energy for any run longer than several minutes, swifter endurance runners must have higher rates of oxygen uptake ($\dot{V}O_2$) to support their higher running speeds. For example, pronghorn antelope sustain speeds of nearly 20 m/s by taking up oxygen at an unusually high rate of 9.5 l/min, whereas similarly sized goats with only one-fifth the aerobic power can sustain running speeds only one-fifth as fast (13). The same relationship holds for running humans; lower maximal rates of oxygen uptake correspond directly to slower running speeds for any all-out effort lasting longer than several minutes, regardless of whether the source of lesser aerobic power is the reduced availability of environmental oxygen (24) or simply a lower fitness level (22).

In contrast to the direct relationship between speed and rates of oxygen uptake during sustained running, maximal speed for runs of only a few seconds is completely independent of oxygen drawn from the environment. The metabolic power required for these bursts is believed to be provided almost completely by anaerobic sources of muscular energy. The poor correlation between sprinting performance and aerobic power in humans (21, 22) and the relatively low aerobic power of mammalian sprinters (20) support the general view that sprinting speeds are closely linked to anaerobic power. However, during all-out runs longer than a few seconds and shorter than a few minutes, rates of oxygen uptake increase rapidly at the onset of running and remain high throughout the run (11, 17). Although the total metabolic energy, and also the relative contribution of aerobic metabolism, under these conditions can only be approximated, oxygen drawn from the environment clearly contributes an appreciable portion and, perhaps, the majority of the metabolic energy powering intermediate and long sprints (11). The absence of a relationship between aerobic power and speed for these runs is puzzling, given the magnitude of the metabolic energy environmental oxygen provides.

In this study, we reduced the oxygen content of the air inspired during brief all-out running to answer a simple question: is human sprinting performance impaired when the environmental oxygen and aerobic energy yields are limited? Because rates of oxygen uptake are high during sprint running (11, 17), and humans have very little oxygen stored in the body (1), we predicted that moderate hypoxia (13.00% O_2) would reduce maximal human running speeds during all-out runs lasting 25 s or longer.

MATERIALS AND METHODS

Subjects

Four healthy men, 22–36 yr of age (mass, 75.7 ± 9.2 kg; height, 180.4 ± 2.9 cm) volunteered and provided written informed consent before their participation. Three subjects had eight or more years of competitive running experience and were training at the time of the study. All four subjects were accustomed to vigorous exercise and had considerable experience in sprinting to exhaustion on the treadmill. Sample size was limited due to subject compliance requirements, necessary treadmill running experience, number of sessions required, and the need for a physician's presence during all hypoxic tests.

Protocol

Each subject participated in eight sessions: four hypoxic and four normoxic. In the first session under each condition, subjects completed a progressive, discontinuous treadmill test to determine maximal oxygen uptake ($\dot{V}_{O_2 max}$) and the metabolic cost of running at 2.0 m/s. During each of the three remaining sessions for each condition, four to six sprints were performed at speeds selected to elicit exhaustion between 15 and 180 s. Hypoxic and normoxic sessions were interspersed and conducted two to three times per week over a 5-wk period, with a minimum respite of 48 h. Treadmill speeds were chosen so that three sprint trials were ultimately completed within each of the following time intervals under both conditions: 14–22, 22–35, 35–60, 60–100, and 100–180 s. Generally, subjects completed one sprint in each time interval during each session. Subjects were instructed to take the time necessary for full recovery between sprints and to discontinue testing if they felt unable to perform fully. Postsprint recovery times were typically 20 min after sprints exceeding 75 s and 10 min after sprints lasting <75 s.

All running was performed at a treadmill inclination of 4.6° (7.9% grade) to keep the treadmill speeds manageable for even the fastest sprints. To ensure safety, an upper-body harness, adjusted to suspend subjects above the treadmill in the event of a fall, was worn for all sprints, and a physician and resuscitation equipment were present for hypoxic sessions. Each sprint was initiated when the subject used the handrails to make the transition from standing while straddling the moving belt to running without any handrail assistance. This was accomplished within 2 s and four steps. Subjects were instructed to terminate the sprint trial only when their inability to maintain the belt speed caused them to begin to move backward on the treadmill. At this time, they were to grab the handrails and restraddle the belt while the treadmill was stopped.

For the hypoxic sessions, dry gas was drawn from a pressurized cylinder through a large-bore polyethylene tubing into a 300-liter meteorological buffer balloon. Inspired air was drawn from the buffer balloon through a corrugated tube via a one-way respiratory valve, which subsequently directed expired air into a series of 120-liter meteorological balloons. Subjects were acclimated to the hypoxic gas for 3 min before each sprint trial while they were standing quietly on the treadmill, during which time arterial oxygen saturation, determined from pulse oximetry (Vet/Ox model SC14402), stabilized at a mean value of 87.0 ± 4.0%. For the normoxic sprints, a buffer balloon was not used; ambient air was inspired directly through the one-way valve.

Measurements

$\dot{V}_{O_2 max}$ *(ml $O_2 \cdot kg^{-1} \cdot s^{-1}$)*. $\dot{V}_{O_2 max}$ was defined as the highest single minute $\dot{V}_{O_2}$ during a progressive, discontinuous treadmill test conducted at an inclination of 4.6° and consisting of 5-min bouts of running, separated by 3-min rest intervals. Tests progressed by increments of 0.15 to 0.2 m/s from an initial speed of 2.0 m/s for the normoxic test and a speed eliciting 60% of normoxic $\dot{V}_{O_2 max}$ for the hypoxic test. Each test continued until the subject was unable to complete a 5-min bout. Expired air was collected in meteorological balloons during the fourth and fifth minute of running at each speed. From each of the bags, aliquots of 375 ml were drawn, dried, and analyzed for O_2 (Ametek SA II O_2 analyzer) and CO_2 fractions (Ametek CD-3A CO_2 analyzer) immediately after analyzer calibration with gas of a known concentration. Gas volumes were determined from a dry-gas meter (Parkinson-Cowan) with simultaneous temperature determination.

Rates of oxygen uptake ($\dot{V}_{O_2}$, STPD) were calculated in accordance with Consolazio et al. (3).

Metabolic cost of submaximal running (ml $O_2 \cdot kg^{-1} \cdot s^{-1}$). Running economy was determined from the rates of $\dot{V}_{O_2}$ during the last 2 min of a 5-min run at 2.0 m/s, completed by all subjects under both conditions.

Sprinting rates of aerobic metabolism (ml $O_2 \cdot kg^{-1} \cdot s^{-1}$). Rates of oxygen uptake, averaged over the duration of each sprint, were determined from the expired air collected in serial meteorological balloons. Collection intervals were 30 s for the first and second bags and 60 s for subsequent bags. Neither total bag number nor collection time in the final bag was known before the sprint trial, but both were determined by the time at which the subject grabbed the handrails to end the run. To ensure that the air expired during the sprint was fully collected and isolated in the balloons, a 2-s pause was taken after the start of each trial before the first bag was open to allow the last gas expired before the sprint to move past the first bag, and a 1-s pause was taken after the end of the trial before the final bag was closed to capture the last air expired during the run.

Sprinting metabolic rates (ml O_2 eq $\cdot kg^{-1} \cdot s^{-1}$). Total metabolic rates during sprinting ($\dot{E}_{tot}$) were estimated by extrapolating the linear relationship between $\dot{V}_{O_2}$ and submaximal running speed to the speed of the respective sprint trial (17). Linear regressions for individual subjects were formulated from a minimum of five, and generally seven, steady-state measurements of $\dot{V}_{O_2}$ without assigning a Y-intercept value. Rates of anaerobic metabolism ($\dot{E}_{an}$) were estimated from oxygen deficits by subtracting the rate of oxygen taken up during the run from the estimated $\dot{E}_{tot}$ (17).

Statistics

Normoxic and hypoxic means for $\dot{V}_{O_2 max}$ and the metabolic cost of submaximal running were compared by using paired t-tests ($P < 0.05$). Sprinting speeds, rates of $\dot{V}_{O_2}$, and rates of anaerobic energy release under normoxic and hypoxic conditions were compared for sprints of 15, 30, 45, 60, 75, 90, 120, 150, and 180 s, also using paired t-tests ($P < 0.05$). Because it was not possible to control the time of sprints to exhaustion to within a single second by using treadmill speed, values at these times were interpolated for each subject to obtain group means for statistical comparisons. Original speed and $\dot{V}_{O_2}$, data as a function of sprint duration for one subject appear in Fig. 1.

Curve-Fitting Procedures

Interpolated values for speed, $\dot{V}_{O_2}$, and $\dot{E}_{an}$ for each subject at the nine standardized sprint durations were determined from equations formulated for each subject by using an iterative best-fit procedure (Kaleidagraph 3.0.1) and the following equation forms

$$\text{Speed} = m_1 + m_2 \cdot e^{(-m_3 \cdot t)} \tag{1}$$

where m_1 is the velocity at $\dot{V}_{O_2 max}$, m_2 is velocity measured for the shortest sprint minus the velocity at $\dot{V}_{O_2 max}$, e is the base of the natural logarithm, and m_3 is the coefficient of the exponent describing the decrease in speed with increases in sprint duration (t).

$$\dot{V}_{O_2} = m_1 + m_2 - m_2 \cdot e^{(-m_3 \cdot t)} \tag{2}$$

where m_1 is presprint $\dot{V}_{O_2}$, m_2 is $\dot{V}_{O_2 max}$ minus presprint $\dot{V}_{O_2}$, and m_3 is the coefficient of the exponent describing the increase in $\dot{V}_{O_2}$ with increases in t.

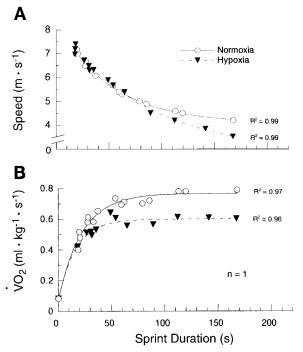$$\dot{E}_{an} = m_1 + m_2 \cdot e^{(-m_3 \cdot t)} \tag{3}$$

A

B

Fig. 1. All-out running speeds (*A*) and average rates of oxygen uptake ($\dot{V}_{O_2}$; *B*) of a representative subject during sprints of different durations under normoxic (○) and hypoxic (▲) conditions.

where m_1 is the minimum value for $\dot{E}_{an}$ measured for the longest sprint, m_2 is the rate of $\dot{E}_{an}$ measured for the shortest sprints, and m_3 is the coefficient of the exponent describing the decrease in $\dot{E}_{an}$ with increases in *t*.

Similar equations describing these three variables as a function of sprint duration have been used previously (6, 7, 23). Their physiological basis has been discussed at length by Wilkie (23). The range of R^2 values for individual curve fits (between 0.95 and 0.99 for all 24 curves; 4 subjects × 3 variables × 2 conditions) appear in the figure legends for the respective variables.

RESULTS

$\dot{V}_{O_{2max}}$

Maximal aerobic power was 30% lower under hypoxic than under normoxic conditions. Mean values were 0.77 ± 0.12 and 1.00 ± 0.15 ml $O_2 \cdot kg^{-1} \cdot s^{-1}$, respectively (Fig. 2).

Metabolic Cost of Submaximal Running

Steady-state $\dot{V}_{O_2}$ at 2.0 m/s under normoxic and hypoxic conditions were 0.57 ± 0.01 and 0.56 ± 0.02 ml·kg^{-1}·s^{-1}, respectively, and were not significantly different (Fig. 2). The oxygen cost of running per meter traveled, provided by the slope of the speed-$\dot{V}_{O_2}$ relationship in Fig. 2, was 0.26 ml·kg^{-1}·m^{-1}.

Sprinting Speed

Speeds under hypoxic conditions equaled those under normoxic conditions for sprints from 15 to 60 s, but were significantly slower for sprints from 75 to 180 s. Under both conditions, speed decreased rapidly with increases in sprint duration from 15 to 60 s, falling from

7.0 ± 0.2 to 5.5 ± 0.4 m/s under normoxic conditions and from 7.0 ± 0.2 to 5.4 ± 0.4 m/s under hypoxic conditions (Fig. 3*A*). For sprints of 75 s and longer, the difference in speed between hypoxic and normoxic conditions increased with increasing sprint duration. For sprints of 75 s, mean speeds under hypoxic conditions were only 0.2 m/s slower, whereas for sprints of 180 s they were 0.7 m/s slower. Speeds for the shortest sprints exceeded those for the longest sprints by 1.6- and 1.8-fold under normoxic and hypoxic conditions, respectively.

Rates of Oxygen Uptake During Sprinting

Time-averaged rates of oxygen uptake for individual sprint trials increased with sprint duration under both conditions, from ~50% of the respective $\dot{V}_{O_{2max}}$ values at 15 s to roughly 70% at 30 s, with further gradual increases toward condition-specific maxima with additional increases in sprint duration (Fig. 3*B*). Mean sprinting $\dot{V}_{O_2}$ values under hypoxic conditions were 12 and 18% lower than normoxic values for sprints of 15 and 30 s, respectively, and 23–25% lower for sprints of 45 s or longer. Across sprint durations and conditions, the proportions of the total metabolic energy provided by environmental oxygen ranged from 20 to 90%.

$\dot{E}_{tot}$ and $\dot{E}_{an}$ During Sprinting

$\dot{E}_{tot}$ was assumed to be proportional to running speed, and, therefore, did not differ between hypoxic and normoxic conditions for sprints of up to 60 s but was lower under hypoxic conditions for sprint durations >75 s. $\dot{E}_{tot}$ values ranged from maximum values 1.8 times normoxic $\dot{V}_{O_{2max}}$ for the 15-s sprints under both conditions to minimum values 1.03 times greater for 180-s sprints under hypoxic conditions. Rates of anaero-
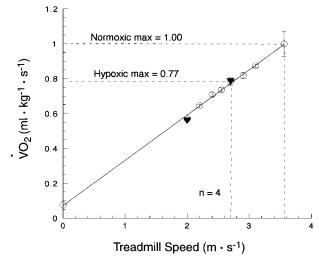

Fig. 2. $\dot{V}_{O_2}$ increased linearly with running speed on a 4.6° incline under normoxic conditions (○; $\dot{V}_{O_2} = 0.076 + 0.26 \cdot$ speed; $R^2 = 0.99$). Maximal rates of $\dot{V}_{O_2}$ (dashed lines), were significantly lower under hypoxic conditions (uppermost ▼) than under normoxic conditions (uppermost ○). Steady-state rates of $\dot{V}_{O_2}$ at 2.0 m/s were not different under hypoxic vs. normoxic conditions (lowermost ▼ and ○, respectively). All values, maximal and submaximal, are means ± SE for 4 subjects.
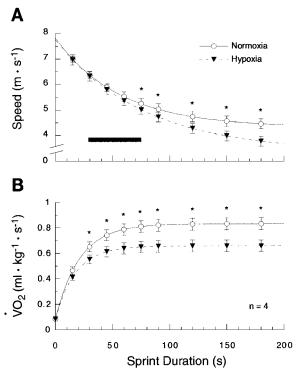
Fig. 3. All-out running speeds (*A*) were the same under hypoxic and normoxic conditions for sprints lasting 60 s or less, but were significantly slower under hypoxic conditions for sprints lasting 75 s or longer. Average rates of $\dot{V}o_2$ (*B*) were significantly lower during hypoxic than during normoxic sprints of 30 s or longer. Bar in *A* marks range of sprint durations for which $\dot{V}o_2$ differed and speed did not ($Speed_n = 4.31 + 3.49 \cdot e^{-0.018t}$; $Speed_h = 3.35 + 4.39 \cdot e^{-0.013t}$; Speed R^2 range = 0.98 − 0.99; $\dot{V}o_{2-n} = 0.08 + 0.75 − 0.75 \cdot e^{-0.057t}$; $\dot{V}o_{2-h} = 0.08 + 0.58 − 0.58 \cdot e^{-0.047t}$; $\dot{V}o_2$ R^2 range = 0.95 − 0.99, where subscripts n and h stand for normoxia and hypoxia, respectively, and *t* is time). Values are means ± SE for 4 subjects. *Significant differences between conditions.

bic energy release $\dot{E}_{an}$ were greater under hypoxic than under normoxic conditions for sprints of all durations, except the shortest and longest sprints of 15 and 180 s, respectively (Fig. 4*A*). Differences in $\dot{E}_{an}$ were greatest for sprints of intermediate duration, being as much as 16–18% greater for hypoxic than for normoxic sprints of 60, 75, and 90 s. Under both conditions, $\dot{E}_{an}$ was nearly four times greater for the shortest (15-s) than for the longest (180-s) sprints.

DISCUSSION

We set out to determine whether human sprinting performance is impaired when environmental oxygen and aerobic energy yields are limited, and we expected that maximal speeds would be decreased under hypoxic conditions for sprints lasting 25 s or longer. Despite reducing the aerobic power of our athletic subjects to the level of sedentary humans and markedly decreasing their rates of oxygen uptake during sprinting, we found they could run just as fast for sprints of up to 60 s and nearly as fast for sprints of up to 120 s. Clearly, maximal human running speeds for short- and intermediate-length sprints are relatively unaffected by large reductions in aerobic power. Because the metabolic cost of sprinting under hypoxic and normoxic conditions

was probably the same, achieving these speeds under hypoxic conditions would not have been possible without additional metabolic energy from anaerobic sources. Rates of anaerobic metabolism increased sufficiently to fully compensate for the aerobic energy lost during hypoxic sprints of up to 60 s and to partially compensate for that lost during hypoxic sprints of up to 150 s. Previously, experimentation indicated that rates of anaerobic metabolism are elevated under hypoxic conditions during submaximal efforts (12, 14), and performance data from Olympic track competition at modest altitude suggested that mild hypoxia did not affect sprinting speeds. Here, we demonstrate directly that sprinting performance is largely unaffected by more severe reductions in oxygen availability, and we identify compensatory increases in rates of anaerobic energy release as a mechanism for maintaining performance.

Our conclusions of increased anaerobic power during hypoxic sprinting rest on several assumptions. We estimated total metabolic rates during sprinting using a controversial extrapolation of the linear relationship between metabolic rate and speed during submaximal running (17). Although the accuracy of these estimates is questionable (9, 18), our experimental design did not require us to know the actual metabolic cost of sprinting. As long as our assumption that this cost was the
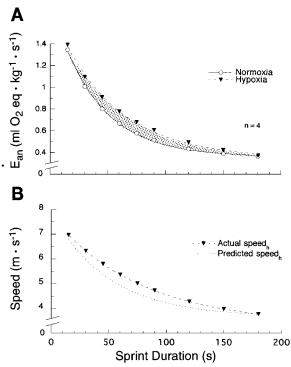

Fig. 4. Rates of anaerobic energy release ($\dot{E}_{an}$; *A*) estimated from oxygen deficits were greater under hypoxic (▼) than under normoxic conditions (○) for sprints of all durations, except 15- and 180-s sprints. Consequently, the actual sprinting speeds (*B*) under hypoxic conditions (▼ and dashed line) exceeded speeds predicted from decreased $\dot{E}_{an}$ (dotted line) for all but the shortest and longest sprints. ($\dot{E}_{an-n} = 0.35 + 1.37 \cdot e^{-0.019t}$; $\dot{E}_{an-h} = 0.37 + 1.43 \cdot e^{-0.026t}$; R^2 range = 0.97–0.99). Values are means ± SE for 4 subjects (on average 6.3 and 4.5% of $\dot{E}_{an-n}$ and $\dot{E}_{an-h}$ values, respectively) and are not shown for clarity.

same under the two conditions was correct, possible errors resulting from extrapolated estimates would have affected both sprint conditions equally. Thus our conclusions are not affected by the accuracy of these estimates. Our direct measurements of the metabolic cost of running below $\dot{V}_{O_{2\max}}$ support the assumption that the energy cost of running under the two conditions was the same. Nonetheless, we cannot exclude the possibility that metabolic rates during high-speed running under hypoxic conditions might have been higher. The increased ventilation, cardiac work, and possible muscle fatigue induced by low-oxygen conditions could have elevated energetic requirements and caused us to underestimate the total metabolic energy expended during hypoxic sprinting. However, any such error, if present, would cause us to underestimate the additional anaerobic energy released under hypoxic conditions and thus not alter our findings.

Additionally, we interpreted non-steady-state measurements of $\dot{V}_{O_2}$ at the mouth as rates of aerobic metabolism, as is practiced widely (9, 11, 17, 18, 22). During sprinting, these measurements underrepresent rates of aerobic metabolism because they do not include oxygen stores present in blood and muscle that are utilized for aerobic energy at the onset of running. However, the size of these oxygen stores would have limited their contribution to an average of 5% or less of the total metabolic power required for the sprints in question. Additionally, the small reduction in these stores under hypoxic conditions would result in rates of $\dot{V}_{O_2}$ at the mouth being closer to the actual rates of oxygen utilization under hypoxic than under normoxic conditions and would, therefore, slightly reduce hypoxic oxygen deficits. Thus an adjustment of oxygen deficit estimates of the anaerobic energy released for oxygen stores under these conditions would also increase our estimates of the additional anaerobic energy released during hypoxic sprinting. Because of the conservative nature of our assumptions and measurements, our estimates of the additional anaerobic energy released while the subjects were sprinting under hypoxic conditions should be regarded as lower limits; we believe that the actual amounts are probably slightly higher.

Our results explain the curiously poor relationship between aerobic power and performance during sprints that rely heavily on aerobic metabolism that puzzled us initially (11, 21, 22). The ability of rates of anaerobic energy release to increase to compensate for the metabolic energy not provided aerobically prevents speed capabilities from being compromised by lesser aerobic power. Consequently, it is not surprising that aerobic power does not correlate with sprinting performance (7, 15, 21, 22), despite contributions from aerobic metabolism that can be relatively large (11). The flexibility in rates of anaerobic energy release suggests that anaerobic metabolism may function to allow the energetic requirements of the active muscles to be fully met when oxygen availability varies. In this respect, we believe this flexibility of anaerobic energy release results in the functional importance of this source of metabolic energy exceeding its fractional contributions.

Our results also highlight a means by which animals that do little or no sustained running can minimize their energetic overhead. Relatively greater rates of anaerobic metabolism during locomotion in animals with low aerobic power would eliminate the energetic expense of maintaining the additional respiratory and cardiovascular structure necessary to provide the metabolic energy to sprint aerobically. Although a greater reliance on anaerobic metabolism can incur subsequent energetic costs (8, 10), the transient costs resulting for sprint specialists and relatively sedentary individuals would likely be smaller than the constant energetic requirement of maintaining the additional structure necessary to provide the same energy aerobically. This possibility is supported by available evidence, suggesting that sprinters rely more heavily on anaerobic metabolism during brief runs (11, 20) and also have a greater ability to release compensatory anaerobic energy when faced with reductions in aerobic power. The relative increases in rates of anaerobic energy release under hypoxic conditions in our swiftest sprinter, a middle-distance runner, who could cover 800 m in 1 min and 51 s, were 1.7 times greater than the average for the other three subjects. These observations suggest a functional role for anaerobic metabolism supplemental to the immediate provision of metabolic energy, and prompt us to propose that anaerobic and aerobic power are set at levels that provide functional locomotor needs for the minimum total energetic cost.

Our results also pose a challenge to metabolic explanations for the decreases in maximal running speed that occur with increases in the duration of all-out runs. These speed decreases are generally attributed to parallel decreases in the maximal rates at which anaerobic sources can resynthesize ATP during runs of different duration in question (7, 19). Our results indicate that rates of anaerobic ATP resynthesis are not truly maximal during normoxic sprint running. Therefore, decreases in maximal running speeds that occur with increases in sprint time cannot be explained simply by concomitant decreases in maximal rates of anaerobic metabolism. Rather, our results imply that the maximal metabolic rates under these circumstances are set by the rates of ATP hydrolysis at the cross-bridge level, and that matching resynthesis rates are provided through flexible rates of aerobic and anaerobic metabolism. Thus the mechanical performance of the muscular system is somewhat independent of the source of the ATP resynthesized to provide metabolic energy under these circumstances. In this regard, our results provide empirical support for theoretical models, suggesting that cross-bridge cycling rates dictate total metabolic rates during brief all-out efforts (15, 16), and highlight the incomplete understanding of the factors that determine the apparent decreases in these cycling rates with increases in run duration.

A better understanding of these factors and of the source of the additional anaerobic energy released

under hypoxic conditions could be gained with further measurements and the use of existing empirical models. DiPrampero and colleagues (2, 5, 6) have accurately predicted human running performance by estimating energy yields from cellular sources of ATP resynthesis by using whole body measurements, including blood lactate. Our data do not indicate whether the source of additional ATP resynthesized anaerobically during hypoxic sprinting is high-energy phosphate stores, glycolysis, or both. Measurements of blood lactate concentrations after sprinting under hypoxic conditions might allow the source of the additional anaerobic energy to be identified. Any differences in blood lactate concentrations under hypoxic and normoxic conditions could be quantitatively related to metabolic power outputs and performance by using the diPrampero model. The proposed measurements would provide an independent test of the model and could advance the general understanding of how metabolic sources of energy are related to the performance of the musculoskeletal system during brief all-out efforts.

We conclude that maximal metabolic power outputs during sprinting are not limited by rates of anaerobic metabolism, and that human running speed is largely independent of aerobic power during all-out sprints lasting <1 min.

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