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Determinants of metabolic cost during submaximal cycling

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McDaniel, J., J. L. Durstine, G. A. Hand, and J. C. Martin. Determinants of metabolic cost during submaximal cycling. *J Appl Physiol* 93: 823–828, 2002. First published May 3, 2002; 10.1152/japplphysiol.00982.2001.—The metabolic cost of producing submaximal cycling power has been reported to vary with pedaling rate. Pedaling rate, however, governs two physiological phenomena known to influence metabolic cost and efficiency: muscle shortening velocity and the frequency of muscle activation and relaxation. The purpose of this investigation was to determine the relative influence of those two phenomena on metabolic cost during submaximal cycling. Nine trained male cyclists performed submaximal cycling at power outputs intended to elicit 30, 60, and 90% of their individual lactate threshold at four pedaling rates (40, 60, 80, 100 rpm) with three different crank lengths (145, 170, and 195 mm). The combination of four pedaling rates and three crank lengths produced 12 pedal speeds ranging from 0.61 to 2.04 m/s. Metabolic cost was determined by indirect calorimetery, and power output and pedaling rate were recorded. A stepwise multiple linear regression procedure selected mechanical power output, pedal speed, and pedal speed squared as the main determinants of metabolic cost ($R^2 = 0.99 \pm 0.01$). Neither pedaling rate nor crank length significantly contributed to the regression model. The cost of unloaded cycling and delta efficiency were 150 metabolic watts and 24.7%, respectively, when data from all crank lengths and pedal speeds were included in a regression. Those values increased with increasing pedal speed and ranged from a low of 73 ± 7 metabolic watts and 22.1 ± 0.3% (145-mm cranks, 40 rpm) to a high of 297 ± 23 metabolic watts and 26.6 ± 0.7% (195-mm cranks, 100 rpm). These results suggest that mechanical power output and pedal speed, a marker for muscle shortening velocity, are the main determinants of metabolic cost during submaximal cycling, whereas pedaling rate (i.e., activation-relaxation rate) does not significantly contribute to metabolic cost.

muscle metabolism; cycling efficiency; crank length; pedaling rate

PREVIOUS INVESTIGATORS HAVE reported that cycling efficiency and metabolic cost vary with pedaling rate (5, 11, 24, 44). The observed variation in metabolic cost and efficiency during cycling at different pedaling rates has been attributed to differences in muscle shortening velocity (5, 17, 24, 31). Pedaling rate, however, governs two distinct physiological phenomena: the frequency of muscle activation and relaxation, and muscle shortening velocity. Pedaling rate per se determines the rate at which muscles must become excited and subsequently relax and thus influences the metabolic cost associated with active calcium uptake (28). Pedal speed, the product of pedaling rate and cycle crank length, governs muscle shortening velocity (33, 51), which has been reported to alter metabolic efficiency (28) and metabolic cost (1, 20, 29, 30, 41, 42). Thus, by varying pedaling rate alone, the metabolic cost associated with excitation-relaxation rate cannot be differentiated from that associated with muscle shortening velocity.

Recently, Martin et al. (33, 35) used an experimental paradigm in which both pedaling rate and cycle crank length were varied. That experimental paradigm produced several pedal speeds (one for each crank length) for any specific pedaling rate. They reported that maximal muscular power did not differ when cycling with crank lengths of 145, 170, and 195 mm, suggesting that muscular function was unaffected within that range of cycle crank lengths. Thus, by using a range of crank lengths, pedaling rate and pedal speed can be decoupled without compromising muscular function. Therefore, the purpose of this investigation was to determine the separate contributions of pedaling rate and pedal speed to the metabolic cost of producing submaximal cycling power and to test the hypothesis that increases in pedaling rate or pedal speed would independently contribute to an increase in metabolic cost.

METHODS

Nine trained cyclists (32.8 ± 6.7 yr, 80.0 ± 12.9 kg) volunteered to participate in this study. The protocol and data collection methods were thoroughly explained, and the subjects signed a statement of informed consent. This investigation was reviewed and approved by the Internal Review Board of the University of South Carolina.

Participants reported to the laboratory on five separate occasions. During the initial visit, lactate threshold (LT) and peak oxygen consumption ($\dot{V}O_{2\,peak}$) were determined. LT was determined during a 25-min protocol in which subjects cycled at intensities intended to elicit 50, 60, 70, 80, and 90% of their estimated $\dot{V}O_{2\,peak}$ while pedaling at 100 rpm. Expired gas volume flow rate and concentrations, heart rate, and mechanical power output were recorded throughout the

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Table 1. *Pedaling rate, crank length, and pedal speed*

	Pedaling Rates, rpm			
Crank Length, mm	40	60	80	100
145	0.61	0.91	1.21	1.52
170	0.71	1.07	1.42	1.78
195	0.82	1.23	1.63	2.04

Values are in m/s. The combination of 3 crank lengths and 4 pedaling rates produced 12 pedal speeds ranging from 0.61 to 2.04 m/s.

protocol. Expired gas volume flow rate and concentrations were analyzed with an electrochemistry (Sunnyvale, CA) 9CD-3A CO_2 analyzer, S-3A O_2 analyzer, and a Vacumetrics (Ventura, CA) airflow meter. All analyzers were interfaced with a computer for the calculation of oxygen consumption ($\dot{V}O_2$) and respiratory exchange ratio (RER). Gas analyzers were calibrated before and immediately after every data collection period by using room air and a calibration gas of known concentration (14.99% O_2, 4.99% CO_2; Holox, Norcross, GA). Mechanical power output, heart rate, and pedaling rate were recorded by a Schoberer Rad Messtechnik power meter (Konigskamp, Germany) mounted on a Monark cycle ergometer that has been shown to provide valid measurements of mechanical power (26, 34). Blood was drawn during the 5th min of each stage through a catheter placed in the antecubital vein. Lactate concentrations were determined by using Sigma Diagnostics lactate assay procedure no. 826-UV. Blood samples were deproteinized with 8% perchloric acid and later analyzed for lactate concentration by using an enzymatic technique (19). LT was defined as the intensity at which plasma lactate concentration increased to 1 mmol above baseline (10). After a recovery period of ~15 min, subjects performed a $\dot{V}O_{2\ peak}$ test. During the $\dot{V}O_{2\ peak}$ test, subjects cycled at 100 rpm while power was increased each minute until volitional fatigue (8–11 min). $\dot{V}O_2$ and RER were calculated at 15-s intervals, and $\dot{V}O_{2\ peak}$ was calculated as the average of the highest two consecutive $\dot{V}O_2$ measurements.

During the second laboratory visit, subjects performed familiarization sessions with the 145- and 195-mm crank lengths. Subjects cycled at a power output intended to elicit 60% of LT for 20 min with each crank. During each 20-min familiarization session, subjects cycled for 5 min at pedaling rates of 40, 60, 80, and 100 rpm. Familiarization trials were not performed with the 170-mm crank length because that length was equivalent to the length used on their own bicycles and thus required no additional familiarization. Finally, subjects performed three 3-s maximum power tests using the inertial load method (36).

Experimental data were recorded during the remaining three laboratory visits. After an 8-h fast, subjects performed the data collection protocol with one of three crank lengths (presented in random order). Pedaling rates (40, 60, 80, and 100 rpm) were also presented in random order. For each pedaling rate, subjects cycled for 15 min during which power was increased every 5 min (30, 60, and 90% of their LT). After each pedaling rate, subjects rested for 2-min before resuming exercise at the next assigned pedaling rate. To minimize the metabolic cost of torso stabilization (especially during low pedaling rate and high intensity), a restraining bar was attached to the back of the seat, which acted to restrict horizontal movement. Subjects were instructed not to grip the handlebars tightly to maintain their position on the seat. Rather, they were instructed to relax their arms and let the restraining bar counteract horizontal forces. The combination of four pedaling rates and three crank lengths used in this protocol produced 12 pedal speeds ranging from 0.6 to 2.04 m/s [pedal speed (m/s) = crank length (m) × pedaling rate (rpm) × 2 π/60; Table 1].

Throughout the experimental protocol, $\dot{V}O_2$ and RER were recorded every minute, and data were corrected for analyzer drift if necessary (4 of the 27 trials). Measurements from the 4th and 5th min of each stage were used in data analysis. Metabolic cost was calculated by using the regression equation of Zuntz (52) based on the thermal equivalent of O_2 for nonprotein respiratory equivalent: metabolic cost (kcal/min) = $\dot{V}O_2 \times (1.2341 \times RER + 3.8124)$. Metabolic cost was also calculated in units of metabolic watts via the conversion factor 69.7 $W \cdot kcal^{-1} \cdot min^{-1}$.

A stepwise multiple linear regression procedure was used to determine which independent variables (mechanical power, crank length, pedaling rate, and pedal speed) were most predictive of metabolic cost. Second-order terms were also included to allow for the possibility that the relationships might be curvilinear. After each variable selection by the stepwise procedure, the regression model residuals were plotted against the remaining independent variables to allow observation of the effects of those remaining variables. Delta efficiency (8, 11, 24) and cost of unloaded cycling (9) were determined from the linear regression of mechanical power vs. metabolic cost data for each crank length and pedaling rate combination. Delta efficiency was calculated as the inverse of the slope of the regression line, and cost of unloaded cycling was determined as the intercept of that regression line.

RESULTS

The $\dot{V}O_{2\ peak}$ and LT of subjects in this investigation were 66 ± 7 $ml \cdot kg^{-1} \cdot min^{-1}$ and 69 ± 8% $\dot{V}O_{2\ peak}$, respectively (means ± SD). The power output that elicited LT was 229 ± 26 W. The first independent variable selected by the stepwise linear regression procedure was mechanical power output ($R^2 = 0.95$; Fig. 1). The residuals of that regression model were curvilinearly related to pedal speed (Fig. 2*A*; $R^2 = 0.55$, $P < 0.0001$), pedaling rate (Fig. 2*B*; $R^2 = 0.41$, $P < 0.0001$), and crank length (Fig. 2*C*; $R^2 = 0.06$, $P < 0.0001$). The next variables selected were pedal speed squared ($P < 0.0001$) and pedal speed ($P < 0.0001$). Those three

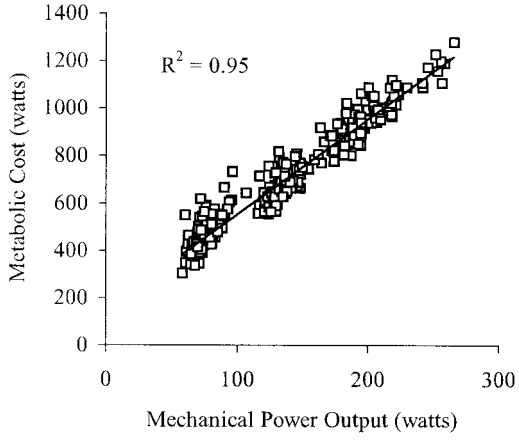

Fig. 1. Metabolic cost as a function of mechanical power. Mechanical power accounted for 95% of the variability in metabolic cost across the range of pedaling rates, pedal speeds, and crank lengths tested.

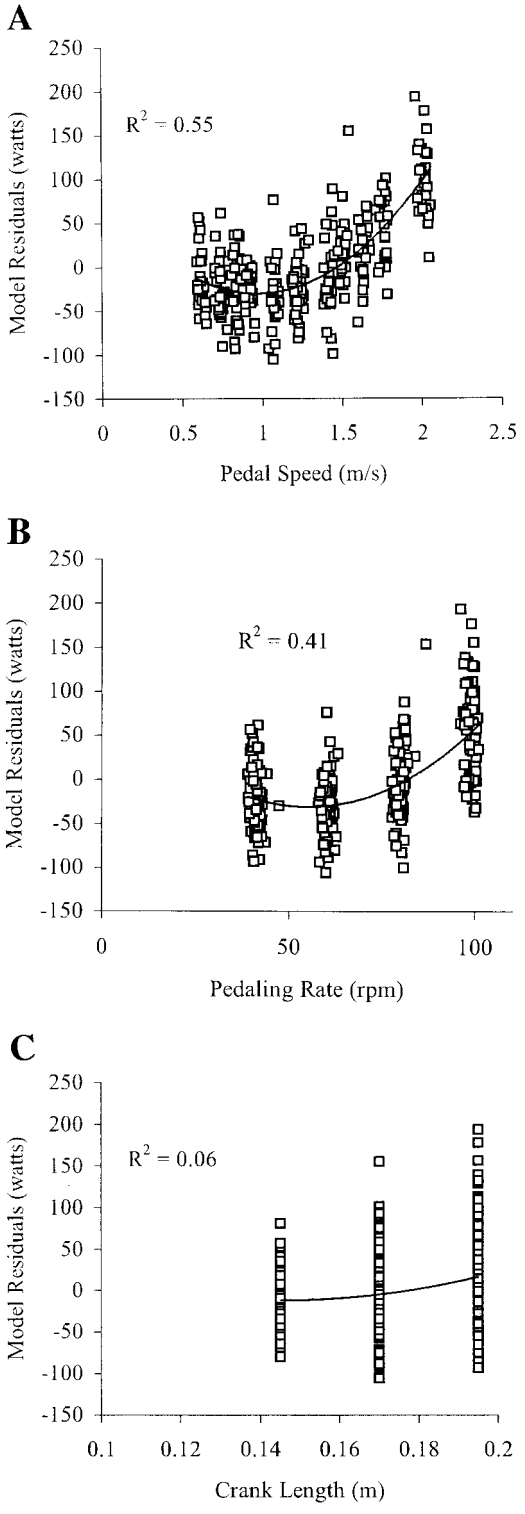

Fig. 2. Residuals of the mechanical power vs. metabolic cost regression model. Residuals were significantly related to pedal speed (*A*), pedaling rate (*B*), and crank length (*C*).

variables accounted for 98% of the total variability of metabolic cost of all nine subjects (Fig. 3). The residuals of that model were independent of pedaling rate ($R^2 = 0.007$, $P = 0.66$) and crank length ($R^2 = 0.006$, $P = 0.54$). Neither pedaling rate nor crank length was

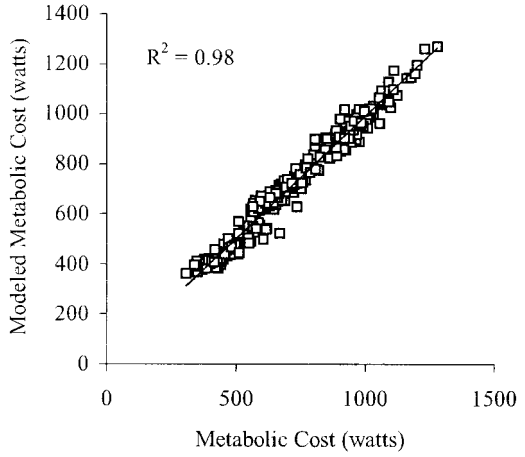

Fig. 3. Metabolic cost as a function of mechanical power and pedal speed. The regression model for metabolic cost as a function of mechanical power, pedal speed, and pedal speed squared accounted for 98% of the variability in metabolic cost of all subjects.

subsequently selected by the stepwise procedure. When the power and pedal speed regression model was applied to each subject's individual data, the coefficient of determination was 0.99 ± 0.01 (means ± SE). Delta efficiency and the cost of unloaded cycling tended to increase with increasing pedaling rate, crank length, and pedal speed but were most clearly related to pedal speed (Fig. 4). When data from all subjects and all treatments were analyzed, the costs of unloaded cycling and delta efficiency were 150 metabolic watts and 24.7%, respectively. When data from each treatment were analyzed (Fig. 4), those values ranged from a low of 73 ± 7 metabolic watts and 22.1 ± 0.3% (145-mm cranks, 40 rpm) to a high of 297 ± 23 metabolic watts and 26.6 ± 0.7% (195-mm cranks, 100 rpm). Maximum cycling power, recorded during the 3-s inertial load power test, was 1,178 ± 37 W (means ± SE), and thus the power outputs that represented 30, 60, and 90% of LT also represented 6, 12, and 18% of the subjects' maximum cycling power, respectively. $\dot{V}O_2$ was stable during the final 2 min of the 90% of LT stages (Fig. 5).

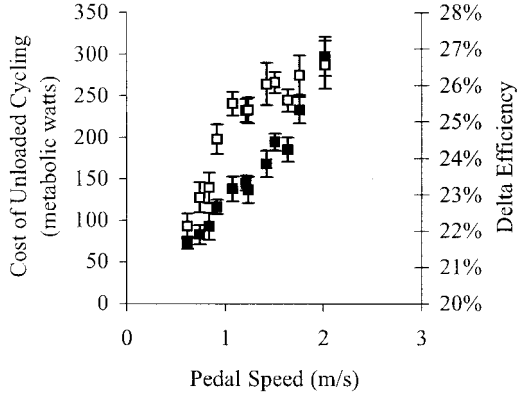

Fig. 4. Delta efficiency (□) and cost of unloaded cycling (■). Delta efficiency and cost of unloaded cycling tended to increase with increasing pedal speed. Values are means ± SE.

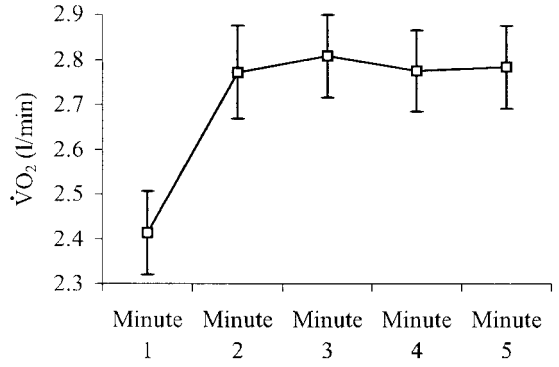

Fig. 5. Oxygen uptake ($\dot{V}O_2$) was stable during the final 2 min of the 5-min stages at 90% of lactate threshold, suggesting that data within each stage were not confounded by a slow component of $\dot{V}O_2$. Values are means ± SE.

DISCUSSION

The main finding of this investigation was that mechanical power output and pedal speed accounted for 99% of the variation in metabolic cost at intensities below LT. When the regression model was applied to each individual subject's data, metabolic cost could be predicted with a standard error of 26 metabolic watts or roughly the equivalent of 0.08 l/min $\dot{V}O_2$. Mechanical power output alone accounted for 95% of the variation in metabolic cost (Fig. 1), suggesting that, even with our wide range of pedaling rates, pedal speeds, and crank lengths, muscles' ability to convert chemical energy to mechanical work was remarkably stable.

Pedal speed vs. pedaling rate. Previous investigators have reported $\dot{V}O_2$ to be curvilinearly related to pedaling rate (5, 7, 32, 47). Our data agreed with those previous reports but also indicated that metabolic cost was more closely related to pedal speed, a surrogate measure for muscle shortening velocity (33, 51). Thus pedal speed or muscle shortening velocity was responsible for the majority of the variability in the conversion of metabolic energy to mechanical power (i.e., differences in metabolic cost or efficiency). Pedal speed probably influences metabolic cost through a combination of physiological, biomechanical, and/or neuromuscular phenomena. The primary physiological phenomena is most likely the increased myosin ATPase activity associated with increased muscle fiber shortening velocity (20, 29, 30, 40, 41). That is, because one ATP is required for each cross-bridge cycle, the rate of ATP hydrolysis is partially dependent on muscle shortening velocity (21, 50). Additionally, because pedal speed governs the rate at which muscle fibers shorten, it will influence metabolic efficiency via the efficiency-velocity relationship of the active fibers (20, 29). Pedal speed may also influence metabolic cost via fiber-type recruitment. Specifically, power is the product of force and velocity, and, if pedal speed is altered, pedal force must be inversely altered to maintain any specific mechanical power output. Thus an increase in pedal speed will require an increase in muscle shortening velocity and a decrease in muscular force. The requirement for increased shortening velocity may elicit greater recruitment of fast-twitch fibers (14), whereas the decreased force production may allow for greater reliance on slow-twitch fibers (43). The concomitant effects of pedal speed on muscular force and shortening velocity make it difficult to predict how pedal speed will affect muscle fiber-type recruitment. Indeed, two previous investigators have reported pedaling rate to have no effect on fiber-type recruitment patterns across a wide range of pedaling rates (2, 18). Consequently, the extent to which fiber-type recruitment may alter metabolic cost remains unclear.

Biomechanical properties of muscle tissue and limb segments may also contribute to the observed variation in metabolic cost. First, viscous losses in muscle tissue (12) can be mathematically modeled with a linear damper (15). For such a damper, force is proportional to shortening velocity, and thus power lost to muscle viscosity is proportional to the square of shortening velocity. Thus viscous loss in muscle tissue might explain the curvilinear relationship that we observed between metabolic cost and pedal speed. Additionally, internal work (49), the muscular work required to accelerate the limb segments, is not included in our measure of cycling power and may influence metabolic cost (48). Indeed, Ferguson et al. (13) recently reported that internal power accounted for a substantial portion of total power during repetitive leg extension. Although it is well established that internal work is lost during gait and open-chain activities (4, 13, 49), the role of internal work during cycling remains controversial. Some investigators have reported that internal work was lost during cycling (48), whereas others have reported that internal work was recaptured at some later point in the pedal cycle (16, 25, 27). Consequently, the contribution of internal work to the metabolic cost of cycling remains unclear. Finally, negative joint work has been reported to increase with increasing pedaling rate (37, 38). That increase has been attributed to incomplete relaxation of the muscles (38) but could also be related to increased viscous losses associated with higher muscle lengthening velocity. The most plausible explanations for the observed relationship of metabolic cost with pedal speed are myosin ATPase activity, muscular efficiency, viscous losses in muscle tissue, and incomplete muscular relaxation. Other possible factors include internal work and fiber-type recruitment patterns.

Pedaling rate per se did not significantly contribute to metabolic cost, and thus these data do not fully support our hypothesis. We hypothesized that pedaling rate would significantly contribute to metabolic cost based on reports by previous investigators that the metabolic cost of activation and relaxation accounted for 30–40% of total metabolic cost (3, 22, 23, 45, 46). Those investigators used protocols with anaerobic conditions (3, 45), electrical stimulation (3, 22, 45, 46), isometric contractions (3, 22, 45), and/or nonphysiological conditions (23, 46). In contrast, our cycling protocol used voluntary cycling at power outputs that represented only 6, 12, and 18% of our subjects' maximal muscular power measured by the 3-s inertial load

test. Those relatively low-power outputs may have required recruitment of fewer and/or lower threshold motor units (6, 43), and thus the cost of activation and relaxation may have been reduced compared with previous protocols. Additionally, myosin ATPase activity has been reported to increase by up to 2.7-fold with increasing shortening velocity compared with isometric contraction (40, 41). Thus, even if calcium ATPase activity remained constant, the relative cost of that activity would decrease from ~30–40 to ~8–11%. The present data suggest that the low power (reduced motor unit recruitment) and muscle shortening in our protocol combined to reduce the metabolic cost associated with activation and relaxation to a nonsignificant portion of the total cost.

Cost of unloaded cycling vs. delta efficiency. Our statistical analysis was designed to determine the relationship of metabolic cost with mechanical power output, pedaling rate, and pedal speed. However, many previous investigators have analyzed the intercept and slope of the metabolic cost (or $\dot{V}O_2$) vs. mechanical power output regression line. The intercept has been termed the cost of unloaded cycling and is thought to represent the cost of moving the limbs (44). The inverse of the slope has been termed delta efficiency and is thought to represent the metabolic cost of producing mechanical power (17, 44). The cost of unloaded cycling tended to increase with increasing pedal speed (Fig. 4) and ranged from a low of 73 ± 7 metabolic watts for the lowest pedal speed to 297 ± 23 metabolic watts for the highest pedal speed. For reasons discussed above, we believe the most likely explanations for that increase to be increased ATPase activity, increased viscous losses in muscle tissue (during shortening and lengthening), and incomplete muscle relaxation, but internal work may also contribute. Delta efficiency also increased with increasing pedal speed from a low of 22.1 ± 0.3% for the lowest pedal speed to a high of 26.6 ± 0.7% for the highest pedal speed. That increase in delta efficiency is an intriguing aspect of this and previous investigations and most likely results from muscle fibers shortening closer to their optimal, or most efficient, velocity (9). If that is the mechanism, then there will be a pedal speed beyond which delta efficiency decreases. To our knowledge, no such point has been reported, but the determination of that point would be an interesting area for future research.

Because both the cost of unloaded cycling and delta efficiency contribute to metabolic cost, gross efficiency (power output/metabolic cost) is a function of power output (7). At low power output, metabolic cost is strongly influenced by the cost of unloaded cycling, and lower pedal speeds provide greater gross efficiency. For example, at a power output of 50 W, our subjects' gross efficiency was greatest (16.7%) at the lowest pedal speed (0.61 m/s). As power output is increased, delta efficiency becomes increasingly deterministic of metabolic cost. If our subjects were able to produce 400 W aerobically (e.g., elite cyclists), their gross efficiency would have been greatest, 23.5%, at a pedal speed of 1.4 m/s.

Validity of pulmonary $\dot{V}O_2$. We used indirect calorimetery to assess the metabolic cost of producing mechanical power, which has been reported to provide a valid indication of $\dot{V}O_2$ by the working muscles (39). Even so, we were aware that $\dot{V}O_2$ drift during the 66-min protocol, $\dot{V}O_2$ slow component within the 5-min steady-state periods, or the cost of torso stabilization might compromise that validity. Our experimental protocol required 66 min of intermittent exercise and $\dot{V}O_2$ drift, or changes in substrate metabolism might have influenced metabolic cost during that prolonged testing period. Therefore, we assessed the effect of $\dot{V}O_2$ drift on metabolic cost during our pilot testing. Experiments with three subjects demonstrated that metabolic cost varied by <1%, despite increases in $\dot{V}O_2$ and decreases in RER. This suggests that a substrate shift from carbohydrate to fat occurred in such a way that metabolic cost remained essentially stable. As shown in Fig. 5, metabolic cost was stable during the final 2 min of the 5-min stages at 90% of LT, suggesting that our data were not confounded by a slow component of $\dot{V}O_2$. Finally, our range of pedal speeds and power outputs might have affected the metabolic cost of torso stabilization, and thus whole body $\dot{V}O_2$ might not have accurately reflected $\dot{V}O_2$ by the legs. The restraining bar allowed subjects to relax their arms and torso and yet remain stable. Thus, by using intensities below LT and a restraining bar, our metabolic cost data were not biased by $\dot{V}O_2$ drift, $\dot{V}O_2$ slow component, or stabilization costs.

In summary, the present data indicate that mechanical power output and pedal speed, a marker for muscle shortening velocity, accounted for 99% of metabolic cost during submaximal cycling. Pedal speed most likely contributed to metabolic cost via changes in myosin ATPase activity, viscous losses in muscle tissue, incomplete muscular relaxation, and muscular efficiency. Other possible contributory factors include internal work and fiber-type recruitment patterns. Pedaling rate per se did not significantly alter metabolic cost, suggesting that the metabolic cost associated with calcium handling may be insignificantly affected by contraction rate during submaximal cycling.

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