Skeletal muscle ATP turnover and muscle fiber conduction velocity are elevated at higher muscle temperatures during maximal power output development in humans

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Gray, Stuart R., Giuseppe De Vito, Myra A. Nimmo, Dario Farina, and Richard A. Ferguson. Skeletal muscle ATP turnover and muscle fiber conduction velocity are elevated at higher muscle temperatures during maximal power output development in humans. Am J Physiol Regul Integr Comp Physiol 290: R376–R382, 2006. First published September 15, 2005; doi:10.1152/ajpregu.00291.2005.—The effect of temperature on skeletal muscle ATP turnover and muscle fiber conduction velocity (MFCV) was studied during maximal power output development in humans. Eight male subjects performed a 6-s maximal sprint on a mechanically braked cycle ergometer. Muscle temperature was passively elevated through the combination of hot water immersion and electric blankets. Anaerobic ATP turnover was calculated from analysis of muscle biopsies obtained before and immediately after exercise. MFCV was measured during exercise using surface electromyography. Preexercise muscle temperature was 34.2°C (SD 0.6) in N and 37.5°C (SD 0.6) in ET. During ET, the rate of ATP turnover for phosphocreatine utilization [temperature coefficient (Q10) = 3.8], glycolysis (Q10 = 1.7), and total anaerobic ATP turnover [Q10 = 2.7; 10.8 (SD 1.9) vs. 14.6 mmol·kg−1·(dry mass)·s−1 (SD 2.3)] were greater than during N (P < 0.05). MFCV was also greater in ET than in N [3.79 (SD 0.47) to 5.55 m/s (SD 0.72)]. Maximal power output (Q10 = 2.2) and pedal rate (Q10 = 1.6) were greater in ET compared with N (P < 0.05). The Q10 of maximal and mean power were correlated (P < 0.05; R = 0.82 and 0.85, respectively) with the percentage of myosin heavy chain type IIA. The greater power output achieved with passive heating was achieved through an elevated rate of anaerobic ATP turnover and MFCV, possibly due to a greater effect of temperature on power production of fibers, with a predominance of myosin heavy chain IIA at the contraction frequencies reached.

cycling exercise; temperature coefficient; myosin heavy chain; surface electromyography; energy production

TEMPERATURE IS AN IMPORTANT determinant of skeletal muscle contractile and metabolic properties (6, 37, 38). One major effect of elevated muscle temperature (ET) is to alter both the force/velocity and power/velocity relationships, which have been demonstrated both in mammals (38) and humans (12, 24). Thus a passive elevation of muscle temperature (Tm) results in an improved performance in terms of both work (3) and power output in humans during cycling (41). Because the temperature-dependent contractile properties are a function of myofibrillar ATPase activity (24), which is itself temperature dependent (48), it is possible that an increased ATP turnover would contribute to the greater maximal power output under ET conditions. Indeed, a greater rate of ATP turnover at high Tm has been reported during sustained submaximal isometric contractions (14) and intense sustained dynamic exercise (18).

Neuromuscular factors may also have an effect on performance and ATP turnover. Muscle fiber conduction velocity (MFCV), for instance, provides important information in relation to the muscle fiber membrane and contractile properties (2). MFCV is the average value of conduction velocities of the motor units active during a contraction and thus reflects motor control strategies (1). A greater MFCV under ET conditions may lead to individual sarcomeres being more rapidly activated, meaning that the contractile speed of the whole fiber would be enhanced. Associated with this would be a greater rate of ATP turnover. It has been previously demonstrated that MFCV is elevated with increased Tm during low-force isometric contractions of the tibialis anterior muscle (15). Moreover, the increase in conduction velocity in single motor units is correlated to the increase in motor unit twitch force and rate of force development (15). It may, therefore, be possible that the temperature-dependent increase in power output is caused by more rapid muscle activation and thus MFCV.

The main purpose of the present study, therefore, was to determine whether a passive increase in Tm altered ATP turnover and MFCV during the development of maximal power output during cycling. It was hypothesized that increasing Tm would lead to an increase in maximal power output due to increases in anaerobic ATP turnover, primarily from phosphocreatine (PCr) utilization and glycogenolysis, and MFCV. Furthermore, it has previously been demonstrated that the temperature-dependent increase in power output is fiber-type specific, with type I fibers being most sensitive to an increase in temperature (44). Therefore, a further hypothesis was that the magnitude of increase in power with temperature would be dependent on the percentage of myosin heavy chain (MHC) type I in the exercising muscle.

METHODS

Subjects

Eight healthy male subjects [age 25 yr (SD 6), height 1.82 m (SD 0.07), mass 77 kg (SD 11); means (SD)], with no history of muscle or metabolic disorders, volunteered for the study. Two of these subjects performed the experimental protocol but gave only one biopsy for MHC determination and are thus not included in any of the results or discussion of ATP turnover and MFCV. All subjects were habitually
physically active, but none was specifically trained. The subjects were fully informed of the purposes, risks, and discomfort associated with the experiment before providing written, informed consent. This study conformed to current local guidelines and the Declaration of Helsinki and was approved by the University of Strathclyde Ethics Committee.

**Experimental Protocol**

Subjects arrived at the laboratory, on two separate occasions, following a 3-h fast and having abstained from any caffeine, alcohol, or strenuous exercise 48 h before each trial. They were also asked to record their dietary intake for 48 h before the first trial and replicate this before the second trial. Subjects inserted a rectal thermistor probe (Grants Instruments) ~10 cm beyond the anal sphincter to allow continuous monitoring of rectal temperature. Following this, in the control condition of normal Tm (N), subjects rested for 30 min at normal room temperature (20–22°C), while the muscle biopsy site was prepared and a flexible Tm probe was inserted. At the same time, an electrode array for electromyography (EMG) signal recording and goniometer were positioned on the contralateral leg for measurement of MFCV. In the ET condition, Tm was first increased by immersing the legs up to the gluteal fold in a water bath maintained at 42.8°C (SD 1.4) for 30 min. The subjects exited the water bath, briefly toweled dry, changed shorts, and put on their shoes before moving to the examination couch for preparation of the biopsy sites, insertion of the Tm probe, and attachment of goniometer and electrode array. During this period, when possible, subjects lay with their legs wrapped in electrically heated blankets. The subjects then lay at rest, with heated blankets in position, until a Tm of ~37–37.5°C was reached. After temperature manipulation or the 30-min rest at room temperature (Tm of ~34°C), a resting muscle sample was taken. The subjects then mounted the cycle ergometer and performed a 6-s maximal sprint from a stationary position, immediately after which another muscle sample was obtained with the subject remaining on the ergometer. The order of the experimental trials was randomly assigned and took place at the same time of day, with a minimum of 14 days between trials. Before experimental trials, the subjects were fully habituated to the sprint cycle, practicing a minimum of three times.

**Measurements**

**Power output.** The sprint exercise was performed on a mechanically braked cycle ergometer (Ergomedic 824E, Monark, Verburg, Sweden), with the load set at 7.5% body mass. Power output was calculated every second from the known frictional load and the measurement of flywheel velocity and corrected for the acceleration of the flywheel (32). Maximal external power output, mean external power output, maximal pedal rate, and mean pedal rate were calculated for the 6-s sprint.

**Tm.** A flexible Tm probe (Ellab, Copenhagen, Denmark) was inserted to a depth of 3 cm in the medial portion of the vastus lateralis muscle. The thermistor was inserted through a flexible venaflow cannula (18 G, Becton Dickinson, Dublin, UK) and advanced ~0.5 cm beyond the end of the cannula into the muscle.

**Muscle biopsies.** Muscle samples were taken from the medial part of the vastus lateralis under local anesthesia (1% Xylocaine) by using the needle biopsy technique (7). Samples were immediately (<10 s) frozen in liquid nitrogen and stored at −80°C for subsequent analysis. Contralateral legs were used for sampling in each of the control and experimental trials, the order of which was counterbalanced.

The freeze-dried samples were dissected free of blood and connective tissue, powdered, and prepared for metabolite analysis. Metabolites were extracted in a solution of 0.5 M perchloric acid and 1 mM EDTA, neutralized to pH 7.0 with 2.2 M KHCO₃, and stored at −80°C. PCR and ATP content were determined, in duplicate, spectrophotometrically (23), and lactate content was determined, also in duplicate, fluorometrically (33). Glycogen was extracted in 1 M HCl and determined, in duplicate, fluorometrically (33).

**MHC.** The MHC content of the samples was determined by polyacrylamide gel electrophoresis after denaturing in sodium dodecyl sulfate (SDS-PAGE) using a method derived from Fauteck and Kandarian (17). Electrophoresis (Bio-Rad Mini-Protein) was carried out on 6% (cross-linking 2.7%) polyacrylamide resolving gels with 4% (cross-linking 2.7%) stacking gels, which were electrophoresed at ~8°C for 16 h at a constant 100 V. Protein bands were visualized by silver staining, using a method modified from Ref. 35 and quantified by densitometry (Bio-Rad GS8000 calibrated densitometer), with each band expressed as a percentage of the total MHC content of the corresponding lane. MHC isoforms were identified according to migration rates compared with molecular weight standards and characterized as types I, IIA, and IIX.

**MFCV.** Multichannel surface EMG (sEMG) signals were detected from the vastus lateralis of the nonbiopsy leg with a linear adhesive array (model EL-SCH008, SPES Medica, Salerno, Italy) consisting of eight electrodes with 5-mm interelectrode distance, in bipolar configuration. The electrodes were positioned as described previously (16). The sEMG signals were amplified (16-channel sEMG amplifier, EMG-16, LISIN, Prima Biomedical and Sport, Treviso, Italy), band pass-filtered (~3–30 Hz bandpass, 10–500 Hz, 40-dB decade), and sampled at 2 kHz. Average MFCV was estimated from the multichannel sEMG signals during the cycling exercise. Off-line analysis of sEMG signals was performed, as described in detail previously (16). For each subject, the same channels were used for estimation of MFCV in both N and ET, avoiding any bias in the comparison of MFCV in the two conditions.

**Calculations**

Anaerobic ATP turnover was calculated as ΔPCr + 1.5 Δmuscle lactate + 2 ΔATP (46), where A is change. The small quantity of ATP produced, or utilized, relating to the accumulation of glycolytic metabolites (e.g., pyruvate and glyceraldehyde-3-phosphate) is neglected, as they represent <2% of the rate of ATP turnover in all cases (47). Furthermore, this calculation relies on the assumption that, during a 6-s maximal sprint, the amount of lactate efflux is not great (5), and there is minimal uptake of glucose from outside of the cell (29). It is also assumed that thigh oxygen uptake is minimal during the 6-s exercise (4) and that there is no difference in thigh oxygen uptake as a result of passive heating (Ferguson RA, Krustrup P, Kjaer M, Mohr M, Ball D, and Bangsbo J., unpublished data).

The temperature dependence of measured variables is presented as temperature coefficients (Q10), calculated as follows (6):

\[ Q_{10} = \left( \frac{R_2}{R_1} \right)^{\left( \frac{10}{T_2 - T_1} \right)} \]

where \( R_2 \) and \( R_1 \) are rate processes, respectively, at temperatures \( T_2 \) and \( T_1 \), with \( T_2 \) being greater than \( T_1 \). \( Q_{10} > 1 \) indicates a positive thermal dependence; \( Q_{10} = 1 \) indicates a thermal independence; and \( Q_{10} < 1 \) indicates a negative thermal dependence.

**Statistical Analysis**

Data were analyzed by either two-way (temperature and time) ANOVA with repeated measures or paired t-tests. Where a significant effect was detected, differences were located with post hoc paired t-tests with Bonferroni correction. Pearson correlation coefficient (R) was computed to assess linear relations between variables. Significance was accepted at \( P < 0.05 \). Data are presented as means ± SD.

**RESULTS**

The heating protocol resulted in a higher (\( P < 0.05 \)) Tm before the onset of exercise [37.5°C (SD 0.6) in ET vs. 34.2°C (SD 0.6) in N]. Rectal temperature was also higher following the heating protocol [37.2°C (SD 0.2) in ET vs. 37.1°C (SD 0.2) in N; \( P < 0.05 \)].
Table 1. Muscle metabolites before and after the 6-s maximal sprint under conditions of normal and elevated muscle temperature

<table>
<thead>
<tr>
<th></th>
<th>Preexercise</th>
<th>Postexercise</th>
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<tbody>
<tr>
<td>PCR N</td>
<td>74.1 (5.7)</td>
<td>41.7 (6.1)*</td>
</tr>
<tr>
<td>PCR ET</td>
<td>76.9 (4.3)</td>
<td>29.2 (9.3)*</td>
</tr>
<tr>
<td>Lactate N</td>
<td>5.3 (0.8)</td>
<td>22.9 (4.1)*</td>
</tr>
<tr>
<td>Lactate ET</td>
<td>5.6 (1.0)</td>
<td>24.9 (5.1)*</td>
</tr>
<tr>
<td>ATP N</td>
<td>23.3 (1.7)</td>
<td>19.6 (1.4)*</td>
</tr>
<tr>
<td>ATP ET</td>
<td>23.9 (1.9)</td>
<td>18.5 (1.9)*</td>
</tr>
<tr>
<td>Glycogen N</td>
<td>483 (154)</td>
<td>447 (161)*</td>
</tr>
<tr>
<td>Glycogen ET</td>
<td>490 (167)</td>
<td>440 (182)*</td>
</tr>
</tbody>
</table>

Values are means (SD) in mmol/kg [dry mass (dm)] except for glycogen, which is in mmol glycosyl units/kg (dm); n = 6. PCR, phosphocreatine; N, normal muscle temperature; ET, elevated muscle temperature. *Significant difference between pre- and postexercise and between conditions, †P < 0.05.

Passive elevation of $T_m$ did not affect the resting metabolite content but resulted in a greater ($P < 0.05$) fall in PCR content and increase in lactate content (Table 1) during the 6-s sprint. Elevating $T_m$ lead to an increase in both MFCV [3.79 (SD 0.47) vs. 5.55 m/s (SD 0.72) in N and ET conditions, respectively; $Q_{10}$ of 3.8; $P < 0.05$] and ATP turnover (Table 2) compared with N, the latter by 35%. This represents a $Q_{10}$ value of 2.7 for ATP turnover. The $Q_{10}$ values for ATP resynthesis from PCR hydrolysis and glycolysis were 3.8 and 1.7, respectively. There was a close association ($P < 0.05$) between MFCV and ATP turnover under both temperature conditions (Fig. 1).

Despite the greater rate of ATP turnover and the overall increase in both maximal and mean power output ($Q_{10}$ of 2.2 and 1.7, respectively) being significant (Table 3), not all subjects demonstrated an increase in power output with heating (Fig. 2). For example, subjects 1, 2, 3, 6, 7, and 8 demonstrated an increase in maximal power output by an average of 30% (~10% per 1°C increase in $T_m$), whereas the power output of subjects 4 and 5 did not increase substantially. This is illustrated by examining the relationship between $Q_{10}$ values for ATP turnover and mean power output (Fig. 3), although there are no data from subjects 7 and 8. All data points are above the line of identity, demonstrating that the magnitude of increase in ATP turnover was greater than the increase in power output in all cases.

The MHC composition- [type I 32% (9), type IIA 53% (SD 6), and type IIX 15% (SD 9)]-dependent effect of $T_m$ on power output is demonstrated in Fig. 4, where the magnitude of increase in both mean and peak external power with ET was positively correlated with the percentage of MHC IIA ($P < 0.05$), whereas there is no such relationship with percentage of MHC I.

Table 2. Rates of anaerobic ATP turnover during 6-s maximal sprint exercise under conditions of normal and elevated muscle temperature

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>ET</th>
</tr>
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<tbody>
<tr>
<td>PCR ATP turnover</td>
<td>5.4 (0.5)</td>
<td>7.9 (1.3)*</td>
</tr>
<tr>
<td>Glycolytic ATP turnover</td>
<td>4.1 (1.0)</td>
<td>4.8 (1.2)*</td>
</tr>
<tr>
<td>Anaerobic ATP turnover</td>
<td>10.8 (1.9)</td>
<td>14.6 (2.3)*</td>
</tr>
</tbody>
</table>

Values are means (SD) in mmol·kg$^{-1}$·s$^{-1}$; n = 6. *Significant difference between conditions, $P < 0.05$.

Fig. 1. Relationship between muscle fiber conduction velocity (MFCV) and total ATP turnover under conditions of normal (○) and elevated (□) muscle temperature. dm, Dry mass. *Significant correlation ($P < 0.05$).

DISCUSSION

The present investigation has demonstrated that passively elevating the temperature of the exercising muscle before the performance of a maximal sprint increases the rate of ATP turnover and MFCV during the exercise. Furthermore, the magnitude of increase in power output that occurred as a result of the passive heating was correlated with the relative content of MHC IIA isoform.

The increase in ATP turnover with heating reflects an elevated rate of cross-bridge cycling and would occur because, as with other enzymatic processes, myofibrillar ATPase activity is temperature dependent (24, 48). This effect was previously shown during isometric contractions (14) and intense dynamic exercise (18). It might have been expected that, during a maximal sprint in which maximal power output is generated, ATP turnover would be at its highest level. Rates of ATP turnover during short-term friction-loaded cycle ergometry under nonheated control conditions have been recorded previously to be 6.0 mmol·kg$^{-1}$·s$^{-1}$ [dry mass (dm)] for PCR utilization and 4.8 mmol·kg$^{-1}$·s$^{-1}$ (dm) for glycolysis, leading to lactate formation (8). These values are similar to those obtained during N in the present study, although other studies have reported higher values (21, 36). On the contrary, the highest rate of human skeletal muscle ATP turnover from the individual metabolic pathways has been reported to be ~9

Table 3. Power output and pedal rate during 6-s maximal sprint exercise under conditions of normal and elevated muscle temperature

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>ET</th>
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<tbody>
<tr>
<td>Maximal external power, W</td>
<td>1,202 (347)</td>
<td>1,462 (435)*</td>
</tr>
<tr>
<td>Mean external power, W</td>
<td>878 (214)</td>
<td>1,006 (242)*</td>
</tr>
<tr>
<td>Maximal pedal rate, rpm</td>
<td>155 (21)</td>
<td>176 (26)†</td>
</tr>
<tr>
<td>Mean pedal rate, rpm</td>
<td>124 (20)</td>
<td>142 (25)*</td>
</tr>
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Values are means (SD); n = 8. Significant difference between conditions: *$P < 0.05$ and †$P < 0.01$. 

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mmol·kg^{-1}·s^{-1} (dm) for both PCr utilization during electrical stimulation (25) and glycolysis during 30-s isokinetic cycling at 140 rpm (27). On this basis, there is clear capacity for ATP turnover to increase.

The close association between ATP turnover and MFCV (Fig. 1) highlights the relationship between muscle activation and energy turnover. The effect of elevating T_m was to increase MFCV, probably due to a temperature-mediated effect on voltage-gated Na$^+$ channels (for a review, see Ref. 39), alongside the elevated ATP turnover. At higher temperatures, the opening and closing of these channels accelerate, allowing less Na$^+$ to enter the cell. A corresponding decrease in action potential amplitude, duration, and area follows, leading to a more rapid onset depolarization, producing a faster MFCV (40). The more rapid action potential delivery to the muscle fibers will lead to a greater Ca$^{2+}$ release from the sarcoplasmic reticulum, leading to a faster rate of cross-bridge cycling, thus requiring the greater rate of ATP turnover observed. It might also be considered that this would lead to a greater ATP turnover by sarcoplasmic reticulum Ca$^{2+}$ ATPase. This is unlikely, however, as the activity of the Ca$^{2+}$ ATPase has been found to be slightly depressed in rat muscle fibers after 30-min incubation at 37°C (45), meaning that the majority, if not all, of the elevated ATP turnover comes from an

Fig. 2. Individual power output during 6-s maximal sprint exercise under conditions of normal (●) and elevated (○) muscle temperature.

Fig. 3. Relationship between temperature coefficient ($Q_{10}$) values for ATP turnover and mean power output ($n=6$).
increased ATP hydrolysis by myofibrillar ATPase at the myosin head. We hypothesized that the greater power output achieved as a result of heating would occur in conjunction with a greater ATP turnover. However, although greater ATP turnover was observed in all subjects during ET, not all subjects increased power output. This is in contrast to the fact that, for a 1°C increase in T_m, there is a 10% increase in power output (41). Furthermore, the change in ATP turnover was not reflected by similar magnitude of change in power output (Fig. 2). There are several possible reasons for this. One explanation might be that the efficiency of contraction is lower at the higher T_m. Mechanical efficiency can be estimated by using values of molar enthalpy during muscle contraction when ATP resynthesis is powered by a net hydrolysis of ATP (35 kJ/mol of ATP used), PCr hydrolysis (55 kJ/mol of ATP used), and the anaerobic utilization of glycogen (67 kJ/mol of ATP used) (see Refs. 10, 22, and 49 for further discussion). With an accurate measure of mechanical power output, which includes the so-called internal work performed to overcome inertial and gravitation forces of the lower limbs (34), we observed that mechanical efficiency was the same between the two temperature conditions (Table 4). Of course, the absolute values are relatively low compared with measurements of mechanical efficiency during sustained moderate (~40 W) and intense (~65 W) knee-extensor exercise [e.g., ~25% (19) and 35–50% (31), respectively], which reflects that high power is achieved at the expense of efficiency (11, 13). The fact that the estimate of mechanical efficiency in the present study is not lower in the heated condition is partially supported by previous studies in which it was suggested that efficiency would actually be increased at a relatively high speed of contraction under ET (20). This is due to the rightward shift in the efficiency-velocity relationship (e.g., Ref. 24). From this shift, it would seem likely that efficiency would increase at the high pedaling rates attained in the present study. This would only be the case, however, if the pedal rate was maintained at a constant velocity, which is not the case while using a friction-braked cycle ergometer. In the present study, the pedal rate increased by ~20 rpm in the heated trial, i.e., moving to the right on the efficiency-velocity relationship. This counters the rightward shift with temperature, the result being that the velocity is at about the same relative point on the curve, and hence efficiency is not altered.

An alternative explanation may be related to the fact that, with the friction-loaded cycle ergometer, the load is fixed, and

Table 4. Muscle energetics during 6-s maximal sprint exercise under conditions of normal and elevated muscle temperature

<table>
<thead>
<tr>
<th>Energy Source</th>
<th>N</th>
<th>ET</th>
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<tbody>
<tr>
<td>Net ATP hydrolysis, J/s</td>
<td>262 (113)</td>
<td>402 (136)*</td>
</tr>
<tr>
<td>Net PCr hydrolysis, J/s</td>
<td>3,518 (625)</td>
<td>5,144 (1,171)*</td>
</tr>
<tr>
<td>Lactate accumulation, J/s</td>
<td>3,204 (900)</td>
<td>3,704 (990)*</td>
</tr>
<tr>
<td>Metabolic input, J/s</td>
<td>6,984 (1,520)</td>
<td>9,251 (2,115)*</td>
</tr>
<tr>
<td>Total mechanical work, J/s</td>
<td>961 (298)</td>
<td>1,144 (347)*</td>
</tr>
<tr>
<td>Mechanical efficiency, %</td>
<td>13.7 (2.4)</td>
<td>12.3 (2.2)</td>
</tr>
</tbody>
</table>

Values are means (SD); n = 6. Metabolic input was calculated as the sum of energy turnover from the net ATP hydrolysis, PCr hydrolysis, and lactate accumulation. Total mechanical work was calculated as the sum of external and “internal” power output. Mechanical efficiency was calculated as the ratio between total mechanical work and metabolic input. *Significant difference between conditions, P < 0.05.
any change in power output is achieved through a change in pedal rate. The maximal pedal rate under control conditions was \(~160~\text{rpm}\), which is much higher than the optimal rate suggested for maximal power development (43) and also approaching the highest possible pedal rate. To obtain a higher power output when heated, the pedal rate would have to increase, as was observed (Table 2). It is possible that the two subjects who did not increase power output under ET conditions had reached, at \(~160~\text{rpm}\) in the N condition, their own biomechanical limit of knee angular velocity. Thus no further increase in velocity was possible when the muscles were heated.

So far, in this discussion, we have been concerned with the effect of passive, local heating on power output and ATP turnover of the active muscles as a whole. It is clear that the whole of the available musculature would be active in the present study (28), so consideration must be given to the contribution of individual muscle fibers, which have diverse contractile and metabolic properties. We observed that the magnitude of the temperature-dependent increase in power output is correlated with the percentage of MHC IIA (Fig. 4), which is in contrast to the previous suggestion that type I fibers are equally sensitive to changes in temperature in the pedal cadence range of 60–140 rpm (44). Because all fibers are equally affected by temperature (24) and these effects are velocity specific, this discrepancy can be explained by the higher contraction velocities achieved with the use of the friction-loaded cycle ergometer. The maximum shortening velocity (\(V_{\text{max}}\)) of fibers with predominantly MHC I is equivalent to \(~165~\text{rpm}~(42)\), with an optimal velocity (\(V_{\text{opt}}\)) of \(~60~\text{rpm}~\). In the present study, these fibers are likely to be working close to or beyond their \(V_{\text{max}}\) thus having a minimal contribution to power output, even in the control condition. The \(V_{\text{max}}\) ratio between fibers with a predominance of MHC I and MHC IIA is \(~2.3~(9)\). With a \(V_{\text{opt}}\) at approximately one-third of \(V_{\text{max}}~(9,~24)\), the hypothetical \(V_{\text{opt}}\) of MHC IIA fibers would be \(~130–140~\text{rpm}\), well within the range of pedal velocities achieved. It is, therefore, likely that these fibers had the dominant contribution to power output in the present study and will be working beyond their optimum on the descending right arm of the power-velocity relationship. A rightward shift in this relationship, with an increase in \(T_m\), will result in a substantial increase in the power output of these fibers.

In a practical sense, Jones and colleagues (26) suggested that warmup would be of the greatest benefit to the power output of athletes with a high proportion of type I fibers. This was considered an unfortunate paradox, as those athletes with the most to gain from an increased power output (e.g., sprinters) will have large type II content and thus gain little from an increased \(T_m\). The present data also suggest that fibers with a high proportion of MHC IIA are most sensitive to temperature at the relatively fast cadences reached in the present study.

REFERENCES

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