Effect of active warm-up on metabolism prior to and during intense dynamic exercise

SUSAN C. GRAY, GIUSEPPE DEVITO, and MYRA A. NIMMO

Strathclyde Institute for Biomedical Sciences, University of Strathclyde, Glasgow, UNITED KINGDOM

ABSTRACT

GRAY, S. C., G. DEVITO, and M. A. NIMMO. Effect of active warm-up on metabolism prior to and during intense dynamic exercise. *Med. Sci. Sports Exerc.*, Vol. 34, No. 12, pp. 2091–2096, 2002. **Purpose:** This study investigated whether active warm-up (AW) would increase muscle acetylcarnitine concentration before exercise, thereby reducing the reliance on nonoxidative ATP production during subsequent high-intensity exercise. **Methods:** Six female subjects performed a 30-s sprint at 120% of their maximal power output on an electronically braked cycle ergometer 5 min after undertaking an active warm-up. To exclude any effect of muscle temperature (T_m) on metabolism, AW was compared with control (C), which involved passively heating the muscle to the same temperature as that achieved by active warm-up (37.1 ± 0.3 vs 37.2 ± 0.2°C AW and C, respectively). **Results:** Active warm-up significantly increased the concentration of acetylcarnitine from 4.5 ± 1.5 mmol·kg dry muscle $(dm)^{-1}$ at rest to 9.4 ± 1.6 mmol·kg dm^{-1} before the onset of exercise. There was no change in acetylcarnitine concentration in C. During exercise the accumulation of muscle lactate was significantly less in AW compared with C (21.9 ± 3.8 vs 34.3 ± 2.3 mmol·kg dm^{-1}, respectively). **Conclusion:** The main finding of this study was that there was less accumulation of blood and muscle lactate during intense dynamic exercise preceded by active warm-up, which could not be accounted for by a difference in T_m between trials immediately before the onset of exercise **Key Words:** ACETYLCARNITINE, HIGH-INTENSITY CYCLING, LACTATE, MUSCLE TEMPERATURE

Warm-up has been reported to alter the metabolic response during a subsequent exercise bout when compared with control (7,8,18,19). It has been suggested that this response is the result of an increase in blood flow and therefore oxygen (O_2) delivery to the active muscles after active warm-up (18). However, a recent study by Bangsbo et al. (1) has shown that O_2 supply to the contracting muscle is in excess of demand in the initial phase of dynamic exercise and that O_2 delivery is not limiting for oxygen uptake ($\dot{V}O_2$) of the contracting muscles. Thus, it does not seem likely that a differing metabolic response during exercise preceded by warm-up is the result of a difference in O_2 delivery to the active muscle.

It has also been proposed that the associated increase in muscle temperature (T_m) during warm-up influences metabolism during subsequent exercise (7,8,18,21). After passive warm-up, it has been reported that there is an increased dependency on anaerobic metabolism during high-intensity dynamic exercise (8) and an increase in muscle glycogen utilization during submaximal exercise (21). During exercise preceded by active warm-up, reductions in the accumulation of both blood and muscle lactate concentrations have been observed (10,18,19). Although both Febbraio et al. (8) and Starkie et al. (21) attributed alterations in metabolism to a direct effect of T_m, Gray and Nimmo (10) observed no difference in blood metabolites during exercise despite a significant difference in T_m between passive and control trials immediately before the onset of exercise, suggesting that other factors may also be responsible for the observed metabolic alterations during exercise preceded by warm-up.

There is evidence to suggest that mitochondrial acetyl group availability may partly determine the relative contribution made by anaerobic and oxidative ATP regenerating pathways at the onset of intense skeletal muscle contraction (22,23,24). An increase in muscle acetylcarnitine concentration after pharmacological activation of the pyruvate dehydrogenase complex (PDC) by dichloroacetate (DCA) results in a reduction in PCr degradation and lactate accumulation during exercise in both canine (22,23) and human (14,24) skeletal muscle. It has also been reported in a refereed abstract by Campbell et al. (3) that a low-intensity warm-up (55% $\dot{V}O_{2max}$) elevates muscle acetylcarnitine concentration without affecting PCr degradation or lactate accumulation during a subsequent bout of intense exercise (3 min at 90% $\dot{V}O_{2max}$). Because the concentration of muscle acetylcarnitine has been shown to be related to exercise intensity and increases with increasing intensity (6,13), it is possible that the intensity of active warm-up employed by Campbell et al. (3) did not result in a sufficient increase in acetylcarnitine concentration before the onset of exercise to significantly reduce the reliance on anaerobic metabolism during exercise.

Address for correspondence: Dr. Susan Gray, School of Life Sciences, Napier University, Merchiston Campus, 10 Colinton Road, Edinburgh, EH10 5DT, United Kingdom; E-mail: s.gray@napier.ac.uk.
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The aim of this study, therefore, was to test the hypothesis that an active warm-up, which includes both low- and high-intensity components, would increase acetyl group availability sufficiently to alter metabolism during subsequent intense dynamic exercise. To exclude any direct effects of T_m on metabolism, the active warm-up trial was compared with a control trial which, through passive warming, heated the muscle to the same temperature as that achieved by active warm-up.

METHODS

Subjects. Six healthy female subjects volunteered to participate in this study which was approved by the University of Strathclyde Ethics Committee. Before participation, all subjects were required to sign a letter of informed consent, having previously had all possible risks and discomforts fully explained to them in both written and verbal form. The mean (±SD) age, height, body mass, percent body fat, peak oxygen uptake ($\dot{V}O_{2peak}$), and maximal power output (PO_{max}) of the subjects were 26 ± 4 yr, 1.64 ± 0.06 m, 60.9 ± 4.0 kg, 24.2 ± 3.7%, 43.3 ± 5.5 $mL \cdot kg^{-1} \cdot min^{-1}$, and 239 ± 26 W, respectively. To control for hormonal fluctuations between tests, the timing of the main experimental trials coincided with two specific points within one complete menstrual cycle. Subjects were tested between 7 and 10 d after the onset of menses and again approximately 12 d after ovulation (subjects having provided a history of their menstrual cycle). Testing was undertaken at these times as it has previously been shown that the metabolic response to intense exercise does not differ between these two phases of the menstrual cycle (16). To exclude any effects of menstrual cycle phase on core temperature, three subjects performed their first trial 7–10 d postmenses whereas the other three subjects performed their first trial postovulation.

Preexperimental protocol. Peak oxygen uptake ($\dot{V}O_{2peak}$) was directly determined for each subject by means of a continuous incremental cycling test to volitional exhaustion on an electronically braked cycle ergometer (Excalibur Sport, Lode BV, Groningen, The Netherlands). The test was initiated at 70 W and increased by 35 W every 2 min for the first 6 min, and thereafter every minute until subjects could no longer complete the given work load. Expired air was analyzed continuously throughout the test by using an automated on-line gas analysis system (Oxycongamma, Mijnhardt B.V., The Netherlands). The workload at which subjects reached exhaustion was taken to be the maximal power output at $\dot{V}O_{2peak}$ (PO_{max}). After this test, all subjects were required to undertake a familiarization trial to fully accustom them with the intense nature of the exercise and with the procedures involved in data collection and blood sampling.

Experimental protocol. The two main experimental trials were performed at the same time of day (between 9 a.m. and 11 a.m.). To control for variations in environmental conditions during the course of this study, trials were performed in an environmental chamber (Sanyo Gallenkamp PLC, Loughborough, UK) maintained at a constant temperature of 22°C and relative humidity of 35%. Subjects reported to the laboratory 3 h postprandial and having abstained from alcohol, caffeine, and strenuous physical activity in the preceding 24 h. In addition, each subject was required to record and replicate the same dietary intake and physical activity pattern during the 48 h period before each trial.

On arrival at the laboratory, subjects inserted a rectal thermistor probe (Grants Instruments Ltd., Cambridge, UK) 10 cm beyond the anal sphincter and were instrumented with a heart rate (HR) monitor (Polar Vantage NV^{tm}, Electro Fitness Technology, Kempele, Finland). Subjects then rested quietly on an examination couch, set up in the environmental chamber, while the thigh of one leg was prepared for muscle biopsy (2). Briefly, two small incisions (approximately 1 cm) were made, 2–3 cm apart, through the skin and fascia superficial to the vastus lateralis muscle under local anesthesia (1% lignocaine; Astra Pharmaceuticals Ltd., London, UK). At this time a flexible T_m probe (Ellab [UK] Ltd., Norfolk, UK) was inserted into the vastus lateralis muscle of the opposite leg to where the biopsies were being taken to allow continuous monitoring of T_m, and an indwelling cannula (Venflon 20G, BOC Ohmeda, Sweden), attached to a three-way valve, was inserted into an antecubital vein. Throughout the trials, the cannula was kept patent by the injection of a small amount (1–2 mL) of sterile saline solution (0.9% sodium chloride BP; B. Braun Medical Ltd., Bucks, UK) at regular intervals. Residual saline in the dead space of the cannula was drawn off and discarded before sampling

A schematic diagram of the experimental protocol is presented in Figure 1. On one visit to the laboratory, subjects undertook an active warm-up protocol (AW). The active warm-up consisted of cycling at 40% PO_{max} for 5 min at a cadence of 60 $rev \cdot min^{-1}$ followed by a 1-min rest period, then four 15-s sprints (120% PO_{max}, 120 $rev \cdot min^{-1}$), with a 15-s recovery period separating each sprint. On the alternate visit, subjects undertook a control trial (C). On this occasion, before exercise, subjects' legs were passively heated to the same preexercise T_m as that induced by the active warm-up (determined either from familiarization or the main trial). This was achieved by wrapping an electric heat blanket around both legs from the ankle to the gluteal fold. A nontreatment control trial (i.e., no elevation in T_m) was not included in the experimental design, as it has previously been shown that there is no difference in blood

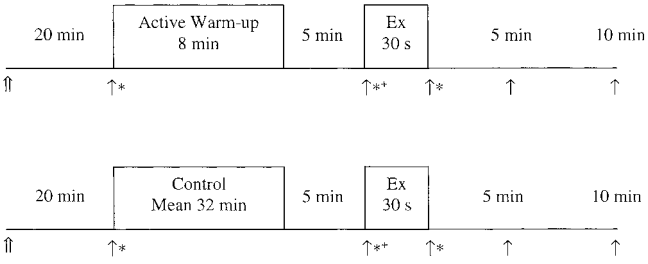

FIGURE 1—Schematic representation of the experimental design; ⇑ indicates biopsy/catheterization procedure; ↑ indicates blood sample collection and HR recording; * indicates muscle biopsy collection; + indicates T_m and T_{re} recording.

metabolite concentrations during high-intensity exercise between a passive and control (no heating) trial (10). The standardized exercise task (Ex), common to both trials, followed 5 min after the completion of the AW and C procedures, and required subjects to cycle on an electronically braked ergometer (Excalibur Sport, Lode BV) at 120% of their individual PO_{max} for a 30-s period, while maintaining a constant pedal cadence of 120 rev·min^{-1}. The order of trials was determined using a counterbalanced design.

At all sampling points, 7.5 mL of whole blood was collected into a plain sterile plastic syringe (10 mL; Plastipak®, Becton Dickinson, Madrid, Spain). For all samples, 2.5 mL of whole blood was dispensed into a tube containing potassium EDTA (K^+EDTA). Duplicate aliquots (100 μL) were removed immediately from the K^+EDTA tube and were deproteinized in 1 mL of ice-cold 0.4 mmol·L^{-1} perchloric acid. The aliquots were centrifuged (Eppendorf 1440, Hamburg, Germany) for 3 min at 4°C and 4000 rev·min^{-1}, and the resultant supernatant stored at −20°C until subsequent analysis. An additional 5 mL of whole blood was removed from the K^+EDTA tube and dispensed into a chilled tube containing lithium heparin, EGTA, and reduced glutathione. All samples were centrifuged (Eppendorf 1440) for 10 min at 4°C and 4000 rev·min^{-1}, and the resultant plasma removed and stored at −80°C until subsequent analysis. All muscle biopsies were obtained according to the methods described by Bergstrom (2), without the use of suction. The resting sample was taken 20 min after the biopsy incisions were made while subjects remained in a supine position on the examination couch. After the muscle heating procedures in AW and C, a preexercise biopsy was obtained from the same incision as the resting sample, again while subjects were on the examination couch. In the AW trial, this meant subjects returning to the couch after the active warm-up procedure. The final biopsy was obtained immediately upon completion of exercise from the second incision while subjects remained seated on the cycle ergometer. During the trials, the biopsy sites were covered with a sterile dressing. Samples (89 ± 34 mg wet weight; range, 35–180 mg) were immediately frozen in liquid nitrogen (N_2), removed from the needle, and stored in liquid N_2 until subsequent analysis.

Analyses. Upon completion of the experimental period, all blood samples were analyzed to determine the concentrations of blood lactate (17), and circulating levels of plasma epinephrine (Epi) and norepinephrine (NE) (9). Biopsy samples were freeze-dried, dissected free of all visible blood and connective tissue, and powdered for subsequent analysis. Aliquots of freeze-dried muscle were extracted with 0.5 M PCA (containing 1 mM EDTA) and neutralized with 2.2 M $KHCO_3$. This extract was used for the determination of ATP, PCr, creatine, and lactate (12). Radioisotopic determination of acetylcarnitine and free carnitine was undertaken according to the methods of Cederblad et al. (4). Muscle metabolites, with the exception of lactate, were normalized to the highest creatine concentration for each subject to account for possible diluting effects of blood or connective tissue in the dried muscle samples.

Calculations. The rate of ATP provision (mmol ATP·kg dry muscle [dm]$^{-1}$) from anaerobic sources was estimated over the 30-s period of intense cycling in each trial, as described by Spriet (20):

$$\text{ATP provision rate} = \Delta\text{PCr} + 1.5\ (\Delta\text{lactate}) + 2\ (\Delta\text{ATP}) \quad (1)$$

where Δ is the difference between pre- and post-exercise concentrations.

Statistical analysis. Significant main effects for HR, blood lactate, plasma Epi and NE, and muscle lactate, ATP, PCr, creatine, acetylcarnitine, and free carnitine were determined using a two-way analysis of variance (ANOVA) with repeated measures on two factors (experimental treatment and sampling time). A paired *t*-test was used to identify differences between trials at specific time points, whereas a one-way ANOVA followed by a Dunnett's *post hoc* test, where appropriate, was used to identify differences over time. A paired *t*-test was used to determine whether differences existed in preexercise T_m and T_{re} between AW and C. Changes in blood and muscle lactate concentration and ATP regeneration from anaerobic sources during exercise were also analyzed using a paired *t*-test. The level of significance was accepted as $P < 0.05$. Results are presented as mean ± standard error of the mean (SEM).

RESULTS

Muscle and rectal temperature. Immediately before the onset of exercise, there was no difference in T_m between AW and C (37.1 ± 0.3 vs 37.2 ± 0.2°C, respectively). However, preexercise T_{re} was significantly higher in AW compared with C (37.6 ± 0.1 vs 37.4 ± 0.04°C, respectively).

Heart rate. Before the onset of exercise, HR was significantly greater in AW compared with C (Table 1). This difference persisted throughout the exercise period such that postexercise HR in AW was still significantly elevated above the corresponding value in C (Table 1). During the 10-min recovery period, there were no further differences in HR between trials.

Blood lactate. After the warm-up period in AW, blood lactate was significantly elevated above the resting value and the corresponding values in C at both pre- and postexercise (Table 1). The change between preexercise and peak lactate concentrations was significantly less in AW compared with C (1.30 ± 0.52 vs 4.50 ± 0.41 mmol·L^{-1}, respectively). There were no further differences between trials during the 10-min recovery period.

TABLE 1. Heart rate (HR) and blood lactate concentration ([La]) in the active warm-up (AW) and control (C) trials.

	Rest	Pre-Ex	Post-Ex	Five	Ten
HR (beats·min^{-1})					
AW	72 ± 5	99 ± 6*†	171 ± 3*†	87 ± 4	83 ± 4
C	69 ± 4	77 ± 2	165 ± 3*	76 ± 6	75 ± 4
[La] (mmol·L^{-1})					
AW	0.9 ± 0.1	5.2 ± 0.4*†	5.4 ± 0.3*†	6.1 ± 0.4*	5.9 ± 0.3*
C	1.0 ± 0.2	1.0 ± 0.2	3.8 ± 0.5*	5.2 ± 0.4*	5.1 ± 0.5*

Values are mean ± SEM; $N = 6$.
* Significantly different from rest, $P < 0.05$.
† Significantly different from C, $P < 0.05$.

TABLE 2. Concentrations of plasma epinephrine (Epi) and norepinephrine (NE) in the active warm-up (AW) and control (C) trials.

	Rest	Pre-Ex
Epi (ηmol·L^{-1})		
AW	0.63 ± 0.17	1.30 ± 0.37*
C	0.76 ± 0.24	1.19 ± 0.22*
NE (ηmol·L^{-1})		
AW	2.80 ± 0.47	5.57 ± 0.62*
C	2.68 ± 0.51	4.75 ± 0.47*

Values are mean ± SEM ($N = 6$).
* Significantly different from rest, $P < 0.05$.

Catecholamines. There was no difference in plasma Epi levels between AW and C, either at rest or immediately before the onset of exercise (Table 2). Similarly, plasma NE was not different between AW and C at rest or preexercise (Table 2). Before the onset of exercise, circulating catecholamine concentrations were significantly elevated above resting values in both trials (Table 2).

Muscle metabolites. There was a significant increase in muscle lactate concentration after active warm-up such that, immediately before the onset of exercise in AW, concentrations of this metabolite were significantly elevated above the corresponding value in C (Table 3). However, upon completion of exercise, muscle lactate concentration was not different between trials. Thus, the exercise-induced increase in muscle lactate was significantly lower in AW compared with C (21.9 ± 3.8 vs 34.3 ± 2.3 mmol·kg dm^{-1}, respectively). There was no difference between AW and C for concentrations of muscle ATP, PCr, or creatine although in both trials postexercise concentrations of ATP and PCr were significantly lower and creatine was significantly higher than resting and preexercise values (Table 3). Total ATP regeneration from anaerobic sources was also not different between AW and C (93.8 ± 13.3 vs 116.1 ± 5.3 mmol ATP·kg dm^{-1}, respectively).

The two-way ANOVA with repeated measures revealed that there was no significant trial effect for either acetylcarnitine or free carnitine. However, there was a significant time effect for acetylcarnitine in AW such that, before the onset of exercise, there was a significant increase ($P < 0.05$) in the concentration of acetylcarnitine after the active warm-up in AW, but no significant increase in C (Fig. 2a). The concomitant decrease in free carnitine during active warm-up failed to reach statistical significance ($P = 0.076$) (Fig. 2b). Individual responses of acetylcarnitine and carnitine to active warm-up are depicted in Figure 3, a and b.

TABLE 3. Concentrations of muscle lactate (m[La]), adenosine triphosphate (m[ATP]), phosphocreatine (m[PCr]), and creatine (m[Cr]) in the active warm-up (AW) and control (C) trials.

	Rest	Pre-Ex	Post-Ex
m[La] (mmol·kg dm^{-1})			
AW	4.2 ± 0.3	15.7 ± 2.4*†	37.6 ± 4.9*
C	3.9 ± 0.4	4.3 ± 1.3	38.6 ± 2.5*
m[ATP] (mmol·kg dm^{-1})			
AW	24.6 ± 1.0	23.8 ± 1.2	18.5 ± 0.8*
C	24.8 ± 0.4	24.5 ± 0.7	18.3 ± 1.2*
m[PCr] (mmol·kg dm^{-1})			
AW	75.3 ± 2.5	76.5 ± 5.5	23.2 ± 3.9*
C	74.3 ± 2.4	73.2 ± 2.5	21.0 ± 1.6*
m[Cr] (mmol·kg dm^{-1})			
AW	55.1 ± 2.6	54.3 ± 5.1	107.8 ± 4.2*
C	56.1 ± 1.9	57.6 ± 3.2	109.8 ± 2.8*

Values are mean ± SEM ($N = 6$).
* Significantly different from rest, $P < 0.05$.
† Significantly different from C, $P < 0.05$.

DISCUSSION

The main finding of this study was that there was less accumulation of both blood and muscle lactate during intense dynamic exercise preceded by active warm-up, which suggests there may be a decreased reliance on energy derived from anaerobic sources during the exercise period after an active warm-up. These findings were observed despite the fact that there was no difference in T_m between trials immediately before the onset of exercise. The current findings support previous work from this laboratory (10) and provide strong evidence to suggest that alterations in metabolism during exercise preceded by warm-up are not a direct effect of T_m alone, as proposed by Febbraio et al. (8) and Starkie et al. (21), but are attributable to other causative factors.

In the present study, there was a small (0.2°C) but significant difference in T_{re} between trials immediately before the onset of exercise. However, it is not thought that this difference was physiologically significant or that it would influence metabolism during intense exercise, as it has pre-

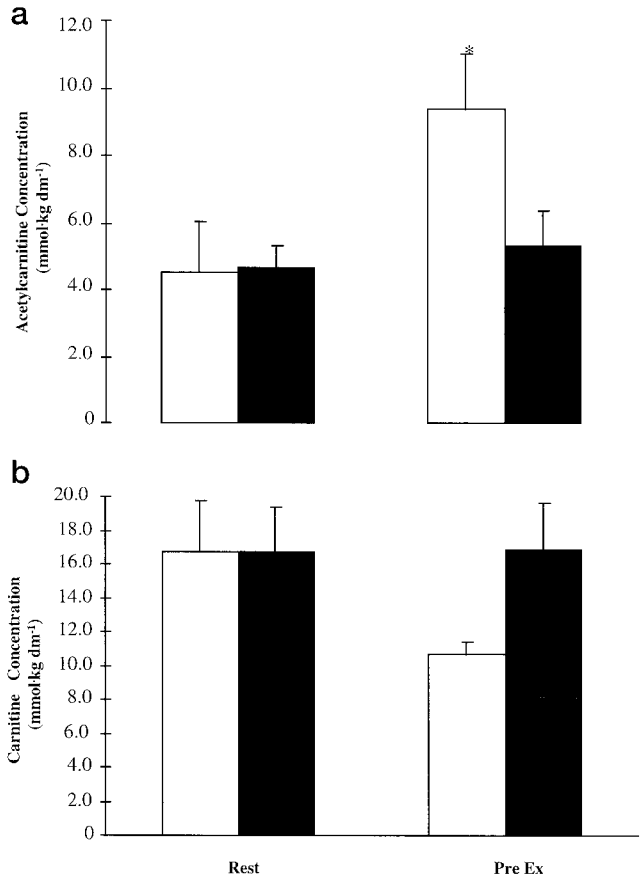

FIGURE 2—Skeletal muscle acetylcarnitine (a) and carnitine (b) concentration at rest and immediately before exercise (Ex) during AW (*clear bars*) and C (*filled bars*). Mean (±SEM) ($N = 6$). * indicates a significant difference from rest ($P < 0.05$).

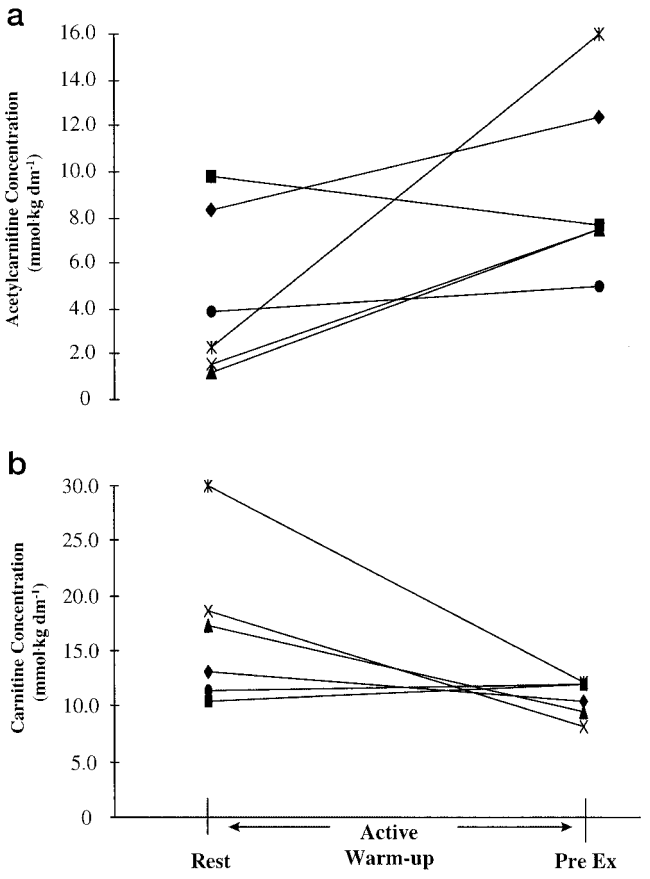

FIGURE 3—Individual response of skeletal muscle acetylcarnitine (a) and carnitine (b) concentration during active warm-up.

viously been demonstrated that a 0.2°C difference in T_{re} between passive warm-up and control trials had no influence on metabolism during subsequent exercise (10).

The use of an active warm-up in this study, which contained both low- and high-intensity components, has clearly demonstrated that this particular type of warm-up significantly increases the concentration of muscle acetylcarnitine before the onset of exercise. It has been proposed that the accumulation of large preexercise stores of muscle acetylcarnitine provides the extra substrate for oxidative ATP production at the onset of exercise (11). This is supported by the findings of the present study which indicate that the increase in muscle acetylcarnitine concentration before the onset of exercise is accompanied by a reduction in the accumulation of both blood and muscle lactate during exercise. Although Campbell et al. (3) also observed a significant increase in acetylcarnitine concentration in female subjects after active warm-up (55% $\dot{V}O_{2max}$), these authors did not report a reduction in muscle lactate accumulation during subsequent intense exercise (3 min at 90% $\dot{V}O_{2max}$). The reason for these equivocal findings is not clear but could be related to the intensity of active warm-up employed. The lower intensity of active warm-up employed by Campbell et al. (3) resulted in less acetylcarnitine accumulation (7.7 ± 1.0 mmol·kg dm^{-1}) before the onset of exercise compared with the present study (9.4 ± 1.6 mmol·kg dm^{-1}). Constantin-Teodosiu et al. (6) observed that the concentration of muscle acetylcarnitine during exercise at 30% $\dot{V}O_{2max}$ was similar to that at rest (6.2 ± 2.3 vs 6.0 ± 2.7 mmol·kg dm^{-1}, respectively) but increased to 10.1 ± 2.4 and 15.2 ± 2.8 mmol·kg dm^{-1} during exercise at 60 and 90% $\dot{V}O_{2max}$, respectively. Further evidence of a link between acetylcarnitine concentration and exercise intensity comes from the work of Howlett et al. (13), who noted that the concentration of muscle acetylcarnitine increased significantly as a function of power output. Thus, it is possible that the lower intensity of active warm-up employed by Campbell et al. (3) did not result in a reduced reliance on anaerobic metabolism during the subsequent bout of intense exercise.

In the present study, there was no significant difference in acetylcarnitine concentration between trials despite an increase in the concentration of this metabolite after active warm-up. However, it is possible that a significant trial effect was not detected due to the large degree of variability (25) in individual responses of acetylcarnitine to active warm-up, although in five of the six subjects acetylcarnitine did increase in AW. This variation in individual responses may also account for the observation that the decrease in free carnitine concentration during active warm-up failed to reach statistical significance ($P = 0.076$), despite the fact that in four of the six subjects there was a decrease in the concentration of free carnitine after active warm-up.

It has been reported that there is a link between the accumulation of acetyl groups, after activation of PDC by DCA, and modifications in metabolism during subsequent exercise. A reduction in PCr degradation and lactate accumulation has been observed during the first 3 min of an 8-min bout of single-leg knee extension exercise (24) and during 10 min of moderate-intensity exercise (65% $\dot{V}O_{2max}$) (14). These findings lend support to the observation of this study that the reduction in lactate accumulation during intense dynamic exercise after active warm-up could be associated with an increase in acetyl group availability before the onset of exercise. However, in contrast to the studies of Timmons et al. (24) and Howlett et al. (14), no difference in PCr degradation was observed during exercise in the present study or in that by Campbell et al. (3). It is possible that these equivocal findings may be associated with the differing mechanisms utilized to increase acetyl group availability (i.e., pharmacological intervention vs exercise). Furthermore, although the results of the present study indicate that increases in acetylcarnitine concentration after active warm-up provide an extra fuel source for oxidative ATP production at the onset of high-intensity exercise, it cannot be concluded from the current findings that there is a concomitant decrease in the reliance on anaerobic fuel sources, as evidenced by the finding of no difference in anaerobic ATP regeneration between trials. Further research in this area is warranted.

Although circulating plasma Epi and NE levels were elevated above resting values before the onset of exercise in AW and C, there was no difference in Epi and NE between trials. However, despite similar preexercise plasma catecholamine levels, a differing muscle lactate response was observed during exercise between AW and C. This observation indicates that metabolism during short-duration high-intensity exercise is influenced by factors other than Epi and

NE, and supports the findings of previous studies (5,15) that small physiological increases in Epi and NE do not appear to influence metabolism during subsequent intense exercise.

In conclusion, it has been shown that after active warm-up there is a reduction in blood and muscle lactate accumulation during subsequent short-duration, high-intensity exercise in eumenorrheic female subjects. It has been proposed that T_m is not the mediator of this metabolic alteration. Rather, it appears that the increase in muscle acetylcarnitine concentration after active warm-up increased acetyl group availability, which, in turn, may have provided a readily available fuel source for oxidative ATP regeneration at the onset of subsequent exercise.

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