Muscle metabolism during exercise in the heat in unacclimatized and acclimatized humans

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KING, DOUGLAS S., DAVID L. COSTILL, WILLIAM J. FINK, MARK HARGREAVES, AND ROGER A. FIELDING. *Muscle metabolism during exercise in the heat in unacclimatized and acclimatized humans.* J. Appl. Physiol. 59(5): 1350–1354, 1985.—The effect of heat acclimatization on aerobic exercise tolerance in the heat and on subsequent sprint exercise performance was investigated. Before (UN) and after (ACC) 8 days of heat acclimatization, 10 male subjects performed a heat-exercise test (HET) consisting of 6 h of intermittent submaximal [50% of the maximal O_2 uptake] exercise in the heat (39.7°C dB, 31.0% relative humidity). A 45-s maximal cycle ride was performed before (*sprint 1*) and after (*sprint 2*) each HET. Mean muscle glycogen use during the HET was lower following acclimatization [ACC = 28.6 ± 6.4 (SE) and UN = 57.4 ± 5.1 mmol/kg; $P < 0.05$]. No differences were noted between the UN and ACC trials with respect to blood glucose, lactate (LA), or respiratory exchange ratio. During the UN trial only, total work output during *sprint 2* was reduced compared with *sprint 1* (24.01 ± 0.80 vs. 21.56 ± 1.18 kJ; $P < 0.05$). This reduction in sprint performance was associated with an attenuated fall in muscle pH following *sprint 2* (6.86 vs. 6.67, $P < 0.05$) and a reduced accumulation of LA in the blood. These data indicate that heat acclimatization produced a shift in fuel selection during submaximal exercise in the heat. The observed sparing of muscle glycogen may be associated with the enhanced ability to perform highly intense exercise following prolonged exertion in the heat.

muscle glycogen; muscle pH; sprint exercise; blood lactate; blood glucose

THE PROCESS of heat acclimatization has been extensively studied with respect to changes in the body fluid spaces, central circulation, and temperature regulation (3, 23, 27, 28). Little is known, however, regarding any adaptive changes that may occur at the tissue (muscle) level.

The importance of muscle glycogen in determining endurance performance has been established (2). Exercise in the heat has also been observed to result in an increased utilization of muscle glycogen compared with exercise in a cold environment (9). The primary objective of this investigation was to study the effect of heat acclimatization on the utilization of muscle glycogen during prolonged submaximal exercise in the heat. The effect of heat-exercise stress on the ability to perform highly intense exercise was also investigated in subjects before and after heat acclimatization.

METHODS

Subjects and design. Ten healthy untrained males volunteered to serve as subjects in this study after being informed of all risks and stresses associated with these experiments. Written consent was obtained from each subject. Maximal O_2 uptake ($\dot{V}O_{2\,max}$) was determined on a stationary bicycle using an incremental protocol, which called for 25-W increases in power output each minute. This test was performed before and 2 days after the second heat-exercise stress trial. Two of the subjects were engaged in heavy resistance training, whereas the remaining subjects were not engaged in any regular vigorous physical activity. Subject characteristics are presented in Table 1.

All trials were conducted during the months of January, February, and March to reduce any natural heat acclimatization. Environmental temperatures during these months averaged −6.8, 1.6, and −1.8°C, respectively.

Before (UN) and after 8 days of heat acclimatization (ACC), the subjects performed a 6-h heat-exercise test (HET) and sprint-exercise tests as described below (Fig. 1). The subjects reported to the laboratory following an overnight fast. A preexperiment nude weight was obtained, and a thermister probe (Yellow Springs Instrument) was inserted 10 cm into the rectum. During all heat exposures, the subjects were dressed in athletic shorts and shoes. An infusion set was introduced into an antecubital vein and was kept patent with 0.9% saline.

Sprint-exercise test. The subjects performed a maximal 45-s ride on a hydraulically braked isokinetic cycle ergometer (Lumex, Ronkonkoma, NY) prior to heat exposure (*sprint 1*) and within 1 min after exiting the environmental chamber following the HET (*sprint 2*). The pedaling rate was set at 90 rpm for all sprint rides. The ergometer was calibrated before the experiment with known torques. Total work accomplished during the 45-s exercise was measured by digitizing the curves obtained from a chart recorder. The fatigue index (percent decline in torque) as defined by Thorstensson (25) was also calculated.

Needle biopsy samples (1) were obtained from the vastus lateralis muscle before and within 5 s following each sprint bout. Muscle samples were quickly frozen in liquid N_2 and subsequently analyzed for Na^+, K^+, Cl^-, glycogen, and pH. Blood was obtained in heparinized

TABLE 1. *Subject characteristics*

Age, yr	Ht, cm	Body Wt, kg		Plasma Volume, % change	$\dot{V}O_{2\,max}$, l/min	
		UN	ACC		UN	ACC
22.5±1.8	181.1±2.4	79.06±2.36	79.57±2.38	9.2±1.7*	3.91±0.12	3.92±0.09

Values are means ± SE; $n = 10$. $\dot{V}O_{2\,max}$, maximal O_2 uptake; UN and ACC, unacclimatized and acclimatized trials. * Significant increase in plasma volume following acclimatization ($P < 0.05$).

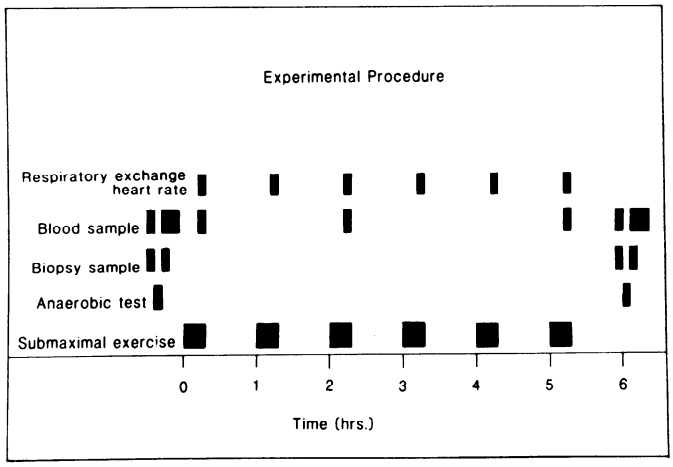

FIG. 1. Experimental procedure.

syringes after 5 min of rest, immediately after sprint exercise, and after 5, 10, and 15 min of recovery for the determination of hemoglobin, hematocrit, lactate, and blood gas concentrations.

Heat-exercise test. Immediately after the 15-min post-sprint blood sample, the subjects moved into the environmental chamber and began the 6-h HET. The mean chamber temperature was maintained at 39.7 ± 0.1°C (SE) dry bulb and 31.0 ± 0.7% relative humidity. During this exposure, the subjects exercised on a cycle ergometer (Monark) for the first 30 min of each hour at an exercise intensity calculated to require 50% $\dot{V}O_{2\,max}$. Respiratory exchange data were obtained from expired gas collected in Douglas bags during the last 10 min of each exercise bout. Heart rates were recorded during the final 30 s of submaximal exercise by use of a cardiotachometer or by auscultation. Rectal temperature was monitored at 5-min intervals throughout the 6 h of heat exposure. During the 1st, 3rd, and 6th h, blood samples were obtained during the last minute of exercise and subsequently analyzed for glucose and lactate concentrations. Pre- and post-HET blood samples were also analyzed for Na^+, K^+, and Cl^- concentrations.

During the final 30 min of each hour, the subjects rested in the supine position. The subjects were given 100 ml of tap water (40°C) at 30-min intervals throughout the HET.

At the conclusion of the HET, the subjects exited the chamber and moved immediately to the bicycle ergometer, where they performed another 45-s sprint rise (*sprint 2*) as described above.

Heat acclimatization. The subjects performed bicycle exercise in the heat for 90 min each day for 8 days. The exercise required a mean $\dot{V}O_2$ of 2.14 ± 0.07 (± SE) l/min, or 54.7% of the subject's $\dot{V}O_{2\,max}$. During the heat acclimatization bouts, the subjects were allowed water ad libitum.

Analytical methods. Muscle pH was measured using a homogenate technique as previously described (6). After hydrolysis of the muscle glycogen with 2.0 N HCl, glucosyl units were determined with a fluorometric method (19). Muscle electrolyte determinations were performed after lipid extraction with petroleum ether and 24 h of extraction in 2.0 N nitric acid. Na^+ and K^+ concentrations in blood and muscle were measured in triplicate by flame photometry. Muscle and blood Cl^- was measured in triplicate by coulometric-amperometric titration (7).

Hemoglobin concentration was determined using the cyanmethemoglobin method. Hematocrit was determined after microcentrifugation. Plasma volume changes were then calculated according to the procedures of Dill and Costill (8). One milliliter of whole blood was kept anaerobic on ice for subsequent blood gas and pH determination. Blood pH, CO_2 partial pressure (P_{CO_2}), and O_2 partial pressure (P_{O_2}) were determined with a BMS 3 MK 2 blood microsystem and PHM 73 pH blood gas monitor (Radiometer, Copenhagen). Blood glucose and lactate concentrations were determined enzymatically (19, 24).

The data were analyzed utilizing analyses of variance for repeated measures designs. Significant mean differences were located with the Newman-Keuls multiple comparison test. The $P < 0.05$ level of significance was chosen.

RESULTS

Submaximal exercise in the heat. Based on changes in hemoglobin and hematocrit, mean resting plasma volume increased 9.2 ± 1.7% during the 8 days of acclimatization ($P < 0.001$, Table 1). Although mean body weight increased 0.51 kg as a result of acclimatization, this difference was not statistically significant. Maximal O_2 uptake was not influenced by the acclimatization procedure.

Mean exercise $\dot{V}O_2$ during the HET was not different in the UN (1.92 ± 0.08 l/min) and the ACC (1.88 ± 0.09 l/min) trials. The respiratory exchange ratio (R) was also unaffected by acclimatization state (UN = 0.81 ± 0.01; ACC = 0.80 ± 0.01). Mean exercise heart rate was significantly reduced following heat acclimatization (160 ± 3 vs. 144 ± 3 beats/min). The change in rectal temperature during the UN trial (1.45 ± 0.15°C) was significantly greater than in the ACC trial (1.13 ± 0.13°C).

Blood glucose concentration did not differ significantly between the UN and ACC trials at any point (Fig. 2). One subject was unable to complete the final 30-min submaximal exercise bout during the UN trial and exhibited severe hypoglycemia (1.6 mmol/l) at 330 min. This subject was unable to perform the subsequent sprint exercise test but was able to perform the entire HET after the acclimatization process.

Blood lactate concentration was significantly higher in the UN trial (3.64 ± 0.38 mmol/l) compared with the ACC trial (2.87 ± 0.42 mmol/l) at 30 min only (Fig. 2). Values obtained at 330 min and post-HET were not significantly different from the resting values in either UN or ACC trials.

Resting muscle glycogen concentrations were not different between the UN and ACC trials (Table 2). Muscle glycogen concentration post-HET was significantly greater in the ACC trial than in the UN trial ($P < 0.05$). Acclimatization thus resulted in a 42% reduction in muscle glycogen utilization.

Sprint exercise. Total work output (Table 3) was significantly reduced during *sprint 2* compared with *sprint 1* in the UN trial. No such decrement in work output was observed as a consequence of the HET in the ACC trial. Peak torque, torque at 45 s, and percent decline in torque (fatigue index) were not significantly different between the UN and ACC trials or between the two sprint exercise bouts.

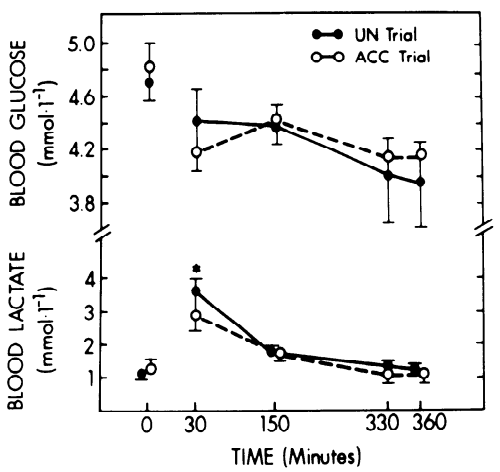

FIG. 2. Blood glucose and lactate concentrations during 6-h heat-exercise test. * Significant difference between unacclimatized (UN) and acclimatized (ACC) trials ($P < 0.05$).

TABLE 2. *Muscle glycogen and pH before and after 45-s sprint-exercise tests*

Sample	*Sprint 1*		*Sprint 2*	
	Pre	Post	Pre	Post
Glycogen				
UN	131.7 ± 9.1	103.7 ± 9.2	46.6 ± 6.9	31.8 ± 5.8
ACC	131.1 ± 9.2	113.1 ± 4.5	80.1 ± 4.4*	60.2 ± 4.7*
pH				
UN	7.15	6.67	7.12	6.86†
ACC	7.14	6.73	7.17	6.77

Values are means ± SE; $n = 9$. UN and ACC, unacclimatized and acclimatized trials; *sprints 1* and *2* refer to before and after 6-h heat-exercise test, respectively. Glycogen is expressed as mmol/kg wet wt. * Significantly different from UN ($P < 0.05$) trial. † Significantly different from *sprint 1* UN trial ($P < 0.05$).

TABLE 3. *Performance data for 45-s sprint-exercise tests*

	Sprint 1		*Sprint 2*	
	UN	ACC	UN	ACC
Total work, kJ	24.01±0.80	24.72±1.02	21.56±1.18*	24.67±1.37
Peak torque, N·m	82.7±3.3	87.1±3.0	80.6±6.2	84.7±4.8
Torque at 45 S, N·m	35.2±1.4	37.7±2.3	32.2±1.9	36.4±2.5
Fatigue index, %	56.9±2.7	56.4±2.8	58.7±3.5	56.2±3.8

Values are means ± SE; $n = 9$. UN and ACC, unacclimatized and acclimatized trials, respectively; *sprints 1* and *2*, before and after 6-h heat-exercise test, respectively. * Significantly different from all other means ($P < 0.01$).

TABLE 4. *Blood lactate concentration before and during recovery from 45-s sprint-exercise tests*

Time, min	*Sprint 1*		*Sprint 2*	
	UN	ACC	UN	ACC
Pre	1.12±0.08	1.27±0.13	1.22±0.14	1.12±0.15
0	6.57±1.15	6.01±0.82	5.15±0.81	6.70±1.23*
5	10.33±1.24	11.16±1.02	9.80±1.07	12.40±1.18*
10	11.48±0.95*	11.44±0.91*	9.85±1.08	12.22±1.09*
15	10.90±0.92*	10.88±0.82*	8.69±1.11	10.40±1.09*

Values are means ± SE expressed in mmol/l; $n = 9$. UN and ACC, unacclimatized and acclimatized trials, respectively. *Sprints 1* and *2*, before and after 6-h heat-exercise test, respectively. * Significantly different from *sprint 2*, UN ($P < 0.05$).

Muscle glycogen use for *sprints 1* and *2* was 28.0 ± 3.9 and 14.8 ± 4.0 mmol/kg for the UN trial. Corresponding values for the ACC trial were 18.0 ± 7.1 and 19.9 ± 5.6 mmol/kg for *sprints 1* and *2*, respectively. These means were not significantly different from each other.

Presprint muscle pH was not significantly altered by acclimatization or HET either in the UN or ACC trials (Table 2). During the UN trial, immediate postexercise muscle pH was significantly lower following *sprint 1* (6.67) than *sprint 2* (6.86). Values for postexercise muscle pH in the ACC trial were 6.73 and 6.77 for *sprints 1* and *2*, respectively, and were not significantly different.

No significant differences were noted in the presprint blood LA concentrations either between the UN and ACC trials or between *sprints 1* and *2* (Table 4). In the UN trial, blood LA concentrations after 10 and 15 min of recovery were significantly lower following *sprint 2* compared with *sprint 1*. Throughout recovery, blood lactate concentrations following *sprint 2* were lower for the UN trial compared with the ACC trial ($P < 0.05$).

No significant differences were observed between the UN and ACC trials for blood pH before or after sprint exercise (Table 5). Nadir values ranging from 7.13 to 7.22 were reached at 5 min postexercise in all sprints.

DISCUSSION

The changes observed in plasma volume, exercise heart rate, and rectal temperature demonstrate that the exer-

TABLE 5. *Blood pH before and during recovery from 45-s sprint-exercise tests*

Time, min	Sprint 1		Sprint 2	
	UN	ACC	UN	ACC
Pre	7.37	7.37	7.41	7.43
0	7.23	7.25	7.34	7.28
5	7.13	7.14	7.22	7.18
10	7.14	7.16	7.22	7.20
15	7.16	7.19	7.25	7.24

Values are means; $n = 9$. UN and ACC, unacclimatized and acclimatized trials, respectively.

cise protocol was successful in promoting acclimatization to exercise in the heat.

Muscle glycogen use. The major finding of this investigation was that muscle glycogen use during 6 h of intermittent exercise was markedly reduced following 8 days of heat acclimatization. These data suggest an alteration in substrate use following repeated days of exercise in the heat. This reduction in glycogen use, without any change in $\dot{V}O_{2\,max}$ or in blood lactate levels during submaximal exercise, suggests that this sparing of muscle glycogen is due to heat acclimatization and not to an effect of endurance training per se.

The mechanism(s) responsible for this decreased reliance on muscle glycogen stores during prolonged exercise in a hot environment can, at present, only be surmised. The reduction in muscle blood flow during exercise in the heat (21) results in a reduced delivery of blood-borne substrates to the exercising muscle. An increased perfusion of active skeletal muscle after heat acclimatization (21, 23) may have resulted in an augmented delivery of both glucose and nonesterified fatty acids and allowed for a reduced dependence on muscle glycogen for energy production.

The reduction in muscle glycogen use during the ACC trial was not associated with any change in R or blood LA concentration, suggesting that the rate of muscle glycolysis was unaltered after acclimatization. In a recent preliminary report, Green et al. (12) observed a reduced muscle glycogen utilization during submaximal exercise in a cool environment following 3 days of a physical training regimen that increased plasma volume by 21%. As was noted in the present study, Green et al. did not observe any difference in blood glucose concentrations or in the R. These results indicate that there was no shift in the use of carbohydrate and lipid for energy production. Furthermore, these investigators found that muscle concentrations of LA and glucose 6-phosphate after 2 h of exercise were unchanged following the 3 days of training. These data support the hypothesis that the acclimatization process did not result in any change in glycolytic flux during prolonged exercise. Hultman (14) has demonstrated that the rate of muscle glycogen utilization is inversely related to the release of glucose from the liver. Thus the finding of a reduced glycogen use without an apparent increase in lipid oxidation may reflect an enhanced rate of hepatic glucose release and subsequent use by active skeletal muscle.

If we assume an active muscle mass during cycling that is equal to 20% of body weight (10), the contribution of fat and carbohydrate to total energy production can be estimated. Muscle glycogen contributed 41 and 24% of the total energy expenditure in the UN and ACC trials, respectively. Total carbohydrate use comprised 37 and 33% of total energy expended in the UN and ACC trials, suggesting that blood glucose contributed little in the UN trial and ~9% in the ACC trial. This contribution of blood glucose, corresponding to a release of ~36 g from the liver, could be met by hepatic glycogen stores alone (15).

The estimates of substrate use presented above are open to question for the following reasons. These calculations assume an active muscle that is equal to 20% of body weight and also that the vastus lateralis reflects accurately the metabolism of the entire lower extremity during bicycle exercise. To our knowledge the active muscle mass and recruitment pattern during bicycle exercise has not been adequately quantified. Another criticism regarding these estimates is that respiratory exchange measurements may not adequately reflect substrate use under extreme environmental conditions (9). Thus direct measurements of glucose and nonesterified fatty acid exchange across the working muscle are necessary to explain the observed differences.

A lowering of plasma catecholamine levels during exercise in the heat may have played a role in the reduced glycogen utilization through direct effects on the exercising muscle (20) and also by improving splanchnic perfusion, thereby increasing hepatic delivery of glucose (22). Plasma catecholamine levels during exercise in the heat have been demonstrated to be higher than identical exercise in a cool environment (11). Although plasma catecholamines were not determined in the present study, reduced rectal and skin temperatures following heat acclimatization (23) might have resulted in decreased circulating levels of catecholamines during exercise in the heat. Furthermore, markedly reduced plasma catecholamine levels have been observed during submaximal exercise after only 1 wk of endurance training (26). This type of exercise training, however, has been shown to result in a significant increase in $\dot{V}O_{2\,max}$ within 1 wk (13), whereas no increase in $\dot{V}O_{2\,max}$ was observed in the present study. The role of decreased catecholamine levels in reducing muscle glycogen use in the heat remains to be clarified.

Sprint exercise. Total work output was significantly reduced in *sprint 2* in the UN trial, whereas in the ACC trial the subjects were unable to maintain sprint exercise capacity following prolonged exercise in the heat. Previous research suggests that thermal dehydration to a body weight loss of 5% has no effect on the performance of a highly intense (30 s) exercise bout (16). In the present study the estimated decrease in plasma volume during HET was similar in the UN (3.2%) and ACC (2.3%) trials, and body weight loss during the two trials was not different (UN = 3.0%, ACC = 3.4%). Thus the loss of body fluids does not appear to be responsible for the observed reduction in work output. As noted by previous investigators (4, 5), prolonged exercise in the heat did not alter the electrolyte content of muscle (data not shown), and no differences were noted between the

UN and ACC trials in the serum concentrations of Na^+, K^+, and Cl^-. In addition no differences were noted in peak torque between the UN and ACC trials during *sprints 1* or *2*. These data suggest that the excitability of skeletal muscle was not appreciably different between the UN and ACC trials before the final sprint bout.

The higher muscle pH observed following *sprint 2* compared with *sprint 1* during the UN trial represents a 75.8 nmol/l (35%) reduction of muscle H^+ accumulation. A diminished accumulation of LA in the blood was also noted following *sprint 2* in the UN trial. In addition venous blood pH tended to be higher following *sprint 2* for the UN trial. Reductions in sprint exercise performance and in the ability to accumulate LA in the blood and muscle have been observed when muscle glycogen is reduced (17, 18). Jacobs (17) has suggested that anaerobic performance is reduced when muscle glycogen is lower than 40 mmol/kg because of a relative lack of substrate for the flux-generating step of glycogenolysis. It is interesting to note that muscle glycogen approached this level (46.6 mmol/kg) in the UN trial after exercise in the heat. These data suggest that the reduction in sprint exercise performance following exercise in the heat in the UN trial was due to a reduction in the capacity for anaerobic energy release.

In summary, 8 days of heat acclimatization resulted in a marked reduction in muscle glycogen utilization during prolonged exercise in the heat. This alteration in substrate use during submaximal exercise was associated with an enhanced ability to perform highly intense exercise following heat-exercise stress.

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