Human muscle metabolism during sprint running

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CHEETHAM, MARY E., L. H. BOOBIS, S. BROOKS, AND C. WILLIAMS. *Human muscle metabolism during sprint running.* J. Appl. Physiol. 61(1): 54–60, 1986.—Biopsy samples were obtained from vastus lateralis of eight female subjects before and after a maximal 30-s sprint on a nonmotorized treadmill and were analyzed for glycogen, phosphagens, and glycolytic intermediates. Peak power output averaged 534.4 ± 85.0 W and was decreased by 50 ± 10% at the end of the sprint. Glycogen, phosphocreatine, and ATP were decreased by 25, 64, and 37%, respectively. The glycolytic intermediates above phosphofructokinase increased ~13-fold, whereas fructose 1,6-diphosphate and triose phosphates only increased 4- and 2-fold. Muscle pyruvate and lactate were increased 19 and 29 times. After 3 min recovery, blood pH was decreased by 0.24 units and plasma epinephrine and norepinephrine increased from 0.3 ± 0.2 nmol/l and 2.7 ± 0.8 nmol/l at rest to 1.3 ± 0.8 nmol/l and 11.7 ± 6.6 nmol/1. A significant correlation was found between the changes in plasma catecholamines and estimated ATP production from glycolysis (norepinephrine, glycolysis $r = 0.78$, $P < 0.05$; epinephrine, glycolysis $r = 0.75$, $P < 0.05$) and between postexercise capillary lactate and muscle lactate concentrations ($r = 0.82$, $P < 0.05$). The study demonstrated that a significant reduction in ATP occurs during maximal dynamic exercise in humans. The marked metabolic changes caused by the treadmill sprint and its close simulation of free running makes it a valuable test for examining the factors that limit performance and the etiology of fatigue during brief maximal exercise.

fatigue; adenosine 5′-triphosphate; glycolysis; catecholamines; lactate; power output; treadmill sprinting; females

WHEREAS THE PHYSIOLOGICAL and metabolic responses to prolonged submaximal exercise have been well documented (29, 33), there is relatively little information on the metabolic responses of human subjects to brief duration maximal exercise. The reason for this relative lack of information included, until recently, the absence of an acceptable exercise protocol and the lack of convenient methods for assessing the metabolic responses to high-intensity exercise.

The reintroduction and development of the Duchenne needle-biopsy technique by Bergström et al. (2) provided a means by which muscle metabolism could be examined directly in human subjects and which was, therefore, suitable for use in brief duration exercise. However, attention was initially focused on submaximal exercise and the responses to high-intensity exercise were confined to an examination of the metabolic responses to constant-velocity cycle ergometer tests at exercise intensities designed to elicit up to 130% of maximum O_2 uptake ($\dot{V}O_{2\,max}$) (24).

The development of a maximal-intensity cycle ergometer test in the 1970's by Bar-Or and colleagues (1) meant that for the first time it was possible to examine the metabolic responses to maximal exercise while monitoring the power output and the time course of the decline in power output that occurred during the test. It has since been shown that maximal cycle ergometer exercise of brief duration results in a marked decrease in muscle phosphagens and glycogen and an increase in the glycolytic intermediates (4). However, no such information is available on the power output during, and metabolic responses to, sprint running.

Recently, we have developed a test procedure based on the use of a nonmotorized treadmill that allows us to monitor the power output and follow the development of fatigue while an individual is running at maximal speed (25, 6).

It was the purpose of this study, therefore, to examine the changes in muscle metabolites following a 30-s exhaustive sprint on the nonmotorized treadmill while simultaneously examining the power output and fatigue developed over the time period of the test. The changes in plasma catecholamines and blood pH were also recorded to examine their relationship to performance and to the changes in muscle metabolism. Furthermore, a description of the metabolic responses to sprint running may lead to a better understanding of the factors that limit performance and in the etiology of fatigue during brief maximal exercise.

METHODS

The equipment used for the sprint test has been previously described (25). Treadmill belt velocity was monitored by means of a DC generator attached to the front rolling drum of the treadmill. During a sprint output voltage from the generator was continuously monitored by a microcomputer via an analog-to-digital converter. To calibrate the output from the generator a 1-hp electric motor was coupled to the treadmill. With the motor driving the belt, the generator output voltage corresponding to different belt speeds was determined and a calibration factor calculated. Restraint force was measured by means of a force transducer mounted on the rear crossbar of the treadmill, which was attached to a nonelastic belt that passed around the subjects waist. The

instantaneous product of restraint force and belt velocity was used to determine the horizontal component of power generated during the test. Results were integrated over 1-s time intervals and displayed by the computer at the conclusion of the sprint. The maximum speed at which the subjects could drive the treadmill belt will be referred to as "peak running speed." The maximum 1-s integral of restraint force and belt speed will be referred to as "peak power output," whereas mean "power output" will refer to the integral of restraint force and belt speed throughout the time period of the test.

Subjects. Eight female subjects (ht 164.90 ± 9.57 cm; wt 59.59 ± 8.15 kg; age 25.63 ± 6.80 yr) gave their informed consent and volunteered to participate in the study, which was conducted in conformity with the principles embodied in the Declaration of Helsinki for experiments involving human subjects. Seven of the subjects were highly trained, including two international games players.

Protocol. The subjects were previously familiarized for treadmill sprinting and, following an overnight fast, completed two 30-s periods of submaximal running on the nonmotorized treadmill (8 and 10 km/h) that served both as a warm-up and to reaccustom the subjects to experimental procedures. Five minutes after this standardized warm-up, a 30-s sprint was completed from a rolling start at 8 km/h. The subjects were asked to run maximally from the start of the test and were verbally encouraged throughout. Heart rate was recorded continuously during the sprint and for the first 5 min of a passive recovery.

The reproducibility of this test procedure has previously been examined and shown to be satisfactory (6).

Blood sampling and treatment. Venous blood samples were obtained 4 min after warm-up and at 3 min following the sprint for the determination of blood pH and plasma catecholamines, while capillary samples were taken 4 min after warm-up and at 5 min following the sprint for the determination of blood lactate and blood glucose concentrations. Venous samples were placed in tubes containing lithium-heparin to prevent coagulation. Blood pH was determined immediately (Corning pH blood/gas 161 meter). The remaining blood (8–10 ml) was centrifuged and the plasma treated with 200 μl of a mixture of 100 mmol/l ethyleneglycol-bis(β-aminoethylether-*N*,*N*′-tetraacetic acid and 100 mmol/l glutathione to act as an antioxidizing and chelating agent. The treated plasma was frozen at −20°C and analyzed at a later date for epinephrine and norepinephrine using high-performance liquid chromatography with electrochemical detection (9). Capillary samples were deproteinized in 2.5% perchloric acid, centrifuged, frozen at −20°C, and analyzed at a later date for blood glucose and blood lactate concentrations using the method described by Maughan (27).

Muscle sampling and treatment. Needle-biopsy samples were obtained under local anesthesia (1% plain lidocaine) from the vastus lateralis at rest and immediately after the sprint. The vastus lateralis was selected for study as during practice sprints subjects experienced fatigue mainly in the upper leg, and sampling from this muscle would allow comparison with the results of previous experiments examining the metabolic responses to maximal exercise that have used a 30-s sprint cycling test as the experimental model. Furthermore, the objective was to obtain the muscle sample as rapidly as possible after the sprint, and for this purpose the vastus lateralis was an easily accessible and safe muscle to use. The time taken from cessation of the sprint to the taking of the biopsy averaged 7.2 ± 0.9 s after which the needle was immediately plunged into isopentane maintained at its freezing point in liquid N_2 (mean delay 0.6 ± 0.1 s). The muscle was stored in the needles in liquid N_2 until freeze drying after which the samples were removed, dissected free of connective tissue and blood, powdered, and the fat removed by ether extraction. An acid extract of the muscle was obtained (15) and the neutralized extract was assayed enzymatically for phosphocreatine (PCr), adenosine 5′-triphosphate (ATP), adenosine 5′-diphosphate (ADP), glucose 1-phosphate (G1P), glucose 6-phosphate (G6P), fructose 6-phosphate (F6P), fructose 1,6-diphosphate (FBP), triose phosphates (TP), pyruvate, and lactate by fluorometric analyses (26). Glycogen was determined both on the neutralized extract and on the muscle pellet left after the extraction procedure by prior hydrolysis in HCl and subsequent analysis for glycosyl units giving an acid soluble and insoluble fraction. All muscle metabolite concentrations are expressed with respect to dry weight.

A Student's *t* test for correlated means was used to examine differences between the pre- and postexercise means. Results are presented as mean ± SD.

RESULTS

Power output. The average power output during each second of the treadmill sprint test is shown in Fig. 1. Mean power output during the test was 347 ± 55.7 W. Peak power output (mean 534.3 ± 85.0 W) was reached at an average of 1.63 ± 0.74 s into the sprint, after which there was a gradual decline in power output so that at the end of the sprint it was only 50 ± 10% of the peak value. As power output during treadmill sprinting has previously been shown to be related to body weight (25),

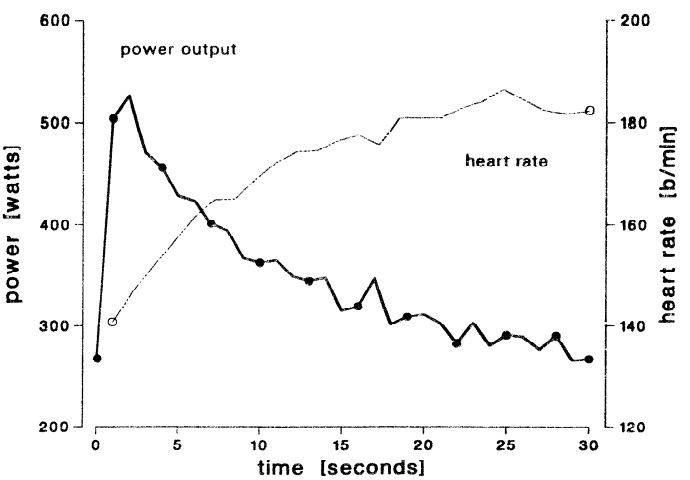

FIG. 1. Power output and heart rate (mean ± SD) during 30-s maximal sprinting on a nonmotorized treadmill for 8 female subjects.

power output was also expressed in relation to this variable. Peak power output/body weight averaged 9.19 ± 2.18 W/kg, and mean power output/body weight averaged 6.00 ± 1.36 W/kg.

The heart rate response to treadmill sprinting is illustrated in Fig. 1 and shows that while power output declined heart rate gradually increased to reach maximum values over the last few seconds of the sprint and first seconds of recovery.

Muscle metabolites. The resting and postexercise muscle metabolites are shown in Table 1. The 25% decrease in muscle glycogen was accompanied by a 64% fall in PCr and a 37% fall in ATP. No significant changes were observed in muscle ADP or glucose content. The glycolytic intermediates before FBP in the glycolytic pathway increased ~13-fold, whereas FBP itself and the TP only increased 4- and 2-fold, respectively. Pyruvate and lactate concentrations increased ~19 and 29 times. The total anaerobic ATP turnover during the 30-s sprint and proportion of ATP derived from glycolysis and PCr were calculated from the changes in muscle metabolites (Table 2). Although the aerobic contribution to the sprint is unknown and it is recognized that some lactate will have left the muscle during the sprint, total anaerobic ATP turnover was considered for the purposes of this study to be equal to the sum of ΔATP + ΔPCr + 1.5 Δlactate + 1.5 Δpyruvate, as has been previously described (31). ATP production from glycolysis was estimated from the accumulation of lactate and pyruvate (Δlactate 1.5 + Δpyruvate 1.5). These calculations showed that 64% of the ATP used during the sprint was supplied from glycolysis with the remainder being derived predominantly from PCr. The rate of ATP production averaged 6.13 ± 1.76 mmol·kg dry $wt^{-1} \cdot s^{-1}$ (range 3.02 to 8.71 mmol·kg dry $wt^{-1} \cdot s^{-1}$), with a tendency for the highest rates of ATP turnover to be associated with the fastest running speeds on the nonmotorized treadmill ($r = 0.70$, NS).

TABLE 1. *Muscle glycogen, phosphagens, glycolytic intermediates, and lactate at rest and post-treadmill sprinting exercise*

	Rest	Postexercise
Total glycogen	280.7±51.4	211.9±35.2*
Acid-insoluble glycogen	247.0±41.6	191.5±25.4*
Acid-soluble glycogen	33.7±11.5	20.4±13.1†
PCr	87.7±9.8	31.2±12.2*
ATP	28.2±3.4	17.9±4.6*
ADP	2.7±0.6	2.2±0.6
Glucose	2.6±0.9	3.6±1.2
G1P	0.2±0.1	2.3±0.8*
G6P	1.4±0.5	18.6±5.9*
F6P	0.4±0.2	4.4±1.5*
FBP	0.1±0.1	0.3±0.1*
TP	0.3±0.1	0.5±0.1*
Pyruvate	0.2±0.1	2.9±0.8*
Lactate	2.7±1.7	78.0±26.2*

Values are means ± SD. Measurements mmol/kg dry wt. Significant difference between pre- and postexercise values. PCr, phosphocreatine; G1P, glucose 1-phosphate; G6P, glucose 6-phosphate; F6P, fructose 6-phosphate; FBP, fructose 1,6-diphosphate; TP, triose phosphates. * $P < 0.01$. † $P < 0.05$.

TABLE 2. *Estimated ATP production and relative contribution of available energy stores to ATP production during 30-s treadmill sprinting*

	ATP, mmol/kg dry wt	% Contribution to Total ATP Produced
Total ATP production	183.8±52.7	
ATP from glycolysis	117.0±40.8	62.9±6.0
ATP from PCr	56.6±13.2	31.7±5.7
ATP depletion	10.3±6.9	5.5±3.0

Values are means ± SD. PCr, phosphocreatine.

Plasma catecholamines. After the treadmill sprint test, norepinephrine (NE) concentration had increased ~4-fold from 2.7 ± 0.8 nmol/l at rest to 11.7 ± 6.6 nmol/l 3 min following the sprint test. There was a similar change for epinephrine (E) from 0.3 ± 0.2 nmol/l at rest to 1.3 ± 0.8 nmol/l following the sprint. A significant relationship was found between the changes in norepinephrine and epinephrine and the estimated energy production from glycolysis (NE, glycolysis $r = 0.78$, $P < 0.05$; E, glycolysis $r = 0.75$, $P < 0.05$) (Fig. 2). In addition, those individuals who had the largest changes in plasma catecholamines tended to have the greatest changes in blood lactate and blood pH (postsprint E, blood pH $r = -0.74$, $P < 0.05$, and postsprint NE, blood lactate $r = 0.73$, $P < 0.05$). The highest correlations were found between the changes in plasma catecholamines and the blood glucose (BG) concentrations following the sprint (NE, BG $r = 0.85$, $P < 0.01$, E, BG $r = 0.88$, $P < 0.01$).

Blood pH. The treadmill sprint resulted in a decrease in blood pH of 0.24 units from 7.40 ± 0.02 at rest to 7.16 ± 0.07 3 min after the sprint. Muscle pH was estimated from the pre and postexercise muscle lactate and pyruvate concentrations [muscle pH = −0.00413 (lactate + pyruvate) + 7.06; see Ref. 30]. Resting muscle pH, calculated from this regression equation, was 7.05 ± 0.01. Immediately after the sprint, the estimated pH had fallen by 0.32 units to 6.73 ± 0.11. This respresents a greater fall than that measured in the blood, although a significant correlation was found between the changes in the measured blood pH and estimated muscle pH ($r = 0.73$, $P < 0.05$) (Fig. 3).

Blood lactate and glucose responses to treadmill sprinting. The blood lactate and glucose responses to treadmill sprinting are shown in Table 3. Whereas the warm-up caused a slight increase in blood lactate and blood glucose concentrations compared with the resting values, this change was relatively small when compared with the marked increases demonstrated following the 30-s sprint. Individuals having the highest postexercise capillary lactate levels also had the highest muscle lactate concentrations ($r = 0.82$, $P < 0.05$). Blood lactate concentration following the sprint was most highly correlated with peak running speed ($r = 0.93$, $P < 0.01$) and with peak power output/body weight ($r = 0.83$, $P < 0.01$).

Changes in plasma volume. Changes in plasma volume were estimated from the pre and postsprint hematocrit and hemoglobin levels (11). The 30-s sprint resulted in a mean decrease in plasma volume of 8.11 ± 3.81%. Those

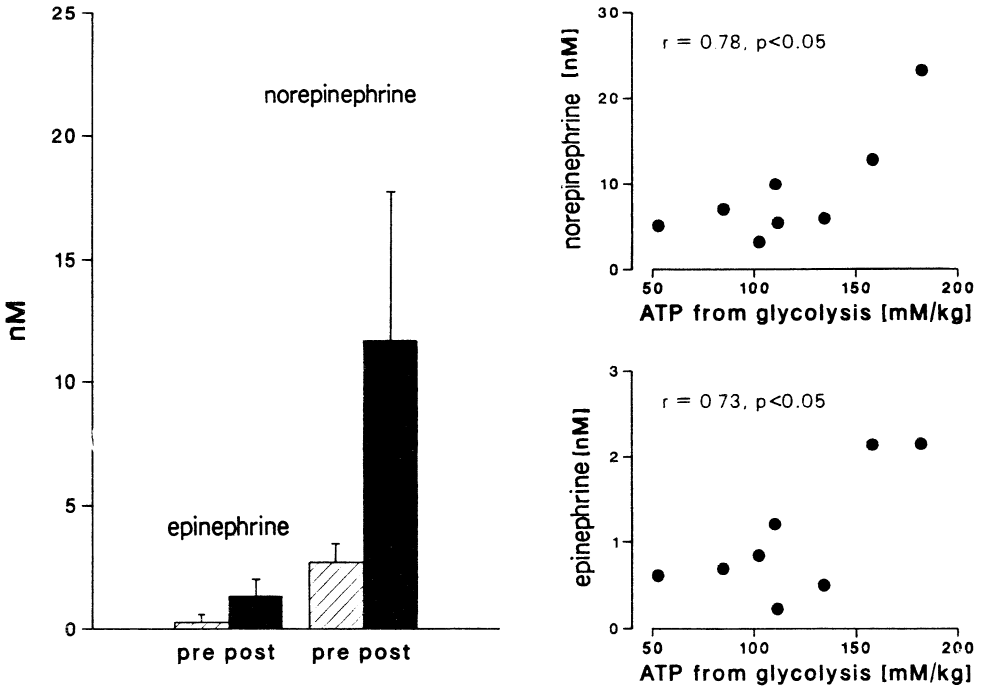

FIG. 2. Plasma epinephrine and norepinephrine concentrations (mean ± SD) at rest and 3 min after treadmill sprinting and relationship between changes in plasma catecholamines and energy produced from glycolysis during sprint.

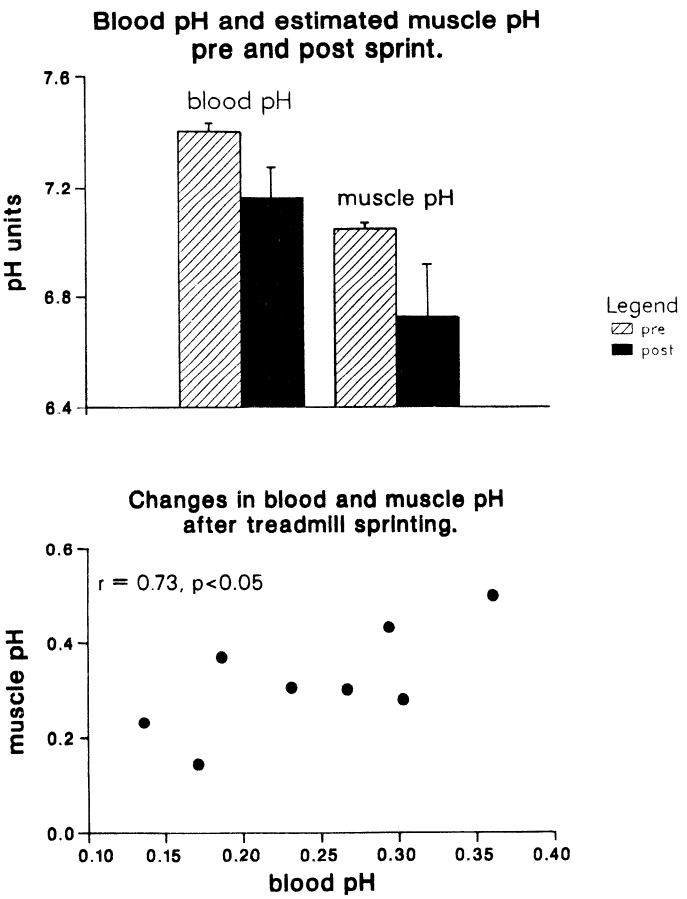

FIG. 3. Blood pH and estimated muscle pH (mean ± SD) (muscle pH, 0.00413 (lactate + pyruvate) + 7.06; Ref. 30) at rest and 3 min after treadmill sprinting and relationship found between changes in blood and estimated muscle pH.

individuals producing the greatest mean power output during the sprint also had the largest changes in plasma volume ($r = 0.95$, $P < 0.01$).

TABLE 3. *Blood lactate and blood glucose concentrations at rest, post warm-up, and at 3 and 5 min post-treadmill sprinting*

	Rest	Post Warm-up	Postexercise	
			3 min	5 min
Blood lactate	0.73±0.29	1.96±1.51	11.57±2.97	13.06±2.79
Blood glucose	4.37±0.46	4.94±0.50	5.68±0.71	6.13±0.84

Values are means ± SD in mmol/l.

DISCUSSION

The peak and mean power outputs generated by the subjects in this study are similar to the values reported previously for active female subjects performing this particular running test (5) and with the power outputs generated by female subjects during maximal sprint cycling tests (21). The treadmill sprint demanded a maximal effort from the subjects as reflected by the marked decrease in power output (50%), which occurred during the latter part of the test. Similar decreases in power output are experienced during maximal cycling tasks (34).

The maximal intensity of the test is also reflected by the marked decrease in muscle glycogen, which occurred as a result of the 30-s sprint. The average change in muscle glycogen between resting and postexercise values was 68.82 mmol/kg dry wt or 2.29 mmol·kg dry $wt^{-1} \cdot s^{-1}$. Because the warm-up was of short duration and submaximal in nature, resulting in only a 1 mmol/l increase in blood lactate concentration, its contribution to the decrease in glycogen concentration was probably quite small. Furthermore, the decrease in glycogen in the present study was similar to that observed after a maximal cycling test when the biopsy was taken after the warm-up (3). The observed glycogen decrease in the present study then, represents one of the highest rates of glyco-

gen degradation recorded for humans during dynamic exercise. Nevertheless, total glycogen was only reduced by 25%, which supports the findings of other authors that during exercise of maximal intensity and brief duration subjects become exhausted before glycogen depletion occurs (18). Therefore, unless there was substantial selective glycogen depletion in type II fibers, the depletion of this substrate is unlikely to have been the main cause of fatigue.

Attention then has been given to factors that may limit the muscles' ability to supply or use energy at maximal rates. An examination of the changes in ATP, the immediate mediator of energy for muscular contraction, is then of interest. Resting ATP levels in the present study were higher than have been reported using the needle-biopsy technique (15). This may be a result of a combination of rapid freezing of the samples, the efficient removal of fat and slight methodological variations which may exist between laboratories. However, the change in ATP during the sprint of 10.3 mmol/kg dry wt, or a decrease of 37%, is in close agreement to the changes found in previous studies following 30-s sprint cycling (3, 20). It has been suggested that such changes in ATP may be overestimated because of possible changes in the reference base, for example in local lipid and glycogen stores and in the increased blood content of the postexercise samples (16). In the present study, such changes were minimized by expressing metabolite concentrations with respect to dry weight and by the removal of fat and surface blood by ether extraction. Therefore the results of this study are consistent with the findings of other authors which show that there is a significant reduction in ATP during short-term maximal dynamic exercise (3, 4, 20, 32). It is, of course possible, as has been previously suggested (32), that an even greater reduction in ATP may occur in certain fiber types. The metabolic characteristics of the type II fibers would suggest that this subgroup may incur a greater reduction of ATP than type I fibers. In particular type II fibers have greater phosphofructokinase (PFK) and myosin adenosine triphosphatase (ATPase) activities, and the deamination of AMP to inosine-monophosphate (IMP), the means by which ATP depletion occurs, happens readily only in type II fibers (28). In fact, a recent study examining a small number of single fibers before and after isokinetic exercise has shown a greater reduction in ATP and increase in IMP in type II compared with type I fibers (22). A significant reduction in ATP may be a contributory factor to fatigue resulting in fewer cross-bridge attachments in all fibers and possibly causing a total failure of force production in some motor units or fibers (13).

A limitation to ATP production is obviously the availability of PCr. Unlike earlier studies examining PCr following dynamic exercise no total depletion was found (2). As it has been calculated that PCr may be restored at a rate of 2–3 mmol·kg dry $wt^{-1} \cdot s^{-1}$ following exercise with full circulation, it is possible that postexercise PCr was actually lower than the value observed 7 s after the sprint in the present study (14). However, it is worth noting that most of this time was spent with the subject slowing down and, therefore, still active. Also in comparison with the amount of lactate produced, the decrease in PCr was not as great as is observed after isometric or electrically induced contractions (19). As the creatine kinase reaction is in equilibrium with H^+ a possible explanation for the different lactate/PCr is a faster efflux of H^+ compared with lactate from the cell during dynamic exercise (30). However, PCr concentration was still reduced by 64% and it therefore seems apparent that ATP resynthesis would have been greatly reduced.

The relatively large changes in G1P, G6P, and F6P compared with the increases in FBP and TP point to PKF as the limiting factor in the glycolytic flux as has been suggested previously (30). Snow and colleagues (32) have recently reported an accumulation of hexosemonophosphates (HMP) of up to 30.6 mmol/kg dry wt in equine muscle after exhaustive exercise, which is similar to the mean value of 25.4 mmol/kg dry wt after the 30-s sprint in the present study. This large accumulation of HMP may be explained by a massive stimulation of glycogen phosphorylase relative to PFK and suggests that activation of glycogen phosphorylase may have been augmented by the release of catecholamines.

In the present study, plasma catecholamines were measured and found to increase to a considerable degree particularly when one considers that the exercise period was only 30 s. Although glycogenolysis in skeletal muscle is increased in the presence of elevated plasma epinephrine concentrations, the quantitative nature of this relationship is, as yet, unknown. Nevertheless, it is not unreasonable to suggest that the correlation found, in the present study, between plasma catecholamines and rate of energy production from glycolysis reflects a contributory influence of epinephrine on muscle glycogenolysis. Similiarly, the correlation found between the blood glucose response to the sprint and plasma catecholamine concentration seems likely to reflect the influence of catecholamines on hepatic glycogenolysis.

The increases in muscle lactate following the treadmill sprint were similar to the increases reported previously for male subjects (3, 4, 21) but were greater than those recorded for female subjects after maximal cycle ergometer exercise (20, 21). Thus there is no support in the present study for the suggestion that females may accumulate less intramuscular lactate than males (21). Whereas it has been previously demonstrated that at submaximal exercise intensities there is a linear relationship between lactate release into the circulation and muscle lactate content (23), the relationship between muscle and blood lactate concentrations after brief maximal exercise is unknown. Blood lactate concentration is thought to peak at ~5 min following this type of exercise (34). In the present study the significant correlation ($r = 0.82$, $P < 0.05$) found between blood lactate concentration at 5 min following the sprint and muscle lactate at the end of the sprint would suggest, for this group of individuals at least, that the blood lactate concentration gives some indication of the energy supplied by anaerobic glycolysis during this particular exercise task.

The changes in blood pH and estimated muscle pH were similar to those reported by other authors after, for

example, 400-m sprinting and 60 s of treadmill running or cycling (7, 17). The estimated postexercise muscle pH of 6.73 units would, in vitro, inhibit PFK activity (8). However, the high F6P levels in the present study may, to some extent, overcome this inhibition. This would appear to be the case during electrically induced isometric contractions where it has been demonstrated that the glycolytic rate is not reduced even after 50 s of stimulation and when the increase in muscle lactate was similar to that found during the present study (19). A more important consequence of the decrease in pH may be in affecting the muscle contractile mechanism itself, by decreasing the energy available for contraction per ATP hydrolyzed (12).

It has previously been shown, by animal experiments and more recently in examining human muscle during electrically induced contractions, that ATP turnover is directly related to the force developed (10, 19). Such a relationship has been suggested (34) but not conclusively shown during dynamic exercise. In the present study, much higher correlations were found between the changes in blood metabolites and performance than between the changes in muscle metabolites and performance, although there was a trend for peak running speed on the treadmill to be related to the estimated rate of ATP turnover ($r = 0.70$, NS). It may be that the changes in blood lactate and blood pH and the decrease in plasma volume are an indication of the muscle mass recruited during the sprint. Furthermore, perhaps a significant relationship between ATP turnover and performance during dynamic exercise should not be expected as the estimation of ATP turnover takes no account of the ATP supplied by oxidative metabolism which may vary among individuals. Also it has recently been demonstrated that trained muscle may be more economical in its use of ATP, i.e., have a greater force production per ATP hydrolyzed (Westra, personal communication). Therefore individual variations in training status may also complicate the relationship between performance and estimated rates of ATP turnover.

In summary, the study has demonstrated that the metabolic responses to sprint running can be satisfactorily examined in the laboratory by using a 30-s maximal running test on a nonmotorized treadmill. Furthermore, the metabolic changes were at least equal to, if not greater than, those changes previously observed for female subjects following maximal cycle-ergometer exercise. Examination of sprint running performance in this way may thus provide a means by which the limitations of performance and causes of fatigue in maximal exercise may be examined.

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