Lactate transport activity in rat skeletal muscle sarcolemmal vesicles after acute exhaustive exercise

H. Dubouchaud, N. Eydoux, P. Granier, C. Préfaud, and J. Mercier. Lactate transport activity in rat skeletal muscle sarcolemmal vesicles after acute exhaustive exercise. J. Appl. Physiol. 87(3): 955–961, 1999.—The effect of a single bout of exhaustive exercise on muscle lactate transport capacity was studied in rat skeletal muscle sarcolemmal (SL) vesicles. Rats were assigned to a control (C) group (n = 14) or an acutely exercised (E) group (n = 20). Exercise consisted of treadmill running (25 m/min, 10% grade) to exhaustion. SL vesicles purified from C and E rats were sealed because of sensitivity to osmotic forces. The time course of 1 mM lactate uptake in zero-trans conditions showed that the equilibrium level in the E group was significantly lower than in the C group (P < 0.05). The initial rate of 1 mM lactate uptake decreased significantly from 2.44 ± 0.22 to 1.03 ± 0.08 nmol·min⁻¹·mg protein⁻¹ (P < 0.05) after exercise, whereas that of 50 mM lactate uptake did not differ significantly between the two groups. For 100 mM external lactate concentration ([lactate]), exhaustive exercise increased initial rates of lactate uptake (219.6 ± 36.3 to 465.4 ± 80.2 nmol·min⁻¹·mg protein⁻¹, P < 0.05). Although saturation kinetics were observed in the C group with a maximal transport velocity of 233 nmol·min⁻¹·mg protein⁻¹ and a Michaelis-Menten constant of 24.5 mM, saturation properties were not seen after exhaustive exercise in the E group, because initial rates of lactate uptake increased linearly with external [lactate]. We conclude that a single bout of exhaustive exercise significantly modified SL lactate transport activity, resulting in a decrease in 1 mM lactate uptake and was associated with alterations in the saturable properties at [lactate] above 50 mM. These results suggest that changes in sarcolemmal lactate transport activity may alter lactate and proton exchanges after exhaustive exercise.

LACTATE IS NOW RECOGNIZED to be an important source of energy for skeletal muscle metabolism, as described in the lactate shuttle theory (5), instead of a simple metabolic end product. For many years, it was thought that L-(-)-lactate simply diffused across the sarcolemmal membrane. However, as reviewed by Poole and Halestrap (26), studies of different cell types have established that lactate uptake and release are mediated via membrane-bound carrier proteins. In skeletal muscles, the existence of such a membrane-bound lactate transporter has been clearly demonstrated by using different models of purified sarcolemmal vesicles (14, 20, 28, 29). It has been shown that the sarcolemmal lactate transporter is stereoselective for L-(-)-lactate and is temperature and pH sensitive (28, 29). This latter property is directly related to the mechanism of lactate transport, which seems to occur as lactate-H⁺ cotransport. Therefore, the sarcosomal lactate carrier appears to be of great importance in carbohydrate distribution and regulation of intracellular pH (5, 15). Thus an understanding of how the regulation of this lactate transporter occurs in different physiological conditions is fundamental for exercise physiology. Lactate transport activity appears very closely related to muscular activity because muscle denervation causes a decrease in lactate transport activity in sarcolemmal vesicles (18, 24). Recently, we were able to avoid the problems involved in removing nervous input after denervation by using muscle hypodynamia induced by hindlimb suspension (7). With this model, we also observed a decrease in lactate transport activity. These studies have demonstrated changes in lactate transport associated with decreased muscle activity. In contrast, some authors reported a stimulatory effect of moderate- and high-intensity training on lactate transport activity in sarcolemmal vesicles (21, 25), whereas others did not confirm these results (30).

The effect of acute exercise has not been extensively studied. There is no difference in lactate transport by isolated rat skeletal muscle previously electrically stimulated to contract (22, 32). Experiments in isolated mouse muscles showed a small increase in lactate transport after treadmill running (2). These conflicting results point up the need for complementary experiments to determine the effects of acute exercise on the membrane lactate transport in muscles. We already know that a single bout of exhaustive exercise causes important modifications in lactate exchanges and that its effects are numerous on muscle cells. For instance, studies have shown some negative effects such as membrane alterations (6), inhibition of some glycolytic enzymes (17), and modifications of structural proteins, and also translocation of the glucose transporter (11). Given these findings, we hypothesized that lactate transport activity in skeletal muscle could be altered by a single bout of exhaustive exercise. To test this hypothesis, we investigated the effect of prior exhaustive treadmill running on the lactate uptake by skeletal muscle sarcolemmal vesicles in rats.

MATERIALS AND METHODS

Animals. Three-month-old male Wistar rats were randomly assigned to either a control (C) group (n = 14) or an exercise (E) group (n = 20). They were housed in individual cages with food and drink available ad libitum, with a 12:12-h light-dark cycle (lights on at 7 PM), allowing exercise during their active phase. All animals were familiarized with treadmill running for 2 wk, 5 min/day. After familiarization, rats from the E group performed treadmill running up a 10% grade at 25 m/min until exhaustion, which was defined as the point at which the animals could no longer right themselves.
when placed on their back. Immediately after the end of running, rats were killed by cervical dislocation.

Reagents. Reagents with the highest quality available were purchased from Sigma Chemical, unless stated otherwise. Muscle glycogen content. Measurements were made according to the method of Lo et al. (16). Muscle portions (30–50 mg) of predominantly red gastrocnemius and red vastus lateralis were boiled in 30% KOH saturated with Na2SO4 until homogenization (usually 30 min). Homogenates were kept on ice, and glycogen was precipitated by addition of 1.2 vol of 95% ethanol. Samples were centrifuged for 30 min at 840 g, and pellets were resuspended in H2O. Assays were conducted on aliquots against appropriate blanks at 490 nm. Results were determined from a standard curve generated at the same time and expressed in milligrams of glycogen per gram of tissue.

Sarcolemmal isolation and characterization. The muscles from one rat from the C group and one rat from the E group were processed on the same day of the experiment. Sarcolemmal vesicles were purified from hindlimb muscles with a procedure already established in our laboratory (7, 10). All subsequent steps were carried out at 4°C. After elimination of fatty, nervous, and connective tissues, muscles (usually 15–20 g) were homogenized in ice-cold 250 mM sucrose, 1 mM EDTA, and 20 mM HEPES, pH 7.4, at 25°C with two bursts (2 × 5 s) of Ultra-Turrax T25 at 80% of maximal power. The homogenate was centrifuged twice at 900 g in a Sorvall RC-28S with a SA-600 rotor. Supernatants were filtered, and a 1-ml aliquot was partitioned and saved at 4°C for subsequent analysis. The remaining crude homogenate (CH) was diluted with a volume of KCl medium (3 M KCl and 250 mM sodium pyrophosphate, pH 7.4) equal to 10% of the CH volume and pelleted by ultracentrifugation in a Beckman 60 Ti rotor (200,000 g; 45 min at 4°C). Pellets were resuspended by hand using Teflon pestle homogenization in 30 ml of sucrose medium. The suspension was centrifuged twice at 280 g, and supernatants were then collected and centrifuged (200,000 g; 45 min at 4°C). Pellets were homogenized by using a glass tissue homogenizer in 7–8 ml of 40% (wt/vol) sucrose. A discontinuous density gradient was constructed by addition of 8 ml of the following solutions of sucrose: 38, 32, 27, and 12% in layers. After an overnight centrifugation (15–17 h) at 130,000 g at 4°C in a SW 27 rotor, the 27% sucrose band (sarcolemmal vesicles) was harvested and diluted with a Krebs-Ringer-HEPES (KRH) buffer [containing (in mM) 118 NaCl, 5 KCl, 1.2 MgSO4, and 50 HEPES, pH 7.5 at 25°C] and washed free of sucrose in a 60 Ti rotor (200,000 g for 80 min at 4°C). The vesicles were resuspended in KRH buffer up to 4 mg/ml and stored at −80°C until use in the transport experiments. Proteins were determined according to the procedure of Bradford (4) by using bovine gamma globulin as a standard. Sarcolemmal characterization was achieved with the K+-stimulated p-nitrophenylphosphatase (K+-pNPPase) assay as described previously (7, 10). Total activity was measured in 40 mM HEPES, 0.8 mM EGTA, 4 mM MgCl2, 20 mM KCl, and 5 mM p-nitrophenylphosphatase, pH 7.4. The absorbance of the p-nitrophenol formed was read at 410 nm. Nonspecific K+-pNPPase activity was determined in a KCl-free medium, which, when subtracted from the total activity, gave the specific K+-pNPPase activity, expressed in micromoles per hour per milligram. The purification index was defined as the ratio of the specific activity from the sarcolemmal vesicle fraction to the specific activity measured in the CH. Skeletal muscle sarcolemmal yield was the ratio of milligrams of sarcolemmal proteins obtained in sarcolemmal vesicles to the muscle weight in grams after the trimming process (wet wt). In a set of experiments, the vesicle “side-ness” was estimated by measurement of the K+-pNPPase activity with and without a previous 2 mM deoxycholate incubation. A previous incubation in deoxycholate has been used to make all vesicles permeable and reveal K+-pNPPase activity in both right-side-out and inside-out vesicles (28). The percentage of vesicles in the native orientation was then estimated by the ratio of K+-pNPPase activity in normally treated vesicles to the K+-pNPPase activity measured after deoxycholate incubation.

Lactate transport studies. All measurements were performed in zero-trans conditions, in duplicate, on three to five different membrane preparations. L-(-)-[U14C]lactate (Amersham, specific activity 155 mCi/mmol) was diluted (1:500 to 1:1,000) in 280 mM sucrose and 50 mM HEPES, pH 7.4, with different unlabeled L-(-)-lactate concentrations. Reciprocal decreases in sucrose were used to maintain the same total isosmotic buffer strength. Uptake of 1 mM L-(-)-lactate was measured at pH 7.4 without a pH gradient. Reactions were initiated by delivering 50 µg of sarcolemmal vesicles into tracer-containing medium and stopped at appropriate time intervals by vacuum filtration on nitrocellulose filters (Whatman WCN, average pore size of 0.45 µm). Filters were then rinsed three times with an ice-cold isosmotic stop solution consisting of KRH buffer with 3 mM HgCl2, pH 7.4, at 25°C, and dissolved with 600 µl of ethylene glycol monomethyl ether. The radioactivity accumulated in vesicles was counted in a scintillation analyzer (Packard 2200 CA). Nonisotropic transport activities (usually <100 counts/min) were determined by preincubation of vesicles in tracer-containing medium with stop solution. Transport activities were corrected for this nonspecific activity, and results were expressed in nanomoles per milligram protein. Saturation kinetics were obtained by measuring the initial lactate uptake of 1, 5, 10, 20, 30, 50, 65, 75, and 100 mM external lactate concentrations at 0, 5, and 10 s. Slopes determined by computer linear regression analysis, using the least squares fit of data, gave initial rates of lactate uptake expressed in nanomoles of lactate per milligram protein per minute.

Vesicle sensitivity to osmotic forces. The effect of medium osmolality on L-(-)-lactate uptake at equilibrium in sarcolemmal vesicles was determined by using different external media containing 1 mM L-(-)-lactate and various sucrose concentrations: 150, 200, 250, 300, or 400 mM in 50 mM Tris, pH 7.4. Intravascular medium consisted of 250 mM sucrose in 50 mM Tris, pH 7.4. Equilibrium L-(-)-lactate uptake was determined after 3-min incubations, and results were expressed in nanomoles per milligram protein per 3 min.

Statistical analysis. Results are expressed as means ± SE. Statistical significance was assessed by Student’s t-test to examine the effect of exercise on the variables studied. Analysis of variance was used to show differences in 1 mM lactate uptake between C and E groups as a function of time and as a function of osmotic pressure. A probability level of P < 0.05 was used throughout the study.

RESULTS

Body weight, muscle glycogen content, and exercise. Body weights reported in Table 1 show that there was no difference between the C and E groups. Exhaustion occurred after 72 ± 19 min of treadmill running up a 10% grade at 25 m/min. This exercise significantly reduced muscle glycogen contents by 66% in red gastrocnemius and 64% in red vastus lateralis in the E group compared with the C group (P < 0.05) (Fig. 1).
Table 1. Morphological and biochemical parameters in control and exercise groups

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Weight, g</th>
<th>Proteins in CH, mg/g</th>
<th>Proteins in SL, mg/g</th>
<th>PI K^-pNPPase</th>
<th>PI K^-pNPPase + DOC</th>
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<tr>
<td>Control</td>
<td>14</td>
<td>356 ± 15</td>
<td>140.1 ± 7.5</td>
<td>0.28 ± 0.03</td>
<td>18.4 ± 1.9</td>
<td>25.7 ± 5.6</td>
</tr>
<tr>
<td>Exercise</td>
<td>14</td>
<td>352 ± 20</td>
<td>131.5 ± 10.1</td>
<td>0.30 ± 0.08</td>
<td>20.7 ± 5.3</td>
<td>34.3 ± 13.5</td>
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Values are means ± SE. n, No. of rats; CH, crude homogenate; PI, purification index; ratio of the specific activity from the sarcolemmal (SL) fraction to the specific activity measured in CH. PI K^-pNPPase and PI K^-pNPPase + DOC, PIs of sarcolemmal enzyme K^-p-nitrophenylphosphatase without and with preincubation in 2 mM deoxycholate, respectively.

Characterization of sarcolemmal vesicles. Using gradient density fractionation, we obtained sarcolemmal vesicle preparations with similar biochemical characteristics in the two groups. Protein yields were 140.1 and 131.5 mg of protein/g (wet wt), and purification indexes of the K^-pNPPase were 18.4 and 20.7 for the C and E groups, respectively (Table 1). Purification indexes of the K^-pNPPase with and without 2 mM deoxycholate suggest that right-side-out vesicles represent 65% of the total vesicle population in the C group and 68% in the E group (Table 1).

Effect of osmotic forces. Figure 2 shows the effect of osmolarity on 1 mM L-(+)-lactate uptake at equilibrium in sarcolemmal vesicles from the C and E groups. Decreasing the intravesicular space induced by higher external sucrose concentration results in less L-(+)-lactate accumulation in vesicles from the two groups at pH 7.4. Moreover, lactate uptake at equilibrium was significantly higher in the C group compared with the E group (Table 1). Purification indexes of the K^-pNPPase with external lactate concentrations from 0 to 100 mM. Figure 4 shows the initial rates of lactate uptake for 1, 50, and 100 mM external lactate concentrations in the two groups. After exhaustive exercise, 1 mM lactate uptake was significantly decreased in the E group compared with the C group (2.44 ± 0.22 vs. 1.03 ± 0.08 nmol·mg protein^-1·min^-1, P < 0.05). There was no significant difference in the initial rates of lactate uptake for 5, 10, 30, or 50 mM external lactate concentrations. For 100 mM external lactate concentration, exhaustive exercise resulted in a higher initial rate compared with control conditions (465.4 ± 80.2 vs. 219.6 ± 36.3 nmol·mg protein^-1·min^-1, P < 0.05).

DISCUSSION

We show that an acute exhaustive exercise bout alters the lactate transport activity in rat skeletal muscle sarcolemmal vesicles. Specifically, there is a decrease in the initial rate of lactate uptake of 1 mM external lactate concentration and an apparent loss of saturable properties of lactate transport at high external lactate concentrations.

After exhaustive exercise by rats, we observed significant decreases in red gastrocnemius and red vastus lateralis glycogen concentrations. These glycogen reduc-

![Graph](image)

**Fig. 1.** Muscle glycogen contents in red gastrocnemius (RG) and in red vastus lateralis (RV) from control (filled bars) and exercise (open bars) rats. Values are means ± SE; n = 5 animals. *Significantly different from control muscle, P < 0.05.

![Graph](image)

**Fig. 2.** Effect of medium osmolarity on L-(+)-lactate uptake into vesicles from control (●) and exercised (○) rats. Values are means ± SE of 4 different membrane preparations. Assays were performed in duplicate at pH 7.4 with external sucrose concentrations adjusted to give indicated osmolalities. *Significantly different from control, P < 0.05.
tions have been reported elsewhere and constitute a common parameter used to confirm the exhaustive character of the exercise protocol studied (8). Glycogen concentrations were measured in portions of red gatroc- nemius and red vastus lateralis muscles rather than from a mixture of all hindlimb muscles for consistency between experiments. The respective sizes of the above-mentioned muscles allowed us to take the same portions each time. Moreover, the sucrose buffer used to purify the sarcolemmal vesicles could interfere with the glycogen assay procedure.

Using sucrose density gradient fractionation, we obtained sarcolemmal vesicles enriched with the sarcolemmal marker K⁺-pNPPase, with purification indexes similar to those reported by others (20). Several lines of evidence, like the presence of GLUT-1 glucose transporter in sarcolemmal fractions from skeletal muscles (13), support the view that this model is representative of the native plasma membrane. Despite the widely recognized problem of the low sarcolemmal recovery from skeletal muscle, isolated sarcolemmal vesicles constitute a nonmetabolic system that allows detailed studies of membrane properties. Such studies are complementary to those conducted on other models like perfused muscle preparations (2, 22, 32, 33). Measurements of K⁺-pNPPase with and without deoxycholate indicate that proportions of right-side-out vesicles were also similar to those of previous studies (20, 28). Moreover, our results showed that exhaustive exercise had no significant effect on the orientation of the purified vesicles. Previous studies from our laboratory using the same sarcolemmal purification procedure have shown that these purified vesicles have detectable lactate transport properties that are sensitive to pH and the monocarboxylate transport inhibitor α-cyano-4-hydroxycinnamate and present some trans-stimulatory properties at high external L-(+)-lactate concentration (7, 10).

In this study, vesicles from the two groups appeared to be sealed because they showed sensitivity to changes in osmotic conditions. In equilibrium conditions, sarcolemmal vesicles from the E group also showed reduced lactate accumulations for every osmotic condition employed. This suggests that exhaustive exercise could either interact with the sarcolemma isolation procedure or induce some membrane alterations that might perturb the lactate transport properties or contribute to membrane fragility, leading to a decrease in lactate accumulation. Because the protein yields and the purification indexes were similar in both groups, it is unlikely that the exhaustive exercise could have affected the purification. In fact, exhaustive exercise is known to increase free radical concentrations and levels of lipid peroxidation responsible for membrane alterations that could contribute to this loss of membrane integrity (6). On the other hand, less accumulation of lactate in the equilibrium condition could also indicate that vesicles from the E group are smaller than those of the C group. It has been reported that, when uptake measurements are made in initial rate conditions, uptake is dependent only on transporter activity, unlike what occurs in equilibrium conditions, where the amount of accumulated substrate in the vesicles is dependent on both transporter activity and vesicle volume (12). Therefore, because we made the measurements in initial rate conditions, such heterogeneity in the vesicle size, if any, would have only minor consequences in our lactate uptake comparisons between the two groups.

The acute exhaustive exercise resulted in a decrease in the initial rate of lactate uptake in our model at 1 mM external lactate concentration (~57%), confirming our results about the effect of the osmotic pressure. We also observed an increase in the lactate transport at

**Fig. 3.** Time course and kinetics of lactate uptake. A: time course of 1 mM L-(+)-lactate uptake into vesicles from control (○) and exercised (○) rats. Values are means ± SE of 5 different membrane preparations. All assays were performed in duplicate in buffered sucrose at pH 7.4 on both sides of membranes. B: kinetics of initial rate of lactate uptake into vesicles from control (○) and exercised (○) rats at various external lactate concentrations. Values are means ± SE of assays, which were performed in duplicate at pH 7.4 on at least 3 different membrane preparations. *Significantly different from control, P < 0.05.
high external lactate concentration (+112%). This increase was associated with a loss-of-saturation curve. This phenomenon was observed in a previous set of experiments with external lactate concentrations of 1, 5, 10, 30, 50, and 100 mM. We confirmed this loss of saturation after exercise by additional experiments in the E group at external lactate concentrations of 20, 65, and 75 mM. The increase in lactate uptake in sarcolemmal vesicles of the E group appears to be independent of lactate transporter activity, but rather is related to an increase in passive diffusion, although no experiment with lactate transport inhibitor was made to specifically measure this. Moreover, as the external lactate concentration increases, the proportion of lactate accumulated by passive diffusion in relation to total uptake increases, resulting in a larger accumulation of lactate in the vesicles. Some studies have shown that this proportion varies from 10% for 1 mM external lactate concentration to >30% for 50 mM external lactate concentration (14, 28, 29). No data are available for higher external lactate concentrations. In our study, we can assume that this proportion is higher in the E group because of some deleterious effect of the exhaustive exercise that leads to an amplification of the difference between results in the two groups.

By using a model of incubated muscle strips (22) or a perfused rat hindlimb model (32), it has been shown that acute periods of muscle activity induced by electrical stimulation do not alter lactate transporter activity. Our findings of a decrease in 1 mM lactate uptake contradict the absence of modifications reported elsewhere (22, 32). Although the model of incubated muscle strips has been described as being comparable to the model of the sarcolemmal vesicles (22), we cannot exclude that, in the same conditions as previously described (22, 32), sarcolemmal vesicles would have shown different results because of the elimination of compartmental barriers, cellular metabolism, and metabolite concentrations that may obviate any specific membrane alterations. However, a small but significant increase in lactate transport has been reported in the mouse after a single bout of treadmill running (2). We believe this significant increase could be due to the difficulty in controlling both sides of the sarcolemmal membrane accurately and to the internal metabolism of the experimental model used in the study (2). Our results are therefore in accordance with these latter findings because we observed a tendency toward elevated lactate uptake in the E group at external lactate concentration of 30 and 50 mM. As the mouse study was conducted only at 4 and 30 mM external lactate concentration, the same comparison is not possible for the higher external lactate concentrations used in our study. Despite its usefulness for studying the effect of contractile activity on cellular responses, electrical stimulation does not mimic true physical activity or exercise (3). The low glycogen concentrations that we measured in our study (−65%) also suggest that the exercise protocol we used was of higher intensity compared with those in other studies (2). Moreover, in the mouse study, the authors have considered only the soleus and the extensor digitorum longus muscles, which are not fully involved in running (2). Because we used a mixture of all hindlimb muscles, our results may reflect the global modifications occurring after exhaustive exercise.

Data from the literature show that the regulation of lactate carrier activity is quite different from that occurring with other transporters, such as the glucose transporter (11). For example, McDermott and Bonen (22) reported that there is a 47% increase in 2-deoxy-D-glucose uptake after electrical stimulation of soleus muscle strips, whereas there is no apparent effect on lactate uptake. It is therefore unlikely that the lactate transporter is sensitive to acute regulation. Our results could be explained by a loss or inactivation of lactate transporters associated with an increase in the passive diffusion at high external lactate concentrations. Thus, as 90% of the lactate exchanges at low external lactate concentrations depend on lactate transporter activity (14, 28), 1 mM lactate uptake would therefore be reduced after acute exhaustive exercise, whereas, because of the increase in passive diffusion, lactate influx would be enhanced at external lactate concentrations above 50 mM.

The physiological implications of such modifications in lactate exchanges in sarcolemmal vesicles after acute exhaustive exercise are still unclear. For lactate
concentrations ranging from 5 to 50 mM, acute exhaustive exercise does not seem to have major consequences because total lactate uptake is similar in both groups. For lower concentrations, a direct consequence could be an impairment in the availability of lactate as a substrate for oxidative muscle during recovery, which affects muscle metabolism, because we know that lactate can be used by oxidative fibers (5). Also, as the lactate transporter is involved in pH regulation during exercise and recovery (15), our study suggests that modifications in lactate exchanges after exhaustive exercise can affect cellular pH recovery. Our purpose was to study the effect of acute exhaustive exercise on lactate transport immediately after the end of exercise.

We cannot rule out that acute exhaustive exercise would have induced more significant changes several hours after the end of exercise. It has been reported recently that lactate transport in perfused rat skeletal muscle is significantly reduced 2 days after unaccustomed eccentric contractions induced by electrical stimulations (23).

The saturation curve obtained for the C group looks like a sigmoidal relationship that could reflect the combination of the activities of two transporter isoforms with different kinetic properties. Such a mechanism has been suggested previously in heart cells (31), although the kinetic parameters of the two isoforms were not fully elucidated. Today, a thorough understanding of the mechanisms of these modifications is impossible because we have only a limited knowledge of the molecular structure of the transport protein (1, 34). A gene encoding for a monocarboxylate transporter that corresponds well with the identified erythrocyte monocarboxylate transporter has been cloned (9). This transporter, called monocarboxylate transporter 1 (MCT1), is expressed predominantly in oxidative muscle, but other possible isoforms are waiting to be discovered (19). Recently, a new isoform, MCT3 (now called MCT4), has been cloned and sequenced (27) and appears to be involved in lactic acid efflux from skeletal muscle (35). Further studies in this area should allow us to elucidate the underlying mechanisms of lactate transporter activity after acute exhaustive exercise.

In summary, this study reports a decrease in the initial rate of lactate uptake at 1 mM external lactate concentration and a loss of saturable kinetic properties in sarcolemmal vesicles isolated immediately after acute exhaustive exercise. These results indicate that acute exhaustive exercise alters sarcolemmal lactate transport properties in rat skeletal muscles and confirm that the lactate transporter is not sensitive to specific acute regulation.

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