Effect of prior eccentric contractions on lactate/H⁺ transport in rat skeletal muscle

HENRIETTE PILEGAARD AND SVEN ASP
Copenhagen Muscle Research Centre, August Krogh Institute, University of Copenhagen, 2100 Copenhagen, Denmark

Pilegaard, Henriette, and Sven Asp. Effect of prior eccentric contractions on lactate/H⁺ transport in rat skeletal muscle. Am. J. Physiol. 274 (Endocrinol. Metab. 37): E554–E559, 1998.—The effect of prior eccentric contractions on skeletal muscle lactate/H⁺ transport was investigated in rats. Lactate transport was measured in sarcolemmal giant vesicles obtained from soleus and red (RG) and white gastrocnemii (WG) muscles 2 days after intense eccentric contractions (ECC) and from the corresponding contralateral control (CON) muscles. The physiochemical buffer capacity was determined in the three muscle types from both ECC and CON legs. Furthermore, the effect of prior eccentric contractions on release and muscle content of lactate and H⁺ during and after supramaximal stimulation was examined using the perfused rat hindlimb preparation. The lactate transport rate was lower (P < 0.05) in vesicles obtained from ECC-WG (29%) and ECC-RG (13%) than in vesicles from the CON muscles. The physiochemical buffer capacity was reduced (P < 0.05) in ECC-WG (13%) and ECC-RG (9%) compared with the corresponding CON muscles. There were only marginal effects on the soleus muscle. Muscle lactate concentrations and release of lactate during recovery from intense isometric contractions were lower (P < 0.05) in ECC than in CON hindlimbs, indicating decreased anaerobic glycogenolysis. In conclusion, the sarcolemmal lactate/H⁺ transport capacity and the physiochemical buffer capacity were reduced in prior eccentrically stimulated WG and RG in rats, suggesting that muscle pH regulation may be impaired after unaccustomed eccentric exercise. In addition, the data indicate that the glycolgenolytic potential is decreased in muscles exposed to prior eccentric contractions.

METHODS

Animals

Male Wistar rats with an average body weight of 247 ± 2 (SE) g were housed two per cage and were kept in a room (24°–26°C) with a dark-light cycle of 12:12 h. The rats were allowed to eat standard chow diet (Altromin no. 1324, C. Petersen, Ringsted, Denmark) and drink water ad libitum.

The experiments were approved by the “Danish Animal Experiments Inspectorate” and complied with the “European Convention for the Protection of Vertebrate Animals Used for Experiments and Other Scientific Purposes” (Council of European 123, Strasbourg 1985).

Eccentric Stimulation

Rats were anesthetized by an intraperitoneal injection of midazolam (Dormicum, 0.5 µg/kg body weight; Roche, Basel, Switzerland) and fentanyl (Hynorm, 20 µg/kg body weight) and fluoanison (1 mg/kg body weight, Janssen, Saunderton High Wycombe, Buckinghamshire, UK), and the calf muscles on one side were stimulated for eccentric contractions (ECC), as previously described (5). Briefly, muscles were subjected to 4 × 10 eccentric contractions of 1-s duration separated by 4 s and with 1 min of rest between each of the four series. Stimulation sessions were carried out between 8 and 12 AM, and after recovery from anesthesia the gait of the rats appeared normal. It has previously been shown that this eccentric stimulation protocol induces accumulation of inflammatory cells in both gastrocnemii muscles but without major changes in the soleus muscle (5). In all rats prepared for vesicle measurements, the muscles of only one side were stimulated for Ecc, so that the contralateral muscles could act as unstimulated controls (CON). For one-half of the rats prepared for hindlimb perfusion, the muscles of only one side were stimulated eccentrically, whereas the calf muscles of both legs were stimulated for eccentric contractions in the other one-half of the rats used for perfusion.

We decided to perform the vesicle experiments and the hindlimb perfusions 2 days after the eccentric contractions, as well as reduced (7, 19, 27) muscle activity. On the basis of these notions, it may be speculated that prior eccentric exercise also will lower the sarcolemmal lactate/H⁺ transport capacity and hence result in a reduced ability of the muscles to release lactate and H⁺.

Therefore, the main purpose of the present study was to examine the effect of prior eccentric contractions on sarcolemmal lactate/H⁺ transport capacity in rat skeletal muscle. Furthermore, we wanted to investigate the release of lactate and H⁺ associated with intense isometric contractions of rat muscle exposed to prior eccentric stimulation. Calf muscles were stimulated electrically for eccentric contractions by use of a model developed in our laboratory (5), and the sarcolemmal lactate transport was measured in giant vesicles, whereas the perfused rat hindlimb preparation was used for in situ determinations of lactate and H⁺ release.

ECCENTRIC CONTRACTIONS involve forced lengthening of active muscle and are included in many forms of exercise. Unaccustomed eccentric exercise is known to cause release of muscle enzymes from muscles to plasma (1, 23) and ultrastructural muscle damage (1), as well as performance decrements (6). Furthermore, it has been demonstrated that this type of muscle activity results in a marked decrease in the content of muscle glucose transporter proteins (GLUT-4) in rats (5, 15) and humans (4), whereas other muscle proteins are unaffected (5). Because eccentric contractions have a pronounced effect on the plasma membrane, it may be that other sarcolemmal transport proteins also are affected. The lactate/H⁺ carriers are membrane transport systems that mediate the main part of the sarcolemmal lactate flux (11, 13, 30, 34) and that possess the highest capacity for H⁺ membrane transport (12). Moreover, it has been shown that the lactate/H⁺ transport capacity is capable of adapting to enhanced (20, 22, 28) as well as reduced (7, 19, 27) muscle activity. On the
because it has previously been reported that the total GLUT-4 content was affected the most and the glycogen concentration remained subnormal in the eccentrically stimulated muscles at this stage (5). Moreover, we wanted to avoid any acute effects of eccentric contractions and ascertain that the rats had recovered fully after the anesthesia.

**Giant Sarcolemmal Vesicles**

Lactate transport. Two days after eccentric contractions, giant vesicles were prepared from eccentric and control muscles as previously described (11, 13). Marker enzyme analyses have revealed that the vesicles are predominantly of sarcolemmal origin (28), and the low concentration of nitrendipine binding sites in the giant vesicles demonstrated that T tubule membranes are not major contaminants in the preparation (35). Moreover, ouabain labeling of the sodium-potassium pumps confirmed results from patch-clamp studies showing that the orientation of the giant vesicles is right side out (28). Muscle samples were obtained from the white (WG) and red (RG) gastrocnemius muscle, consisting primarily of fast-twitch glycolytic and fast-twitch oxidative glycolytic fibers, respectively, as well as from the soleus muscle, containing mainly slow-twitch oxidative fibers (2). The different muscle types were kept and treated separately, and muscles from four rats were pooled to obtain a sufficient amount of muscle tissue.

The rate of sarcolemmal lactate transport was determined as previously described (11, 13). Briefly, incubation of fresh muscles with collagenase resulted in spontaneous production of sarcolemmal giant vesicles, and by use of a three-layer step-density gradient followed by mild centrifugation, the vesicles formed a band between the two upper layers. The band was collected, and after isolation of the vesicles by centrifugation, the vesicles formed a band between the two upper layers. The band was collected, and after isolation of the vesicles by centrifugation, they were preincubated with L-[3H]lactate, 30 mM unlabeled lactate, and [14C]sucrose at pH 7.4. The [14C]sucrose was used as an extravesicular marker. The tracer efflux started when loaded vesicles were transferred to a medium containing 30 mM unlabeled lactate at pH 7.4. The 30 mM concentration was chosen because it is in good agreement with the muscle concentrations obtained in the perfused rat hindlimb preparation during supramaximal electrical stimulation as well as during high-intensity exercise (2). The free sample obtained from the efflux medium using a syringe mounted with a 0.22-mm filter, and the 3H and 14C activities were determined with a Tri-Carb 2000CA liquid scintillation counter. On the basis of previous observations (11, 13), it was assumed that equilibrium was established at the time of the last sample (12 min), and the experimental data were therefore normalized in relation to the last sample. The diameters (range 2–26 mm) of >2,000 vesicles were measured by phase contrast microscopy, and the median diameter of the vesicles was 4.0 μm. Average vesicle diameter distributions were determined for the various experimental groups, and there was no difference in the diameter distributions between control and eccentric groups. The lactate efflux from a mixture of vesicles with different diameters can be described by a sum of exponentials $y = \sum_{i=1}^{n} e^{-k_i t} S_{i} F_{i} V_{i}$, where $y$ is the accumulated lactate in the efflux medium, $t$ is efflux time, $F_i$ is frequency of vesicles with diameter $d_i$, $V_i$ is vesicle volume, $S_i$ is surface area, and $k_i$ is the rate constant for vesicles with $S_i / V_i = 1$. This means that the rate constant for vesicles with a specific diameter is given by $k(S_i / V_i)$. The best curve describing the efflux of lactate from the vesicles was obtained by fitting this multieponential formula to the normalized experimental data by the nonlinear least-squares regression method. The efflux per square centimeter membrane was determined from the initial concentration change, the vesicular diameter, and the vesicular lactate space.

**Perfused Rat Hindlimb**

Perfusion apparatus and perfusion medium. The perfusion apparatus was similar to that described by Ruderman et al. (31) as modified by Goodman et al. (10). The composition of the bovine erythrocyte-containing perfusate was as previously described (26), resulting in a mean arterial perfusate pH of 7.36 ± 0.01 (SE) and partial pressures of carbon dioxide (Pco2) and oxygen (P02) of 44 ± 1 and 165 ± 9 mmHg, respectively.

Experimental procedure. Two days after eccentric contractions, rats were prepared surgically for hindquarter perfusion as previously described (29, 31). The perfusate was circulating at a rate of 12 ml/min (corresponding to ~0.31 ml·min⁻¹·g perfused muscle⁻¹) (see Ref. 31) during a 15-min equilibration period (26, 29), wherein the vessels perfusing the left hindlimb were ligated. The right leg was immobilized, and an electrode connected to a stimulator (Disa Electronic, Herlev, Denmark) was placed around the sciatic nerve. After adjustment of the resting muscle length to obtain maximum tension on stimulation, the muscles were made to contract for 2 min by stimulating the sciatic nerve with supramaximal (15–20 V) trains of 200-ms duration at a frequency of 100 Hz and applying the trains 1/s. The tension developed by the muscles was recorded on a high-speed plotter (Clevite Brush Mark 220, Clevite, Brush Instruments), and the total area under the tension vs. time curve was used as an indicator of performance. To maintain a constant composition of the perfusate at the arterial side of the hindquarter, the perfusate was noncirculating from onset of stimulation and throughout recovery. With only one hindlimb perfused, the flow rate (12 ml/min) corresponded to ~0.61 ml·min⁻¹·g perfused muscle⁻¹ (29).

Sampling of perfusion medium and muscles. Perfusate samples were drawn from the venous side before stimulation, after 1 min of stimulation, and at 0, 1, 2, and 3 min of recovery, whereas samples from the arterial side were obtained before stimulation and after 3 min of recovery. Resting muscle samples were obtained from the left hindlimb at the end of the equilibration period, and samples were taken from the right hindlimb at the end of stimulation or after 3 min of recovery. Samples were obtained from the WG and RG muscle as well as from the soleus muscle. The muscle samples were immediately freeze-clamped between aluminum tongs precooled in liquid N2 and stored at –80°C until analyzed.

**Analytic Methods**

Perfusate samples were immediately placed in ice and analyzed within 20 min. The Pco2, PO2, and pH were measured with the Astrup technique, and from these the actual base excess was calculated as described by Siggaard-Andersen (32) (ABL 30, Radiometer, Copenhagen). Oxygen saturation and hemoglobin concentration of the perfusate were determined on an OSM3 analyzer (Radiometer, Copenhagen, Denmark), and lactate concentration was determined with a YSI analyzer (Yellow Springs Instruments, Yellow Springs, OH).

Total water content of the muscles was determined by weighing the samples before and after freeze-drying to constant weight. Muscle lactate concentrations were determined fluorometrically (18). Muscle pH was measured by a small glass electrode (Radiometer GK2801) after homogenization of freeze-dried muscle samples in a nonbuffering solution containing (in mM) 145 KCl, 10 NaCl, and 5 iodoacetic acid. The nonbicarbonate-dependent physiochemical buffer capacity was determined on resting muscle samples by titration with 0.01 M HCl and 0.01 NaOH (for review see Ref. 25). The buffer...
LACTATE TRANSPORT AND ECCENTRIC CONTRACTIONS

capacity is expressed in micromoles of H⁺ per gram wet weight per pH unit, as well as in micromoles of H⁺ per gram dry weight per pH unit.

Calculations. Oxygen content in a blood sample was estimated as previously described (26). Lactate release, oxygen uptake, and change in actual base excess across the hindquarter were determined by multiplying the perfusate flow rate with arteriovenous concentration differences. Total exchange of oxygen, lactate, and H⁺ during stimulation as well as recovery were determined as the area under the corresponding exchange curve, with time on the x-axis and expressed in relation to the muscle mass assumed to be recruited during the isometric stimulation (2.7% of body weight; Ref. 33). In the latter calculations, resting exchange by the part of the hindlimb not being stimulated was subtracted from the total exchange before division by the recruited muscle mass.

Statistics

Data are presented as means ± SE. A two-way analysis of variance with repeated measures was used to evaluate differences in perfusate parameters. Otherwise, comparisons between ECC and CON muscles were made by use of Student’s t-test. P < 0.05 was considered significant.

RESULTS

Giant Sarcolemmal Vesicles

Lactate transport. The sarcolemmal lactate transport in ECC-WG (29%) and -RG (13%) was lower (P < 0.05) than in the CON muscles, whereas the rate of lactate transport in soleus was unchanged 2 days after eccentric stimulations (Fig. 1).

Perfused Rat Hindlimb

Buffer capacity. The physiochemical buffer capacity (µmol H⁺·g wet wt⁻¹·pH⁻¹) in ECC-WG and ECC-RG was 13 and 9% lower (P < 0.05), respectively, than in CON-WG and CON-RG, whereas the buffer capacity of soleus was unaffected by prior eccentric contractions (Fig. 2). The decline (P < 0.05) in physiochemical buffer capacity was 8 and 6% in WG and RG, respectively, when given as micromoles of H⁺ per gram dry weight per pH unit.

Performance and oxygen uptake. Performance was not significantly different for ECC (2.9 ± 0.1 N/g body weight) and CON hindlimbs (3.2 ± 0.2 N/g body weight) during the isometric contractions. Resting oxygen uptake and the increase in oxygen uptake during stimulation were similar for the two experimental groups, and there was no difference between groups in oxygen uptake during stimulation (2 min), during recovery (3 min), or during the entire period (7.2 ± 0.6 and 8.1 ± 0.6 µmol/g stimulated muscle for ECC and CON rats, respectively).

Muscle lactate and muscle pH. Muscle lactate concentrations in ECC-WG, ECC-RG, and ECC-soleus were lower (P < 0.05) immediately after (34, 41, and 33%, respectively) and 3 min after (32, 30, and 32%, respectively) the isometric stimulation than in the corresponding nonstimulated control muscles (Table 1). The only significant difference in muscle pH between ECC and CON muscles was found in WG at the end of stimulation (Table 1).

Lactate and H⁺ release. There was no difference between ECC and CON rat muscles in resting lactate release [1.08 ± 0.29 (n = 10) and 1.83 ± 0.86 µmol/min (n = 11), respectively], which may be due to the large standard error for the control data. Total lactate release during stimulation was also similar. However, com-

Fig. 1. Lactate transport rate (µmol·cm⁻²·s⁻¹) measured at equilibrium exchange conditions with 30 mM lactate and pH 7.4 in sarcolemmal giant vesicles obtained from white (WG) and red gastrocnemius (RG) and soleus muscle 2 days after eccentric contractions of the calf muscles on 1 side of hindlimb, with contralateral muscles as controls. Values are means ± SE. No. of experiments (n) = 6 except for soleus, where n = 5. No. of rats (N) = 24 except for soleus, where N = 20. *Significantly different from control muscles, P < 0.05.

Fig. 2. Physiochemical buffer capacity (µmol H⁺·g wet wt⁻¹·pH⁻¹) of WG, RG, and soleus muscle 2 days after eccentric contractions of the calf muscles on 1 side of hindlimb and of corresponding nonstimulated control muscles. Values are means ± SE. No. of experiments = 10. *Significantly different from control, P < 0.05.
Table 1. Muscle lactate concentrations and muscle pH in white and red gastrocnemius and soleus before and after stimulation

<table>
<thead>
<tr>
<th></th>
<th>Pre 0 3</th>
<th>Pre 0 3</th>
<th>Pre 0 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ECC</td>
<td>2.3 ± 0.3</td>
<td>25 ± 1.5*</td>
<td>21 ± 2.5*</td>
</tr>
<tr>
<td>CON</td>
<td>2.2 ± 0.2</td>
<td>38 ± 1.5</td>
<td>31 ± 2.2</td>
</tr>
<tr>
<td>pH</td>
<td>7.16 ± 0.03</td>
<td>6.65 ± 0.02*</td>
<td>6.77 ± 0.05</td>
</tr>
<tr>
<td>ECC</td>
<td>7.09 ± 0.02</td>
<td>6.47 ± 0.03</td>
<td>6.68 ± 0.07</td>
</tr>
<tr>
<td>CON</td>
<td>7.15 ± 0.03</td>
<td>6.80 ± 0.03</td>
<td>6.88 ± 0.04</td>
</tr>
</tbody>
</table>

Values are means ± SE. Muscle lactate concentrations (µmol/g wet wt) and muscle pH in white (WG) and red gastrocnemius (RG) as well as soleus before (Pre), immediately after (0), and 3 min after (3) supramaximal isometric stimulation in perfused rat hindlimb 2 days after eccentric contractions (ECC) and after no intervention (CON). No. of experiments = 6–10. *Significantly different from control muscle, P < 0.05.

pared with CON rats, total lactate release from the stimulated muscles of ECC rats was 20% lower (P < 0.05) during recovery (3 min) and 19% lower (P < 0.05) during the entire period of stimulation and recovery (5 min) (Fig. 3).

Resting H⁺ release before stimulation and total H⁺ release from the stimulated muscles during and after the isometric stimulation were not affected by prior eccentric contractions.

DISCUSSION

The main results of the present study are that the sarcolemmal lactate/H⁺ transport capacity and the physiochemical buffer capacity of rat skeletal muscle are reduced 2 days after eccentric contractions. In addition, the glycogenolytic potential appears impaired in muscles that have been stimulated eccentrically.

Muscle pH is largely determined by the physiochemical buffer capacity, which in intact muscle is primarily dependent on the content of protein-bound histidine residues, bicarbonate, and inorganic phosphate (for review see Ref. 25). Sarcolemmal H⁺ transport is another important determinant of muscle pH, and the lactate/H⁺ transporters are the membrane transport systems with the highest capacities for H⁺ removal (12) and responsible for the main part of the sarcolemmal lactate flux (11, 13, 30, 34). In the current study, the sarcolemmal lactate/H⁺ transport capacity and the physiochemical buffer capacity were decreased in WG and RG muscles subjected to prior eccentric contractions (Figs. 1 and 2). Thus prior unaccustomed eccentric contractions affect the main lactate efflux pathway and major determinants of muscle pH and, hence, very probably the exercise-induced muscle changes in pH and lactate concentrations. On this basis it may be expected that prior eccentric exercise can result in reduced work capacity during high-intensity exercise and slower recovery from such exercise, because low muscle pH (for review see Ref. 9) and high muscle lactate concentrations (8) can impair various processes in the muscle.

The reduction in sarcolemmal lactate/H⁺ transport capacity and in physiochemical buffer capacity occurred in the WG and RG muscles, with the former being changed the most, whereas these parameters were unaffected in the soleus. This distribution of the effects among the various muscle types is in line with previous observations of total muscle content of glucose transporters (GLUT-4) after eccentric contractions (5). Although this pattern may reflect the particular contraction model used, the idea that glycolytic fibers are less resistant to eccentric muscle damage than oxidative fibers is in accordance with findings in both rats (16) and humans (3). The concomitant effect of prior eccentric contractions on the sarcolemmal lactate/H⁺ transport and the GLUT-4 content (15) may indicate that this type of muscle activity is followed by a general impairment of plasma membrane proteins. Previous studies demonstrated that total GLUT-4 content was unaffected immediately after eccentric contractions (4, 5) and may suggest that the reduction in the lactate/H⁺
transport capacity was caused by processes that are delayed after the contractions. The local inflammatory response is an example of such a delayed process that may enhance protein degradation in the muscle, because infiltrating phagocytic cells have been shown to possess proteolytic activity (17). The observed decline in the lactate transport rate was smaller than the previously reported decrease in sarcolemmal GLUT-4 content (15), which may be ascribed to a specific effect of eccentric contractions on the rate of muscle GLUT-4 gene transcription, possibly via different cytokines as suggested recently (15). Alternatively, structural or functional characteristics of these membrane transport proteins may play a role. Thus the lactate/H⁺ transporters are generally recognized as permanent integral plasma membrane proteins, whereas GLUT-4 proteins move from an intracellular pool to the plasma membrane on muscle activity or insulin stimulation (14), and this cycling may render GLUT-4 more susceptible to eccentric muscle damage. Finally, release of muscle enzymes to plasma after eccentric contractions (23) may contribute to the reduced physiochemical buffer capacity of the eccentric muscles.

Additional experiments were performed using the perfused hindlimb preparation to examine whether the reductions in lactate/H⁺ transport capacity and physiochemical buffer capacity after the eccentric stimulation had an effect on the release and muscle content of lactate and H⁺. Control and eccentric calf muscles were stimulated supramaximally for isometric contractions 2 days after eccentric contractions. This stimulation protocol resulted in uniform force development in the two experimental groups, which was surprising because unaccustomed eccentric exercise is known to decrease performance (6), but it may reflect limitations in our setup. The isometric force measurements during the perfusion included all calf muscles, and these muscles are affected to various degrees by prior contractions in our eccentric model (5). In accordance with the sarcolemmal lactate transport capacity determined in the giant vesicles, lactate release during recovery from stimulation was 20% lower for muscles subjected to prior eccentric contractions than for control muscles (Fig. 3). However, the intense isometric contractions were associated with less muscle lactate accumulation (Table 1) and, hence, apparently lower lactate gradients across the sarcolemma in the eccentric muscles. With different driving forces for lactate efflux, it cannot be deduced to what extent the reduced lactate/H⁺ transport capacity per se contributed to the lower release of lactate from muscles that had been stimulated eccentrically. However, interestingly, the lower lactate concentrations in the eccentric muscles strongly indicate that the glycolytic potential (ability to produce lactate) was impaired in these muscles, because the lactate release was similar for control and eccentric muscles during the preceding isometric contractions (Fig. 3). A possible explanation for the reduced glycolytic potential may be the subnormal muscle glycogen concentrations previously demonstrated with our eccentric rat model (5) as well as after eccentric exercise in humans (3, 24), but other mechanisms also seem to be involved. Thus there was less accumulation of lactate in the eccentric soleus muscle than in the control after the isometric contractions (Table 1), even though the glycogen concentration in this particular muscle is unaffected 2 days after eccentric contractions when the present model is used (5). In light of the smaller muscle lactate accumulation in eccentric muscles, it might have been expected that the pH decline would be less pronounced in these muscles. However, this was only the case in WG at the end of stimulation (Table 1), and the lack of any further pH differences between eccentric and control muscles may involve the reduced physiochemical buffer capacity.

It deserves to be mentioned that the sarcolemmal giant vesicle preparation does not include T tubule membranes (35) and that a recent preliminary report demonstrated that T tubule membranes contain lactate/H⁺ transporter proteins (21). Although the importance of these proteins is unknown, they may have contributed to the apparent discrepancy between our vesicle transport measurements and the results obtained with the perfused rat hindlimb. Moreover, it cannot be excluded that the eccentric contractions affected the various parts of the sarcolemma differently.

In conclusion, the present data show that prior eccentric muscle contractions reduce the sarcolemmal lactate/H⁺ transport capacity and the physiochemical buffer capacity of rat skeletal muscle, which may suggest that muscle damage induced by eccentric exercise can be associated with impaired muscle pH regulation. In addition, muscles that have been stimulated eccentrically produce less lactate than control muscles during supramaximal stimulation, indicating that unaccustomed eccentric exercise may lower the glycogenolytic potential of the muscles.

A. Honig, M. Vannby, and I. Kring provided skillful technical assistance.

The research was supported by the Danish National Research Foundation (504-14) and Idrætens Forskningsråd.

Address for reprint requests: H. Pilegaard, August Krogh Institute, Univ. of Copenhagen, Universitetsparken 13, 2100 Copenhagen, Denmark.

Received 13 June 1997; accepted in final form 10 December 1997.

REFERENCES


5. Asp, S., S. Kristiansen, and E. A. Richter. Eccentric muscle contractions reduce the sarcolemmal lactate/H⁺ transport capacity and the physiochemical buffer capacity of rat skeletal muscle, which may suggest that muscle damage induced by eccentric exercise can be associated with impaired muscle pH regulation. In addition, muscles that have been stimulated eccentrically produce less lactate than control muscles during supramaximal stimulation, indicating that unaccustomed eccentric exercise may lower the glycogenolytic potential of the muscles.


