ABSTRACT: The purpose of this study was to assess the effect of physical deconditioning on skeletal muscle’s oxidative metabolism as evaluated by phosphorus-31 magnetic resonance spectroscopy (31P MRS). Twenty-seven subjects without muscle disease, representing a wide range of fitness levels, were evaluated with 31P MRS. Spectra were obtained at rest and during recovery from in-magnet exercise. The data show a significant correlation between maximum resting metabolic equivalent (MET) score and the following 31P MRS recovery indices: adenosine diphosphate and phosphocreatine recovery half-time; initial phosphocreatine resynthesis rate; calculated estimation of mitochondrial capacity; pH at end of exercise; and phosphocreatine depletion. In addition, significant differences between the deconditioned and conditioned group were found for all of the aforementioned recovery indices. At rest, only the inorganic phosphate concentration was significantly different between the two groups. These data indicate that physical activity level should be taken into account when assessing patients’ oxidative metabolism with 31P MRS.


MUSCLE PHOSPHORUS MAGNETIC RESONANCE SPECTROSCOPY OXIDATIVE INDICES CORRELATE WITH PHYSICAL ACTIVITY

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Physical deconditioning due to reduced physical activity is common in patients with chronic illnesses as well as in healthy subjects who lead a sedentary lifestyle. The effects of deconditioning on muscle structure and metabolism include: muscle atrophy; reduction in mitochondrial oxidative enzyme content; greater lactic acid production and acidification during exercise; and conversion to more anaerobic muscle fiber types.2,3,10,11,14,16,17,19–21,26,28,31 Much of what is known about metabolic adaptation due to deconditioning stems from biopsy or extract data that is not suitable for measuring kinetic changes in energy metabolism.

Phosphorus magnetic resonance spectroscopy (31P MRS) can noninvasively monitor in vivo metabolic changes in the concentrations of phosphocreatine (PCr), adenosine triphosphate (ATP), and inorganic phosphate (Pi).8,30 Furthermore, 31P MRS can be used to calculate the concentration of metabolically active adenosine diphosphate (ADP) and cytoplasmic pH. These features lend themselves well to kinetic studies of in vivo energy metabolism in healthy and diseased skeletal muscle, especially during recovery from exercise.8 Because recovery of muscle energy state after exercise is almost totally dependent on oxidative metabolism, 31P MRS recovery indices have been used to characterize changes

Abbreviations: ADP, adenosine diphosphate; ADP<sub>endex</sub>, ADP concentration at end of exercise; ADP<sub>1/2</sub>, ADP recovery half-time; ANOVA, analysis of variance; ATP, adenosine triphosphate; CNS, central nervous system; k<sub>CK</sub>, creatine kinase equilibrium constant; MET, resting metabolic equivalent; MET<sub>max</sub>, maximum MET score; PCr, phosphocreatine; PCr<sub>dep</sub>, PCr depletion; PCr<sub>endex</sub>, PCr at end of exercise; PCr<sub>1/2</sub>, PCr recovery half-time; PCr<sub>Δ</sub>, change in PCr concentration; pH<sub>Δ</sub>, change in pH; pH<sub>endex</sub>, pH at end of exercise; pH<sub>min</sub>, minimum pH during recovery; Pi, inorganic phosphate; Pi<sub>1/2</sub>, Pi recovery half-time; 31P MRS, phosphorus-31 magnetic resonance spectroscopy; Q<sub>max</sub>, calculated estimation of mitochondrial capacity; TCr, total creatine concentration; V, initial PCr resynthesis rate

Key words: physical deconditioning; oxidative metabolism; resting metabolic rate (MET); phosphorus-31 magnetic resonance spectroscopy (31P MRS); skeletal muscle

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in mitochondrial metabolism in various muscle diseases.4,8,10,27

$^{31}$P MRS has also been used to monitor muscle’s metabolic adaptations to aerobic training. A faster postexercise PCr resynthesis rate has been observed in trained subjects as compared with sedentary subjects starting at the same PCr concentration and pH.29 Less exercise-induced acidosis and faster PCr recovery rates have been reported by McCully et al.24 in the wrist flexor muscles of rowers relative to sedentary controls and by Minotti et al.25 in the dominant forearm relative to the nondominant forearm of the same individual. Changes in other indices of oxidative phosphorylation were not reported and little is known about the effects of different physical conditioning levels on the kinetics of various phosphate metabolites involved in energy metabolism.

To our knowledge, no attempts have been made to correlate a range of fitness levels with $^{31}$P MRS indices. This could be an important factor in the use of $^{31}$P MRS to detect mitochondrial dysfunction in patients complaining of exercise intolerance, as well as assessing normal muscle status of healthy subjects. Our aim was to determine the relationship of $^{31}$P MRS indices to conditioning level.

METHODS

**Subjects.** A group of 27 normal subjects, aged 21–55 years (14 men, 13 women), without muscle disease, were recruited for the study. Informed consent was obtained from all subjects. The Ethics Committee of the Montreal Neurological Institute and Hospital and the McGill University Ethics Review Board approved the study.

**Measure of Physical Conditioning.** A scale of the energy cost of various physical activities in multiples of the resting metabolic rate (METs) was used to estimate the subjects’ level of lower-body physical conditioning. The scale was based on MET values for various daily activities obtained from the *Compendium of Physical Activities*4 and included only activities that primarily utilize the lower extremities, because we only evaluated calf muscles with our $^{31}$P MRS protocol. A value of 1 MET is considered to be equivalent to a measured normal resting oxygen uptake of 3.5 mL/kg per minute. The subjects were asked to fill out a physical activity questionnaire including the time spent doing each activity per week. This was then used to evaluate the weighted average of the MET score for their most active 3 h/week ($MET_{max}$).

We chose 3 h because the American Sports Association recommends a minimal whole body training level of at least 1.5 h/week, and because we used the subjects’ reports of their weekly activity we ran the risk of overestimating their $MET_{max}$ scores if we only considered 1.5 h. All individuals participating in this study had been at their $MET_{max}$ value for at least 8 weeks.

We defined as deconditioned 11 subjects who had a $MET_{max}$ score of <5. $MET_{max}$ values <5 were mostly associated with daily activities that would not induce training in conditioned subjects (e.g., MET of walking briskly = 4.5; MET of showering or walking moderately = 4; MET of cleaning or walking for pleasure = 3.5; MET of making bed = 2; MET of sitting quietly = 1), especially since weekly cumulative duration was used to calculate the $MET_{max}$ score. The average $MET_{max}$ of the deconditioned group was 3.5 and ranged from 2.2 to 4.3. This group included 3 subjects with extremely low $MET_{max}$ scores; 2 had undergone leg immobilization for 2 months because of injury (not to their calf muscle) and were now in their first week of physiotherapy, and 1 person was recovering from brain surgery. They were studied because we wanted to include extreme deconditioning of the calf muscles, as occurs with immobilization. The rest of the deconditioned group comprised people who carried on normal activities that were not demanding on the lower extremities (N = 8).

The conditioned group (N = 16) ranged in $MET_{max}$ score from 5 to 15.5 with an average $MET_{max}$ of 8.1. This group was composed of athletes (N = 2), of people who exercised regularly or did recreational sports (N = 12) and people who carried on daily activities that were demanding on the lower extremities (N = 2) (e.g., MET of walking upstairs = 8; MET of walking/running or playing with children vigorously = 5).

$^{31}$P MRS of Muscle. **Exercise Protocol.** The in-magnet exercise protocol we used has been previously described.4 A custom-built apparatus installed in the magnet allowed subjects to exercise their calf muscles. The subjects lay supine with one foot placed against a pedal connected to a piston and cylinder with a needle valve outlet and a ball-valve return. A thigh blood pressure cuff, inflated to 180 mmHg, was used to occlude circulation to the limb. Plantar flexion of the foot against the pedal resulted in isokinetic exercise of the calf muscles (gastrocnemius and soleus). The subjects did a 1-min warm-up and then exercised until exhaustion, a duration of 1–3 min. At exhaustion the muscles were relaxed for 16 s, still under ischemia. The thigh cuff was then deflated as collection of recovery data began.

$^{31}$P MRS Data Acquisition. Muscle spectra were
acquired at rest and during recovery from exercise, as previously reported from this laboratory.\textsuperscript{4,15,22} A combined magnetic resonance imaging and spectroscopy system operating at 1.5 T (Gyrosan ACSII, Philips Medical Systems, Best, The Netherlands) and a 6-cm-diameter surface coil placed under the gastrocnemius muscle were used to acquire the phosphorus spectra. Two rest spectra of the gastrocnemius muscle were obtained using a nominal 90° pulse with a 2-s and a 30-s interpulse delay to correct for saturation at the rapid pulse rate. Postexercise recovery spectra were obtained using a 2-s interpulse delay. Serial recovery spectra were acquired for 15 min using progressively longer acquisition blocks.

\textit{\textsuperscript{31}P MRS Data Analysis.} Resonance intensities and positions were determined from fitting raw data in the time domain, as previously described.\textsuperscript{8,15} PCr and Pi concentrations at rest were calculated from fully relaxed spectra by comparing their intensity values relative to ATP and assuming an ATP concentration of 7.97 mmol/L. Recovery spectra were first corrected for differential saturation and then concentrations were determined using the same assumption for ATP concentration as for rest data.

Cytoplasmic pH was determined from the chemical shift between PCr and Pi.\textsuperscript{29} Cytoplasmic metabolically active ADP, although not directly visible with \textsuperscript{31}P MRS due to its low concentration, was calculated using the creatine kinase reaction, which was assumed to be near equilibrium so that ADP = [ATP] [TGr - PCr]/[H+] [PCr] k\textsubscript{CK}. A k\textsubscript{CK} (creatinine kinase equilibrium constant) of 1.66 × 10\textsuperscript{9} M and a TCr (total creatine concentration) of 42.9 mmol/L were used for calculations.\textsuperscript{8,15} Data from the first time point after exercise were used to determine end-of-exercise indices: PCr depletion and PCr concentration (mmol/L) and pH at end of exercise (PCr\textsubscript{endex}, PCr\textsubscript{rest}, pH\textsubscript{endex}, and pH\textsubscript{rest}, respectively). PCr depletion was calculated as PCr\textsubscript{dep} = (PCr\textsubscript{rest} - PCr\textsubscript{endex})/PCr\textsubscript{rest}. Changes in PCr concentration (PCr\textsubscript{dA}) and pH (pH\textsubscript{A}) were calculated by subtracting end-of-exercise measures from rest. The lowest pH (pH\textsubscript{min}) achieved by the muscle was also calculated; this is usually observed during the first minute of recovery.

Recyly recovery indices calculated included: recovery half-times for PCr, Pi, and ADP (PCr\textsubscript{1/2}, Pi\textsubscript{1/2}, and ADP\textsubscript{1/2}); initial PCr resynthesis rate (V); and estimated mitochondrial capacity (Q\textsubscript{max}). PCr\textsubscript{1/2} and Pi\textsubscript{1/2} were calculated by fitting the recovery with a single exponential function.\textsuperscript{4,15} ADP concentration has been observed to decrease below the resting concentration during the first 5 min of recovery so ADP

\textit{Statistical Analyses.} Data Transformation. A series of one-sample Lilliefors tests was used to determine whether subjects’ MET\textsubscript{max} values and their \textsuperscript{31}P MRS values were normally distributed. Those measures that were not normally distributed were log-transformed and then tested again to see if the transformation sufficiently normalized the data. This log-transformed data were used for all subsequent analyses.

Correlations. Pearson product-moment correlations were used to determine the relationships between individual subjects’ MET\textsubscript{max} scores and their values on the 16 \textsuperscript{31}P MRS indices. Equality between any two correlations was determined using Fisher’s Z transformation.

Group Differences. Fisher’s exact test was used to determine if there was an association between conditioning groups and gender. One-way analysis of variance (ANOVA) was used to determine if the conditioned and deconditioned groups differed in their mean age or MET\textsubscript{max} scores. Three separate one-way, multivariate analyses of variance (MANOVA) were used to determine if the groups differed in their patterns across (i) their five rest, (ii) their six end-of-exercise, and/or (iii) their five recovery \textsuperscript{31}P MRS indices. Subsequent one-way ANOVAs were used to determine whether the two groups differed significantly on any of the 16 \textsuperscript{31}P MRS indices. For each of the ANOVAs, the squared multiple correlation (r\textsuperscript{2}) indicates how much of the variance in the dependent variable is explained by the grouping variable.

All statistical analyses were performed using Systat 7.01 software.

\textbf{RESULTS}

\textbf{Group Data.} Descriptive and inferential statistics for the conditioned and deconditioned groups are shown in Table 1. The groups did not differ in their mean (log-transformed) ages and there was no association between conditioning group and gender (P = 0.054).

\textit{\textsuperscript{31}P MRS Data.} Correlations. There were no significant linear relationships between the individuals’ (log-transformed) MET\textsubscript{max} values and any of the five resting \textsuperscript{31}P MRS measures. When examined separately, it appeared that the conditioned individuals

\textsuperscript{31}P MRS and Deconditioning
had a different linear relationship between their METmax values and their Pi values \((r = 0.458)\) than did the deconditioned individuals \((r = -0.054)\). This was not significant \((P = 0.118)\) and most likely reflected an artifact of the small range of METmax scores in the deconditioned group.

There were significant linear relationships between the individuals’ (log-transformed) METmax values and their six end-of-exercise 31P MRS measures: negative relationships with \(pH_{\text{endex}}\) \((r = -0.606, P = 0.0008)\), \(\text{pH}_{\text{min}}\) \((r = -0.533, P = 0.0042)\), and \(\text{PCr}_{\text{endex}}\) \((r = -0.631, P = 0.0004)\) and positive relationships with \(\text{PCr}_{\text{dep}}\) \((r = 0.576, P = 0.0017)\), \(\text{PCr}_{\Delta}\) \((r = 0.419, P = 0.0296)\), and \(\text{pH}_{\Delta}\) \((r = 0.606, P = 0.0008)\).

As can be seen in Figure 1, there were significant linear relationships between the individuals’ (log-transformed) METmax values and their values on four of the five recovery 31P MRS measures: negative relationships with \(\text{PCr}_{1/2}\) and \(\text{ADP}_{1/2}\) (both log-transformed) and positive relationships with \(V\) and \(Q_{\text{max}}\). The highest correlation observed was between METmax and \(V\), which had 45% of their variance in common. There appeared to be no difference between the two groups in terms of their separate relationships between their METmax values and their end-of-exercise and recovery 31P MRS measures.

**Group Differences.** MANOVA testing found no significant difference between the two groups’ profiles across their five resting 31P MRS indices \((F_{5, 21} = 1.63, P = 0.196)\), their six end-of-exercise 31P MRS indices \((F_{6, 20} = 1.10, P = 0.396)\), and their five recovery 31P MRS indices \((F_{6, 20} = 2.63, P = 0.053)\). Subsequent ANOVA testing showed that the deconditioned group had a greater mean Pi concentration at rest, greater mean \(pH_{\text{endex}}, \text{pH}_{\text{min}}, \text{and PCr}_{\text{endex}}\) values, and slower mean \(\text{PCr}_{1/2}\) and \(\text{ADP}_{1/2}\), whereas the conditioned group had a greater mean \(\text{PCr}_{\text{dep}}, \text{pH}_{\Delta}, V\), and \(Q_{\text{max}}\). Further descriptive and inferential statistics for the conditioned and deconditioned groups’ 31P MRS values are shown in Table 1.

**DISCUSSION**

Physical deconditioning accompanies many chronic illnesses as well as a sedentary lifestyle in normal, healthy individuals. The purpose of our study was to

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**Table 1.** Descriptive and inferential statistics for the conditioned and deconditioned groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Conditioned subjects ((n = 16))</th>
<th>Deconditioned subjects ((n = 11))</th>
<th>Analysis of variance ((F_{1,25}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects</td>
<td>METmax 5.00–15.50 8.09 3.33</td>
<td>2.50–4.30 3.53 0.75</td>
<td>41.40(^{ig}) 0.000(^{g}) 0.62</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>21.00–55.25 28.20 8.75</td>
<td>22.20–47.80 30.95 9.66</td>
<td>0.59(^{ig}) 0.449 0.02</td>
</tr>
<tr>
<td>Rest</td>
<td>pH 7.00–7.06 7.04 0.02</td>
<td>6.98–7.09 7.03 0.03</td>
<td>0.21 0.653 0.01</td>
</tr>
<tr>
<td></td>
<td>PCr 29.47–36.49 33.24 2.14</td>
<td>27.90–41.49 34.89 3.82</td>
<td>2.06 0.164 0.08</td>
</tr>
<tr>
<td></td>
<td>Pi 1.83–4.53 3.32 0.89</td>
<td>2.89–7.10 4.36 1.25</td>
<td>6.46 0.018(^*) 0.21</td>
</tr>
<tr>
<td></td>
<td>PCr/Pi 6.53–17.82 10.92 3.65</td>
<td>5.42–11.98 8.48 2.16</td>
<td>3.89(^{ig}) 0.060 0.13</td>
</tr>
<tr>
<td>ADP</td>
<td>9.18–24.61 15.53 4.64</td>
<td>1.74–32.04 12.88 8.23</td>
<td>3.09 0.295 0.04</td>
</tr>
<tr>
<td>End of exercise</td>
<td>pH(_{\text{endex}}) 6.361–6.898 6.60 0.16</td>
<td>6.51–6.90 6.73 0.13</td>
<td>5.07 0.033(^*) 0.17</td>
</tr>
<tr>
<td></td>
<td>pH(_{\text{min}}) 6.17–6.89 6.54 0.19</td>
<td>6.46–6.83 6.68 0.13</td>
<td>4.34 0.049(^*) 0.15</td>
</tr>
<tr>
<td></td>
<td>PCr(_{\text{endex}}) 4.80–22.47 12.18 4.17</td>
<td>6.62–21.39 16.23 4.34</td>
<td>5.95 0.022(^*) 0.19</td>
</tr>
<tr>
<td></td>
<td>PCr(_{\text{dep}}) 0.38–0.87 0.63 0.12</td>
<td>0.40–0.79 0.53 0.12</td>
<td>4.48 0.044(^*) 0.15</td>
</tr>
<tr>
<td></td>
<td>PCr(_{\Delta}) 13.79–31.69 21.12 4.40</td>
<td>13.59–25.35 18.66 4.89</td>
<td>1.86 0.184 0.07</td>
</tr>
<tr>
<td></td>
<td>pH(_{\Delta}) 0.12–0.68 0.44 0.16</td>
<td>0.14–0.54 0.30 0.13</td>
<td>5.35 0.029(^*) 0.18</td>
</tr>
<tr>
<td>Recovery</td>
<td>PCr(_{1/2}) 0.44–1.15 0.69 0.20</td>
<td>0.55–2.16 1.02 0.50</td>
<td>5.97(^{ig}) 0.022(^*) 0.19</td>
</tr>
<tr>
<td></td>
<td>PCr(_{1/2}) 0.29–1.16 0.57 0.19</td>
<td>0.42–1.30 0.61 0.26</td>
<td>0.22(^{ig}) 0.641 0.01</td>
</tr>
<tr>
<td></td>
<td>ADP(_{1/2}) 0.13–0.59 0.27 0.12</td>
<td>0.24–1.33 0.53 0.33</td>
<td>10.83(^{ig}) 0.003(^{f}) 0.30</td>
</tr>
<tr>
<td></td>
<td>V 9.32–38.32 22.87 8.12</td>
<td>6.70–24.93 14.47 5.48</td>
<td>8.93 0.006(^{f}) 0.26</td>
</tr>
<tr>
<td></td>
<td>Q(_{\text{max}}) 15.82–58.17 37.14 11.26</td>
<td>9.57–43.37 25.89 10.33</td>
<td>6.95 0.014(^{g}) 0.22</td>
</tr>
</tbody>
</table>

Descriptive and inferential statistics for the conditioned and deconditioned groups’ ages; METmax values; and 31P MRS resting, end-of-exercise, and recovery values. Group ranges, means and standard deviations are shown, as well as the results of one-way ANOVA testing \((^* P < 0.05; ^{ig} P < 0.01; ^{f} P < 0.001)\). The following indices were not normally distributed and were log-transformed: METmax, age, PCr/Pi, PCr\(_{1/2}\), PCr\(_{1/2}\), and ADP\(_{1/2}\). All concentrations are in millimoles per liter except ADP, which is in micromoles per liter; recovery half-times \((1/2)\) are in minutes; \(V\) and \(Q_{\text{max}}\) are in millimoles per liter per minute.
determine the impact of a muscle’s physical activity level on 31P MRS indices. The results reveal that fitness level (as indicated by METmax scores) correlates significantly with many 31P MRS kinetic indices of oxidative energy metabolism measured during recovery from exercise. Despite the large range in 31P MRS values observed across our normal subjects, a number of postexercise indices and one rest index were significantly different between the deconditioned and conditioned groups. Fitness level did not correlate with the concentration of phosphate-containing metabolites at rest. This is not surprising given that differences in oxidative capacity become more evident when energy metabolism is stressed by muscle work.

All our results indicate that conditioned subjects have a better overall oxidative metabolism in their muscle than the deconditioned subjects. V, the defined measure of the initial PCr resynthesis rate, was strongly correlated with METmax as well as being significantly different between the two groups. The faster rate observed in the conditioned group resulted from both a larger PCr concentration change with exercise as well as a shorter PCrt1/2. ADP t1/2, which has been proposed as the most sensitive indicator of mitochondrial dysfunction in metabolic myopathies, also displayed significant correlation with METmax scores and was significantly different between the two groups. The longer ADPt1/2 associated with the lower MET max scores implies that ADP recovery rate is also affected by physical deconditioning. Physical deconditioning may thus be partly responsible for the slower rates of ADP recovery observed in some patients with metabolic myopathies and exercise intolerance of undetermined origin.

FIGURE 1. Scatterplots showing the relationship between METmax values (abscissa) and recovery 31P MRS values in the conditioned subjects (○) and deconditioned subjects (●). Solid lines represent the linear regression fit across all subjects and the broken lines represent the separate linear regression fits for the conditioned (right) and deconditioned (left) subjects. Pearson product-moment correlations (r) and associated p values are shown above each plot (*P < 0.05; **P < 0.01; ***P < 0.001). All recovery half-times (t1/2) are in minutes; V and Qmax are in millimoles per liter per minute.
drial density and capacity and of substrate and oxygen supply, but independent of muscle mass, also displayed significant correlation with MET\text{max} scores and was significantly lower in the deconditioned group.

The faster mean PCr recovery rate observed in our conditioned group is particularly noteworthy. A faster PCr\text{1/2} in athletes has been reported elsewhere. The main difference is that, in our study, PCr\text{1/2} was significantly shorter in subjects with higher MET\text{max} scores despite their having achieved a lower pH and a greater PCr depletion. It is well known that acidosis causes a longer PCr\text{1/2}, yet this effect was masked in our study. We believe that the increased oxidative metabolic capacity associated with greater physical conditioning countered the effect of acidosis to slow PCr resynthesis in our conditioned group. We speculate that the deconditioned subjects depleted significantly less PCr than the conditioned group because they were less tolerant of the muscle pain associated with exhaustive ischemic exercise as used in our MRS protocol and less accustomed to pushing themselves to their limit.

At rest, only the Pi concentration was significantly different between the two groups. An elevated Pi at rest has been reported in diseases with muscle fiber damage, mitochondrial myopathies, Duchenne muscular dystrophy, sporadic inclusion-body myositis, and certain central nervous system (CNS) disorders. It is unclear why Pi is higher in the deconditioned group. This may be indicative of poor mitochondrial function, but it is possible that some other common mechanism exists.

The aforementioned data imply that the level of physical conditioning or deconditioning of skeletal muscle is reflected in its oxidative capacity, which can be measured by 31P MRS. Disuse of skeletal muscle leads to morphological and biochemical changes: loss of muscle mass, a decline in oxidative enzymes, and a decrease in mitochondrial density. Exercise training, on the other hand, results in muscle changes in the opposite direction and an increase in capillary density and muscle blood flow. Any of the aforementioned changes could be responsible for the differences between the conditioned and deconditioned groups observed in this study.

In conclusion, our results indicate that physical deconditioning of skeletal muscle is clearly detectable with 31P MRS and that physical activity level must be taken into account when interpreting 31P MRS data for normal subjects and patients with various illnesses. Although it has been suggested previously that physical deconditioning could affect 31P MRS results, our study clearly demonstrates its impact and strengthens the notion that 31P MRS can be used to monitor training effects on the peripheral skeletal muscle of patients and normal subjects.

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