Influence of late exercise training on myostatin and follistatin expression in soleus muscle of rats with chronic heart failure

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INTRODUCTION
Physical exercise plays an important role in heart failure treatment. However, the mechanisms involved in exercise-induced improvement of functional capacity are not clear. Myostatin, a member of the transforming growth factor-β family, has been shown to modulate muscle growth and mass. Early aerobic exercise in infarcted rats prevented myostatin changes in skeletal muscle. Follistatin is one of the most commonly studied myostatin antagonists.

OBJECTIVE
The aim of this study was to evaluate the effects of late exercise training on myostatin and follistatin protein expression in soleus muscle from rats with myocardial infarction-induced heart failure.

RESULTS

- Physical activity was performed on a treadmill at 16 m/min, 40 min/day, 5 days/week, for 12 weeks.

- Echocardiographic evaluation was performed before and after the exercise period.

- Infarct size was measured in left ventricular histological sections stained with Picrosirius red.

- Fiber cross-sectional area and splitting frequency were evaluated in histological sections stained with hematoxylin-eosin.

- Myostatin and follistatin protein levels were analyzed by Western blot.

CONCLUSION
Late exercise training increases splitting fibers frequency and prevents myostatin protein expression change in skeletal muscle of infarcted rats.

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METHODS

Male Wistar rats (200-250g)

Sham (Sh)-operated animals

Sham sedentary (Sed) (n = 12)

Sham exercised (Ex) (n = 12)

Myocardial Infarction (MI) Surgery

MI sedentary (Sed) (n = 11)

MI exercised (Ex) (n = 10)

Surgery 3 months

Exercise 6 months

Echocardiogram

Echocardiogram

Sh-Sed
Sh-Ex

MI-Sed
MI-Ex

Arrows indicate infarcted region

Echocardiographic data

<table>
<thead>
<tr>
<th></th>
<th>Sh-Sed</th>
<th>Sh-Ex</th>
<th>MI-Sed</th>
<th>MI-Ex</th>
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</thead>
<tbody>
<tr>
<td>Diastolic Area (cm²)</td>
<td>0.55 ± 0.07</td>
<td>0.55 ± 0.10</td>
<td>1.06 ± 0.16</td>
<td>0.96 ± 0.23</td>
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<tr>
<td>Systolic Area (cm²)</td>
<td>0.16 ± 0.04</td>
<td>0.16 ± 0.03</td>
<td>0.82 ± 0.15</td>
<td>0.68 ± 0.18</td>
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<tr>
<td>Δ Area (%)</td>
<td>70.1 ± 6.35</td>
<td>70.0 ± 5.69</td>
<td>22.8 ± 7.86</td>
<td>29.7 ± 5.51</td>
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<tr>
<td>LVDD (mm)</td>
<td>8.73 ± 0.66</td>
<td>9.17 ± 0.34</td>
<td>11.79 ± 1.03</td>
<td>11.26 ± 1.20</td>
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<tr>
<td>LVDD/BW (mm/Kg)</td>
<td>16.8 ± 1.24</td>
<td>18.3 ± 1.43</td>
<td>22.2 ± 2.35</td>
<td>22.5 ± 2.29</td>
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<tr>
<td>LA (mm)</td>
<td>6.41 ± 0.55</td>
<td>6.13 ± 0.53</td>
<td>8.58 ± 1.41</td>
<td>8.15 ± 1.09</td>
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<tr>
<td>LA/BW (mm/Kg)</td>
<td>12.4 ± 1.72</td>
<td>12.2 ± 0.94</td>
<td>16.2 ± 2.98</td>
<td>16.2 ± 1.85</td>
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<tr>
<td>PWSV (mm/s)</td>
<td>38.3 ± 3.07</td>
<td>36.4 ± 3.16</td>
<td>20.1 ± 5.33</td>
<td>21.6 ± 4.91</td>
</tr>
</tbody>
</table>

Δ Area: fractional area change; LVDD: left ventricular (LV) diastolic dimension; BW: body weight; LA: left atrium dimension; PWSV: posterior wall shortening velocity. Mean ± standard deviation. * p < 0.05 vs Sh-Sed; † p < 0.05 vs Sh-Ex; ‡ p < 0.05 vs MI-Sed. ANOVA and Bonferroni.