Muscle glycogen depletion and BCAA supplementation in trained rats: influence on performance and anaplerotic reactions

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Introduction
BCAA have been largely utilized by athletes to either enhance performance or to prevent muscle damage caused by endurance activities. High levels of leucine in muscle might trap alpha-keto-glutarate from Tricarboxilic Acids Cycle (TCA), when glycogen stores are depleted, according to some authors, and therefore impair sports performance. However, BCAA’s carbon groups might be used as fuel to oxidation, according to the literature. The aim of this work was to verify if BCAA supplementation in trained rats submitted to glycogen depletion and intense exercise affected performance and TCA intermediates concentration in muscle.

Methods
40 male Wistar rats (age 8 weeks) were divided into 5 groups (n= 8)(BCAA, ISOleucine, LEUcine, VALine, PLAcebo) and were trained using a swimming protocol of 8 weeks, 1 hour a day, 5 days a week, with a 5 % load attached to their tails. In the sixth week, they performed an anaerobic treshold test (lactate). Then they were given BCAA for the last 7 days (166 mg/kg weight/day). One day before the final experiment, they trained 1 hour, and were kept fasted during 24h to deplete glycogen stores. The final test consisted on swimming for 10 min without any load on their tails (warm up) and then to swim with 7 or 8% load according to their lactate treshold test (submaximal exercise) until fatigue was reached. Then they were euthanized by decapitation, blood and tissues were collected and kept in liquid N2 until further analysis. The parameters utilized were the following: muscle and liver glycogen, resistance to fatigue (s), muscle alphaketoglutarate level and citrate synthase total activity.

Results

<table>
<thead>
<tr>
<th>Groups</th>
<th>Time of Exercise (s)</th>
<th>AKG (mg/mg wet tissue)</th>
<th>Citrate Synthase Activity (umol/min/ mg tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLA</td>
<td>400,4 + 222</td>
<td>0,8 + 0,1e</td>
<td>12,3 + 4</td>
</tr>
<tr>
<td>BCAA</td>
<td>257 + 110</td>
<td>0,33 + 0,2e</td>
<td>12,3 + 5</td>
</tr>
<tr>
<td>ISO</td>
<td>347,3 + 183</td>
<td>0,049 + 0,03e</td>
<td>12,5 + 4</td>
</tr>
<tr>
<td>LEU</td>
<td>398,6 + 306</td>
<td>0,010 + 0,06e</td>
<td>11,8 + 2</td>
</tr>
<tr>
<td>VAL</td>
<td>283 + 147</td>
<td>1,5 + 0,8 a,b,c,d</td>
<td>9,6 + 3</td>
</tr>
</tbody>
</table>

Table 1: Parameters of performance and TCA cycle in muscle

Exercise performance was not different among experimental groups, regardless of their supplementation. AKG levels in muscle tended to be very low in LEU group, but still they were not different from the others, except the VAL Group. The latter had higher concentrations of AKG then all the others. Citrate synthase activity was the same for all experimental groups.

Muscle Glycogen was lower in LEU group when compared to PLA. Liver Glycogen did not feature any statistical difference among experimental groups.

Discussion/Conclusion
It was not possible to observe any statistical difference among experimental groups regarding exercise performance, however muscle AKG levels tended to be lower in LEU group. We cannot affirm AKG was deviated to transaminate Leucine, though, and it did not impair TCA cycle, as measured by Citrate Synthase activity. It seems that BCAA supplementation or any of them supplemented individually tended to keep higher liver glycogen throughout intense exercise. They might have been transaminated in muscle to generate glucose in liver through alanine-glucose cycle.

Fig 1: Glycogen content

References