Ingestion of branched-chain amino acids and tryptophan during sustained exercise in man: failure to affect performance


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1. An increased uptake of tryptophan in the brain may increase serotoninergic activity and recently has been suggested to be a cause of fatigue during prolonged exercise. The present study, therefore, investigates whether ingestion of tryptophan or the competing branched-chain amino acids (BCAAs) affect performance. Ten endurance-trained male athletes were studied during cycle exercise at 70–75% maximal power output, while ingesting, ad libitum and double-blind, drinks that contained 6% sucrose (control) or 6% sucrose supplemented with (1) tryptophan (3 g l⁻¹), (2) a low dose of BCAA (6 g l⁻¹) or (3) a high dose of BCAA (18 g l⁻¹).

2. These treatments greatly increased the plasma concentration of the respective amino acids. Using the kinetic parameters of transport of human brain capillaries, BCAA supplements were estimated to reduce brain tryptophan uptake at exhaustion by 8–12%, while tryptophan ingestion caused a 7- to 20-fold increase. Exercise time to exhaustion was not different between treatments (122 ± 3 min).

3. The data suggest that manipulation of tryptophan supply to the brain either has no additional effect upon serotoninergic activity during prolonged exhaustive exercise or that manipulation of serotoninergic activity functionally does not contribute to mechanisms of fatigue.

Prolonged exercise inevitably leads to fatigue. Traditionally, fatigue mechanisms have focused on events in skeletal muscle leading to loss of contractility. Glycogen depletion (Hermansen, Hultman & Saltin, 1987), a decrease of the resting membrane potential as a consequence of potassium losses (Søgaard, 1990), failure of the calcium pump in the sarcoplasmatic reticulum, an increase in the free ADP concentration, and failure of the neuromuscular transmission (Edwards, 1981) have all been associated with fatigue during sustained exercise. Newsholme and colleagues (Newsholme, Aeworth & Blomstrand, 1987) have more recently proposed that fatigue during prolonged exercise may be caused by increased serotoninergic activity in the central nervous system.

In the ‘central fatigue hypothesis’ it is suggested that during prolonged exercise the increased oxidation of BCAA (Kasperek, Dohm & Snider, 1984; Wagenmakers et al., 1991) and the displacement of tryptophan from albumin by the increasing fatty acid concentration (Davis, Bailey, Woods, Galano, Hamilton & Bartoli, 1992) leads to an increase in the tryptophan/BCAA ratio. The increase in this ratio would lead to an increased tryptophan transport across the blood–brain barrier, since BCAA and tryptophan compete for entry by the large neutral amino acid (LNAA) transporter. Once taken up, conversion of tryptophan to 5-hydroxytryptamine would occur and lead to a local increase of this neurotransmitter (Knott & Carzon, 1972; Chacouloff, Laude, Guesseneo & Elghozzi, 1986a). That this increase occurs during exercise in certain brain areas has been established in the rat (Chacouloff et al., 1986a), but not in man. The increase in serotoninergic activity would subsequently lead to central fatigue, forcing athletes to stop exercise or reduce pace or workload. One of the implications of the ‘central fatigue hypothesis’ is that ingestion of BCAA could reduce the exercise-induced increase of brain tryptophan uptake and thus delay fatigue by giving athletes the ability to push on when peripheral fatigue mechanisms come into operation. This indeed has been suggested to be the case in a field study with marathon runners (Blomstrand, Hassmén, Ekblom & Newsholme, 1991). Another implication is that ingestion of tryptophan would reduce time to exhaustion.

The present study therefore was undertaken to investigate whether oral ingestion of BCAA or tryptophan has an effect, predicted by the ‘central fatigue hypothesis’, on time to exhaustion during prolonged exercise in a controlled laboratory study. The amino acids were given in doses that greatly influence the circulating levels to values well outside the normal physiological ranges. It was chosen to add the amino acids to a carbohydrate solution for two reasons;
(1) in order to obtain similar experimental conditions as in the field study of Blomstrand et al. (1991), where BCAA supplementation indeed was claimed to improve performance when given in combination with carbohydrates, and (2) to avoid a potential interfering effect of BCAA supplementation on muscle fatigue in conditions of low carbohydrate availability. BCAA supplementation has previously been reported to have a negative effect on performance in patients with a glycogen breakdown defect in muscle (McArdle's disease, muscle phosphorylase deficiency) supposedly by a further reduction of the activity of the tricarboxylic acid cycle in conditions of depleted glycogen stores and low carbohydrate availability (Wagenmakers, Oakley & Edwards, 1980).

METHODS

Subjects

Ten healthy endurance-trained male athletes participated in this study after they had been informed about possible risks involved and had given their voluntary consent to participate. Their age, weight, and maximal workload (mean ± s.d.) were 23.3 ± 4.4 years, 70.6 ± 4.9 kg, 373.1 ± 27.3 W, respectively. The study was approved by the Medical Ethical Committee of the University of Limburg.

Procedures

The subjects were studied while exercising on an electromagnetically braked cycle ergometer (urate, The Netherlands). The maximal power output (P_{max}) of each subject was measured as described previously (Kuijpers, Keizer, Boumans & Saris, 1987). Within 2–3 weeks of assessment of P_{max} the subjects performed a pretest, to get familiar with the protocol, and four experimental tests until exhaustion at 70–75% P_{max} at a freely chosen, power output-independent pedalling rate (90–120 r.p.m.). During these four experimental tests they received, at random and double-blind, different drinks. The experimental drinks contained 6% sucrose without additives (control) or with one of the following amino acids: 3 g 1% of tryptophan (BUPA, The Netherlands); 2 g 1% each of valine, isoleucine and leucine (Degussa, The Netherlands) or 6 g 1% each of valine, isoleucine and leucine (high BCAA). The low BCAA dose is comparable to the amount given by Blomstrand et al. (1991) in their field study. The control, tryptophan and low BCAA drink were flavoured with small amounts of quinine sulphate to simulate the bitter taste of the high BCAA drink.

Subjects were instructed to avoid intense exercise on the day prior to each test. At 07.30 h subjects ate a standardized breakfast that consisted of three slices of whole grain bread with low fat margarine and honey or marmalade, and tea. The actual performance test started at 09.00 h with a warming-up period of 10 min at 100 W. After the warming up the workload was increased to 70–75% P_{max} and subjects had to cycle until exhaustion, defined as the inability to maintain a pedalling rate of 60 r.p.m. despite encouragement to push on. The test drinks were provided after 5 min of warming up as a bolus of 4 ml (kg body weight)^{-1} and then every 15 min as a bolus of 2 ml (kg body weight)^{-1}. During the exercise bout the subjects were additionally allowed to drink water ad libitum (0–200 ml). During the exercise bout the subjects received on average 1.31 water, and 7.8 or 23.4 g of BCAAs for the low and high BCAA dose, respectively, or 3.9 g tryptophan during the tryptophan trial.

Blood analysis

Blood was sampled with heparinized syringes from a forearm vein using a catheter; patency was realized by flushing the catheter with saline. Blood samples were taken just before the start of the performance test and at the moment of exhaustion. Blood was immediately centrifuged to obtain plasma. An aliquot was directly frozen in liquid nitrogen for enzymatic determination of ammonia using a modification of the enzymatic determination with glutamate dehydrogenase performed on a COBAS B101 analyzer (Roche, The Netherlands) (Janssen, van Berlo, van Leeuwen & Soeters, 1988). Plasma for amino acid analysis was deproteinized with sulfosalicylic acid (6 mg (100 ml plasma)^{-1}), and stored at −80 °C until analysed by HPLC (Van Eijk, Van Der Heijden, Van Berlo & Soeters, 1988).

Rates of unidirectional tryptophan transport across the blood–brain barrier

The rate of unidirectional influx (v) of circulating plasma tryptophan to the brain may be estimated from the Michaelis–Menten equation:

\[ v = \frac{V_{\text{max}} [\text{Trp}]}{K_{\text{n(app)}} \text{Trp} + [\text{Trp}]} \times K_{\text{d}} [\text{Trp}] \]

where [Trp] is the plasma tryptophan concentration; K_{d} is the non-saturable transport constant; V_{\text{max}} is the maximal transport rate of tryptophan; K_{\text{n(app)}} is the apparent K_{m} of tryptophan in the presence of competing amino acids; K_{\text{m(app)}} is the absolute K_{m} in the absence of competing amino acids and K_{\text{m(app)}} is K_{m} of each competing amino acid. K_{\text{m(app)}} is calculated from the formula

\[ K_{\text{m(app)}} = \frac{K_{\text{m(app)}}}{1 + \sum ([AA]/K_{\text{m(app)}})} \]

(Pardridge, 1977). For the calculations of the rate of tryptophan

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**Figure 1. Time to exhaustion is given as means ± s.d. of ten endurance-trained subjects**

Subjects ingested one of four drinks which were given double-blind and in random order (4 ml (kg body weight)^{-1}) during the warming up and then 2 ml (kg body weight)^{-1} every 15 min during exercise. Drinks were (1) control (6% sucrose); (2) tryptophan (6% sucrose + 3 g 1% tryptophan); (3) low BCAA (6% sucrose + 0.6 g 1% BCAA); and (4) high BCAA (6% sucrose + 18 g 1% BCAA).
influx the $K_{ra}$, $V_{max}$ and $K_{in}$ were used, as reported by Hargreaves & Partridge (1988) for human brain capillaries studied in vitro.
Rates of tryptophan transport were calculated from these formulas
(Table 2) both using the plasma total tryptophan concentration
assuming that all tryptophan is available for transport (Partridge,
1983) or the estimated plasma-free tryptophan concentration
assuming that only free tryptophan is available for transport
(Chacoullif, Kennett, Serrurier, Marino & Curzon, 1986;
Newsholme et al. 1987). It is assumed that about 10% of the total
tryptophan is free (not bound to albumin) at rest and 30% is free
at exhaustion (Blomstrand, Cuhing & Newsholme, 1988).

Statistics
All data are given as means ± s.e. Differences among the four tests
were tested for significance with one-way repeated measure
ANOVA and location of significance was determined with the
Fisher's PLSD post hoc test. Two-tailed Student's $t$ tests were
used to determine differences between rest and exhaustion for the
different tests. Statistical significance was set at $P < 0.05$.

RESULTS

Performance
Time to exhaustion (Fig. 1) and heart rates (not shown)
were not different between the four tests. The individual
times to exhaustion are shown in Fig. 2. Figure 2 shows
that most of the variation in Fig. 1 was caused by the
intersubject difference in performance time and not by a
large intraindividual variation between treatments. The data

Table 1. Ammonia and amino acid concentrations at rest (pre) and at the moment of exhaustion (exh)

<table>
<thead>
<tr>
<th>Metabolite</th>
<th>Control</th>
<th>Tryptophan</th>
<th>Low BCAA</th>
<th>High BCAA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ammonia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>49 ± 18</td>
<td>43 ± 23</td>
<td>48 ± 25</td>
<td>68 ± 27</td>
</tr>
<tr>
<td>exh</td>
<td>105 ± 56†</td>
<td>93 ± 55†</td>
<td>157 ± 55*†</td>
<td>180 ± 56*†</td>
</tr>
<tr>
<td>Tryptophan</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>44 ± 7</td>
<td>47 ± 8</td>
<td>44 ± 5</td>
<td>45 ± 10</td>
</tr>
<tr>
<td>exh</td>
<td>43 ± 7</td>
<td>304 ± 61*†</td>
<td>40 ± 8†</td>
<td>38 ± 7†</td>
</tr>
<tr>
<td>Lysine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>130 ± 29</td>
<td>145 ± 16</td>
<td>155 ± 42</td>
<td>135 ± 22</td>
</tr>
<tr>
<td>exh</td>
<td>115 ± 13</td>
<td>108 ± 8†</td>
<td>252 ± 49*†</td>
<td>636 ± 133*§</td>
</tr>
<tr>
<td>Isoleucine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>62 ± 13</td>
<td>68 ± 7</td>
<td>80 ± 38</td>
<td>63 ± 13</td>
</tr>
<tr>
<td>exh</td>
<td>51 ± 8</td>
<td>48 ± 4†</td>
<td>102 ± 58*†</td>
<td>561 ± 137*§</td>
</tr>
<tr>
<td>Valine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>244 ± 42</td>
<td>259 ± 15</td>
<td>287 ± 81</td>
<td>252 ± 25</td>
</tr>
<tr>
<td>exh</td>
<td>214 ± 24†</td>
<td>216 ± 14†</td>
<td>503 ± 85*†</td>
<td>1200 ± 221*§</td>
</tr>
<tr>
<td>BCAA</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>443 ± 92</td>
<td>471 ± 35</td>
<td>521 ± 160</td>
<td>450 ± 54</td>
</tr>
<tr>
<td>exh</td>
<td>360 ± 42†</td>
<td>372 ± 23†</td>
<td>947 ± 186*†</td>
<td>2397 ± 483*§</td>
</tr>
<tr>
<td>Tyrosine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>65 ± 10</td>
<td>71 ± 14</td>
<td>88 ± 8</td>
<td>96 ± 11</td>
</tr>
<tr>
<td>exh</td>
<td>65 ± 10</td>
<td>66 ± 11</td>
<td>61 ± 9†</td>
<td>56 ± 11*†</td>
</tr>
<tr>
<td>Phenylalanine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>57 ± 8</td>
<td>60 ± 6</td>
<td>59 ± 8</td>
<td>58 ± 8</td>
</tr>
<tr>
<td>exh</td>
<td>54 ± 5</td>
<td>48 ± 4*†</td>
<td>52 ± 9†</td>
<td>51 ± 6</td>
</tr>
<tr>
<td>Methionine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre</td>
<td>10 ± 3</td>
<td>19 ± 4</td>
<td>17 ± 4</td>
<td>17 ± 4</td>
</tr>
<tr>
<td>exh</td>
<td>14 ± 2</td>
<td>15 ± 3</td>
<td>15 ± 6</td>
<td>12 ± 2</td>
</tr>
</tbody>
</table>

Values are means ± s.e. from ten subjects in nmol l⁻¹. Statistical significance, $P < 0.05$, is expressed as
follows: * vs. control at the moment of exhaustion, † rest vs. exhaustion and § high BCAA vs. low BCAA dose.
Table 2. $K_m$ (app)$^{Trp}$ and tryptophan influx rate to the brain at rest (pre) and exhaustion (exh)

<table>
<thead>
<tr>
<th>Supplementation</th>
<th>Control Trp</th>
<th>Low BCAA</th>
<th>High BCAA</th>
<th>Low BCAA</th>
<th>High BCAA</th>
</tr>
</thead>
<tbody>
<tr>
<td>$K_m$ (app)$^{Trp}$</td>
<td>1008</td>
<td>1087</td>
<td>1003</td>
<td>355</td>
<td>1286</td>
</tr>
<tr>
<td>($\mu$mol l$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>exh</td>
<td>872</td>
<td>1286</td>
<td>1286</td>
<td>872</td>
<td>1286</td>
</tr>
<tr>
<td>$v$ (influx) total Trp</td>
<td>26</td>
<td>25</td>
<td>26</td>
<td>26</td>
<td>25</td>
</tr>
<tr>
<td>(nmol min$^{-1}$ mg$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>exh</td>
<td>177</td>
<td>22</td>
<td>177</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>$v$ (influx) free Trp</td>
<td>2.6</td>
<td>2.6</td>
<td>2.6</td>
<td>2.6</td>
<td>2.6</td>
</tr>
<tr>
<td>(nmol min$^{-1}$ mg$^{-1}$)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>exh</td>
<td>54.9</td>
<td>6.6</td>
<td>54.9</td>
<td>6.6</td>
<td>6.6</td>
</tr>
</tbody>
</table>

For calculation of $K_m$ (app) and $v$ (rate of tryptophan influx) for total and free tryptophan concentration see Methods. Trp, tryptophan.

imply that administration of tryptophan or BCAA had neither a positive nor a negative effect on performance during prolonged cycle ergometer exercise.

**Amino acids and ammonia**

Ingestion of a mixture of BCAAs or tryptophan during exercise caused an increased plasma concentration of those amino acids at the moment of exhaustion (Table 1). The 3-fold higher BCAA dose in the high BCAA test significantly increased plasma BCAA concentration at the moment of exhaustion compared with the low BCAA test ($P < 0.0001$). The plasma BCAA concentration in the control and tryptophan test decreased with exercise. Tryptophan concentration decreased with exercise when BCAAs were ingested, whereas in the control test the concentration remained constant. Plasma ammonia increased at exhaustion in all four tests. The BCAA supplement, however, amplified the exercise-induced increase in ammonia concentration at the moment of exhaustion (Table 1).

**Unidirectional tryptophan transport across the blood–brain barrier**

We calculated the rate of unidirectional tryptophan transport across the blood–brain barrier (into the brain) from the concentrations of tryptophan and competing amino acids in the different treatments using kinetic parameters of transport of human brain capillaries measured in vitro (Hargreaves & Partridge, 1988). Calculations were made under two ultimate assumptions. In the first case it was assumed that total tryptophan was available for transport as suggested by Partridge (1983); in the second case it was assumed that only free tryptophan is available as suggested by Chaculoff et al. (1986b) and Newsholme et al. (1987). In the first case the rate of inward transport does not change when going from rest to exercise in the control treatment, while a 12–15% reduction occurs at exhaustion in the BCAA treatments and a 6.5-fold increase in the tryptophan treatment. In the second case the rate of inward transport is increased 2.9-fold when going from rest to exhaustion, while a 2.5- to 2.7-fold increase occurs in the BCAA treatments and a 20-fold increase in the tryptophan treatment.

**DISCUSSION**

In 1987, Newsholme and colleagues (Newsholme et al. 1987) proposed the 'central fatigue hypothesis' (for details see the introduction). One of the implications of this hypothesis is that oral ingestion of BCAA would reduce central fatigue and would enable athletes to maintain a higher pace during prolonged competitive exercise. Physical performance has previously been investigated in a field test by Blomstrand et al. (1991). They studied 193 male subjects running a marathon race. Subjects were randomly divided into an experimental group receiving 16 g of BCAA in plain water during the race and the placebo group receiving flavoured water. The subjects additionally had ad libitum access to carbohydrate-containing drinks and other drinks. No difference was observed in the marathon time of the group receiving BCAA and those receiving placebo.

However, when the original subject group was divided into groups of fast and slower runners, then a small significant reduction in marathon time was observed in the slower runners only. Three main criticisms can be raised against the design of the study of Blomstrand et al. (1991): (1) in a performance test investigating a potentially ergogenic effect subjects in the two groups should have been matched for previous performance, (2) carbohydrate intake and nutritional status should have been controlled, and (3) division of subjects in a group of fast and slower runners, taking an arbitrary marathon time as selection criterion is not in accordance with accepted statistical methods. Each of those points may have biased the data obtained by Blomstrand et al. (1991).

Here we fail to find an effect of oral ingestion of two doses of BCAA in a 6% saccharose solution on time to exhaustion in a controlled laboratory setting. The rate of ingestion of the low dose of BCAA is similar to that used in the study of Blomstrand et al. (1991). The rate of ingestion of the high dose of BCAA is 3-fold higher. In accordance with our data, Verger, Aymard, Cynobert, Anton & Luigi (1994) also failed to find an effect of intra gastric BCAA supplementation on time to exhaustion in rats running on a treadmill in comparison with a water control.
Another implication of the 'central fatigue hypothesis' is that oral ingestion of tryptophan could lead to premature fatigue. Here, however, we also fail to find an effect on time to exhaustion of an oral dose of tryptophan.

Controversy exists in literature as to whether total plasma tryptophan or free tryptophan (not bound to albumin) is available for transport across the blood–brain barrier. A correlation between plasma-free tryptophan and brain tryptophan concentration made Chauloff et al. (1985, 1986b) suggest that plasma-free tryptophan rather than total tryptophan is the determinant of increased brain tryptophan transport during exercise. However, it has also been proposed that binding of tryptophan to albumin appears to be of little importance for the uptake of tryptophan into the brain (Madras, Cohen, Messing, Munro & Wurtman, 1974; Yuwiler, Okendof, Geller & Braun, 1977; Pardridge & Feuer, 1969; Fernstrom & Fernstrom, 1993). Pardridge (1983) presented evidence suggesting that during the course of plasma flowing through the capillary, albumin-bound tryptophan molecules instantaneously dissociate into the free intermediate state and become available for reassociation with either albumin or the carrier-binding site. In the calculations of the inward transport rate of tryptophan across the blood–brain barrier (Table 2) we used the concentration of total and free tryptophan. When total plasma tryptophan is available for transport across the blood–brain barrier then no increase in the rate of tryptophan transport is observed when going from rest to exhaustion (Table 2). However, if the free plasma tryptophan is only available for transport then a 3-fold increase in the rate of tryptophan influx is observed at the moment of exhaustion. The effect of BCAA and tryptophan ingestion seems to be independent of whether total or free tryptophan is considered to be available for transport. In both cases BCAA ingestion reduced tryptophan uptake only minimally, while tryptophan ingestion greatly increased tryptophan uptake (Table 2).

The 6- to 20-fold (in the case of free tryptophan) increase in tryptophan transport does not seem to affect performance. This may imply that increased tryptophan influx across the blood–brain barrier does not increase the brain 5-hydroxytryptamine concentration in the human subjects. This could be due to a proportional increase in tryptophan efflux from the brain and/or a simultaneous increase in the activity of intraneuronal monoamine oxidase leading to an acceleration of the degradation of 5-hydroxytryptamine. In agreement with the latter possibility Grahame-Smith (1971) found that tryptophan administration only resulted in functional behavioural effects in the rat when monoamine oxidase activity was inhibited. In another study tryptophan administration only increased 5-hydroxytryptamine concentration in the brain after inhibition of monoamine oxidase activity (Marsden, Conti, Strope, Curzon & Adams, 1979). Another possibility is that brain 5-hydroxytryptamine concentration is increased after tryptophan ingestion, but that the increase in serotoninergic activity does not functionally contribute to fatigue and exhaustion.

A few studies with pharmacological agents seem to contradict the latter explanation. 5-Hydroxytryptamine agonists were reported to lead to premature fatigue during prolonged exercise in rat (Bailey, Davis & Ahlborn, 1993). A 5-hydroxytryptamine reuptake inhibitor was also reported to hasten fatigue during prolonged exercise in man (Wilson & Mangham, 1992). It is evident that more research is required in this area.

Both the low and high dose BCAA treatment increased plasma ammonia concentration at exhaustion as previously reported in healthy controls (MacLean & Graham, 1993) and in patients with McArdle's disease, a glycogen breakdown defect in muscle (Wagenmakers et al. 1990). In the patients, oral ingestion of BCAA (doses equivalent to the high dose used in this study) not only increased the ammonia production by the exercising muscle, but also caused premature fatigue during incremental exercise. The mechanistic explanation given for this observation was that increased oxidation of BCAA in muscle may use a carbon-dioxide on the tricarboxylic acid cycle as the BCAA amino- transferase reaction uses 2-oxoglutarate as an amino group acceptor. The possibility, therefore, should be considered that the large dose of BCAA may have a negative effect on muscle performance in conditions of a limited glycogen availability. Here, however, we have chosen to give BCAA in combination with carbohydrates as we aimed at improving performance via the potential inhibition of BCAA on brain uptake of tryptophan. Carbohydrate ingestion on its own may also reduce brain uptake of tryptophan as ingestion of carbohydrates reduces the increase in free fatty acids during prolonged exercise (Davis et al. 1992) and therefore may reduce the proportion of tryptophan present in the free form.

In conclusion, we fail to show an effect of oral ingestion of BCAA or tryptophan in a carbohydrate-containing solution on time to exhaustion in a controlled laboratory study. This implies that oral ingestion of these amino acids either does not change the 5-hydroxytryptamine concentration in relevant local areas in the brain or that a change in serotoninergic activity during prolonged exercise contributes little to mechanisms of fatigue and exhaustion.


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