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Changes in plasma taurine levels after different endurance events

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Summary. The sulphonated amino acid taurine increased significantly in the plasma of trained athletes after three endurance exercises of different duration and intensity, a 90 min run on a treadmill at 75% of an individual's VO_2 peak, a Marathon, 42.2 km and a 100 km run, by 19%, 77% and 36%, respectively. Such results indicated that the speed at which the exercise is performed, referred to as the intensity, rather than the duration of the exercise, correlated with the elevated taurine levels possibly indicating its release from muscle fibres. The plasma amino acid pool decreased significantly in relationship with the duration of the exercise, caused by their utilisation for glucogenesis. The possible sources of the increased plasma taurine are discussed.

Keywords: Amino acids – Taurine – Marathon – Endurance exercise

Introduction

Taurine, 2-aminoethane sulfonic acid, a non-essential amino acid, is present in muscle at relatively high concentrations. It is more abundant in slow oxidative type I fibres than in type II fibres (Dunnett et al., 1992). Taurine clearly plays a role in the regulatory mechanism of Ca^{++} homeostasis within heart muscle during contraction as well as increasing the sensitivity of force generating myofilaments to calcium (Steele et al., 1990). Its possible interaction with head groups of neutral phospholipids may mediate membrane conformational changes although extensive debate continues (reviewed in Schaffer et al., 1995). In addition taurine may alter the excitability of the muscle membrane by changing membrane chloride conductance (Conte Camerino et al., 1987).

What effect different exhaustive exercises may have upon muscle taurine levels in man has not been described previously but may be indirectly ascertained by assaying changes in the plasma taurine content. We have therefore assayed the plasma concentrations of taurine after three different endurance exercises together with alterations in the plasma amino acid pool.

Methods

Subjects and sampling procedure

Male athletes undertaking regular running exercise participated in each of these studies. Ethical permission for each of the studies was obtained from the Sports Medicine Department of the University of Louvain, Belgium, while written consent was obtained from each participant.

Kinetics of plasma taurine after exercise

A sedentary healthy 35 y man of reasonable fitness (weight: 100 kg – height 190 cm) exercised to 75% of his VO_2 peak (previously determined at 46 ml·min^{-1}·kg^{-1} by a classical incremental test) on a treadmill for 1.5 h. A blood specimen was taken prior to the exercise, immediately on its completion and then at 1, 2, 4, 8, 24 and 48 h after. Blood specimens were placed in lithium heparin bottles on ice, centrifuged immediately at 3,000 rpm for 15 min and the plasma stored at – 20°C prior to analyses.

Marathon

Eight trained male athletes participated in this study. Their average age was 35.6 y (SD 7.1, range 24–47) weight 70.0 kg (SD 5.2, range 63–77), height 175.8 cm (SD 3.9, range 169–180). Each subject completed a Marathon, commencing at 12.00 h and finishing in a mean time of 205.2 min (SD 18.2, range 179–235). The runners drank freely of the beverage (*Isostar*) provided by the race organisers which did not contain taurine. The air temperature was approximately 8–9°C during the race. A heparinised blood specimen was taken from the antecubital vein before and within 60 minutes of the completion of the Marathon, and processed as above.

100 km runners

Four trained male athletes participated in this study. Their average age was 42.8 y (SD 3.8, range 39–46), weight 76.3 kg (SD 10.7, range 72–89) and height 183.0 cm (SD 9.0, range 175–195). Each subject completed a 100 km run, starting at 06.00 h and finished at approximately 16.00 h. Intake of nutrition during the race was recorded, liquid intake was approximately 3.0 l (*Extra Orange*) with a solid intake every two hours (*Power Bar*); neither of these nutrients contained taurine. A heparinised blood specimen was taken from the antecubital vein 20 minutes before and within 30 minutes of the completion of the 100 km run, and processed as above.

Control subjects

Six healthy sedentary male subjects acted as control subjects. Their age was 42.2 y (SD 4.7, range 39–49), weight 84.5 kg (SD 7.0, range 82–91) and height 1.90 cm (SD 7 cm, range 181–197). Blood samples were collected at 08.00 h and at 17.00 h and processed for their amino acid content as described above.

Analyses of plasma taurine and amino acids

Plasma amino acids were assayed by an automated HPLC technique, (*Pharmacia Biotech biochrome 20*) system, with UV detection after reaction with ninhydrin reagent. Amino acid pool is defined as the sum of leucine, isoleucine, valine, phenylalanine, tyrosine, glutamine, glutamic acid, aspartate, lysine, histidine, arginine, methionine, threonine, serine, alanine, glycine, proline and cystine.

Statistics

Results are presented as mean ± standard deviation. The between subject differences were assessed by ANOVA design and by a Bonferroni post-hoc test, while the percentage changes in taurine and the amino acid pool between the different exercises were analysed by ANOVA with Tukey-Kramer procedure. The within subject effect (prior to and post event) versus control conditions was assessed by means of a repeated measures ANOVA design. Prior to all tests, the distributions of different variables were verified for normality by a Kolmogorov-Smirnov test. Application conditions of parametric tests were achieved for all variables. The level of significance was set at $P < 0.05$.

Results

Figure 1 shows the plasma changes in both taurine and the total amino acid content after exercise under 75% of VO_2 peak conditions in one individual. After completion of the exercise, a small increase of taurine concentration, 5%, was assayed. An hour later, the level of taurine had increased by 19% in comparison to the pre-exercise value. After this time point plasma taurine decreased, the kinetics clearly showing two components during the subsequent 48h. Changes in the plasma amino acid content were apparent only immediately at the cessation of exercise, a 11% increase, and 4h after the completion of the exercise when there was a decrease of 18%. Ten hours after the completion of the exercise each of the amino acids had returned to their appropriate reference ranges.

Figure 2 (top) compares the changes in plasma taurine content before and after the completion of either the Marathon, 42.2km, or the 100km, (the

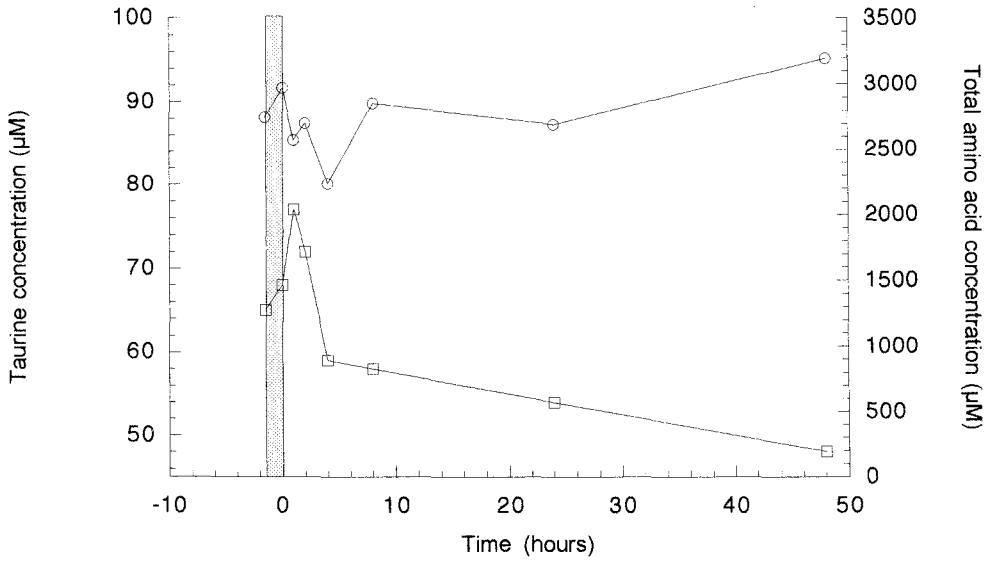

Fig. 1. Changes in the plasma taurine concentration (□) and the total amino acid pool (○) in an individual exercising 90min at 75% of the VO_2 peak before and at various time points after the exercise (the shaded area indicates the period of exercise)

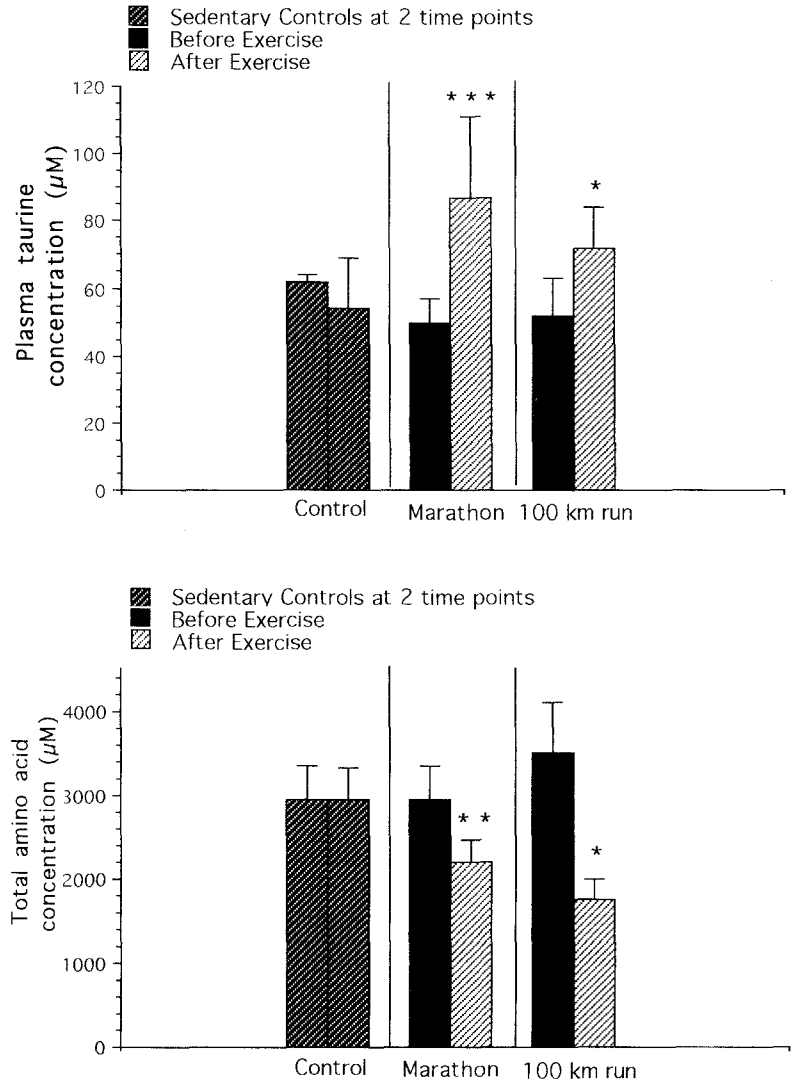

Fig. 2. Mean (±SD) plasma taurine concentration (top) and total amino acid pool (bottom) in marathon and the 100km runners before and after the endurance event in comparison to age and sex matched control subjects. Significance is shown as: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

samples being taken within the first hour after the completion of each race) and compared with the values of the sedentary control subjects. Even though the pre-race mean value for taurine in the Marathon runners was lower than in the controls, ANOVA analysis of the pre-race values did not show any differences between the mean values for the three groups. At the completion of the Marathon and 100km run, a significant increase in the mean plasma taurine content was discernible, $P = 0.001$ and $P = 0.041$, respectively. The intensity of the taurine elevation was highest in the Marathon runners, 77 ± 34%, while a lower value of 36 ± 26% was evident in the 100km runners, by comparison to the pre-race value. Furthermore the percentage increase in

plasma taurine levels was significantly greater in the Marathon runners by comparison to the 100 km runners, $P < 0.01$.

Figure 2 (bottom) shows the changes in the total amino acid pool after either the Marathon or 100 km run. Before the commencement of either exercises there was no significant difference between the mean value of the total amino acid content in any of the three groups. At the completion of either the Marathon or the 100 km run, there were significant decreases, $P = 0.03$ and $P = 0.012$ respectively, the greater decrease being associated with the longer endurance event. The decrease in the total amino acid pool, by comparison to the pre race value, was significantly greater in the 100 km runner, 52 $\pm$ 11% by comparison to that of the Marathon runners, where its decrease was only 25 $\pm$ 14.

Discussion

In this present study we have shown that the plasma taurine content significantly increased in each of the athletes after either a 42.2 km or a 100 km run and also in an individual exercising to 75% of his VO_2 peak in comparison to their respective pre-exercise value. In addition, the higher increase in the plasma taurine content in Marathon runners by comparison to the 100 km runners may indicate that duration of the exercise is not the prime determining factor in the elevation of plasma taurine, but that the speed at which the exercise was performed, i.e. km/min, referred to as the intensity of the exercise, is a more important factor. The Marathon runners completed a kilometre in an average time of 4.9 minutes by comparison to the 100 km runners who took 7.2 minutes.

Taurine is the end product of the metabolism of sulphur containing amino acids namely cysteine and methionine, and, has received little attention in other studies of the effects of exhausted exercise on the amino acid pool. The relevance of changes in its plasma concentration is unknown; in catabolic diseases, such as HIV, there are increased plasma levels of taurine (Hortin et al., 1994) while patients presenting with first time ischaemic cardiac pain showed significantly elevated blood taurine levels, (Bhatnager et al., 1990).

The source of the increased taurine plasma content after exercise requires elucidation as this may reflect muscle damage, muscle fatigue or some adaptation to changes in osmolarity within the blood.

The selectivity and high concentration of taurine in different muscle types exemplifies the point that specific transporters and channels must exist within the muscle membranes for its import and export. The entry of taurine into the cell is by means of a specific sodium dependent transporter (Huxtable, 1992) while its release is less well characterised but is possibly via a sodium independent mechanism (Pasantes-Morales et al., 1990). The skeletal muscle remains the most plausible candidate to explain the increase in plasma taurine concentration assayed after the endurance exercises. The peak in the plasma taurine content occurred immediately at cessation of exercise which does not parallel the increase in creatine kinase, a muscle marker of membrane disruption, which is known to occur approximately 24–48 hours later. The release of

the taurine from the muscle could be related to variety of factors including changes in electrophysical properties of the muscle membrane, its co-release with water to maintain plasma volume and consequently in some undefined way, Ca^{++} homeostasis.

A redistribution of taurine from other blood constituents, e.g. platelets and erythrocytes could occur, due to the change in plasma osmolarity induced by exercise, in part caused by dehydration and the significant depletion of the plasma amino acid pool. Plasma volume decreases after a marathon, between 4.3% and 12.1% (Rocker et al., 1989; Whiting et al., 1984). However, the mean percentage increase in plasma taurine content in the man exercising to 75% of his VO_2 peak and both the Marathon and the 100 km was considerably higher that this figure, 19%, 77% and 36%, respectively, such that alterations in plasma volume alone do not account for the increased plasma taurine content.

Changes in the excretion of taurine by the kidney may also occur due to changes in osmolarity within the blood. Renal clearance of taurine decreased by ~50% in 100 km runners (Décombaz et al., 1979) and could in part explain the increase of plasma taurine in such runners.

However if taurine clearances are calculated in the 100 km runners investigated in this present study inserting the values for the clearance of taurine from the publication of Décombaz et al. (1979) and assuming a space of distribution of 60% of the body mass i.e.

$$([\text{plasma taurine}]_{\text{before}} \times \text{clearance}_{\text{before}}) - ([\text{plasma taurine}]_{\text{after}} \times \text{clearance}_{\text{after}}) \times \text{race time/space of distribution},$$

the calculated result of 1.87 μM approximates to only 10% of the increase in plasma taurine concentration assayed in these runners.

During prolonged exercise, depletion of glycogen stores in the muscle occurs, which results in gluconeogenesis using non-essential amino acids as substrates. Alteration in the concentrations of a number of amino acids have been reported after the completion of a Marathon, (Conlay et al., 1989; Blomstrand et al., 1988 and 1992; Bazzarre et al., 1992) or a 100 km run (Decombaz et al., 1979). Extensive changes in the amino acid pool occurred in both endurance events in this present study, which was associated with the time of endurance of the exercise, a decrease of 25% and 52% respectively.

In conclusion in these present studies we have shown that after three different types of endurance exercises, there are significant increases in the plasma concentration of taurine. Changes in plasma volume and urinary clearance may contribute to the increase in plasma taurine, although its release from muscles may be a major factor for its elevation in the blood, which is possibly related to the control of osmolarity and indirectly Ca^{++} homeostasis.

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