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The effect of a taurine-containing drink on performance in 10 endurance-athletes

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Summary. To determine the effect of a taurine-enriched drink "Red Bull" on performance, 10 endurance-athletes performed three trials. After 60 min. cycling at approximately 70% VO_2 max, the subjects pedalled to exhaustion on a cycle ergometer. During each exercise, the subjects received 500 ml of a test-drink after 30 min. submaximal cycling: "Red Bull" without taurine, without glucuronolacton (U1), "Red Bull" without taurine, without glucuronolacton, without caffeine (U2) and "Red Bull" original drink containing taurine, glucuronolacton and caffeine (U3).

The heart rate level was significantly lower in U3 ($p = 0{,}0031$) 15 min. after application. The plasma catecholamines increased slightly from begin of exercise to 15 min. after application of the drinks in all trials but remained on a significantly lower level in U3 (epinephrine ($p = 0{,}0011$) and norepinephrine ($p = 0{,}0003$). Endurance time was significantly longer with "Red Bull" original in U3 ($p = 0{,}015$). The results of this study show a positive effect of a taurine-containing drink on hormonal responses which leads to a higher performance.

Keywords: Amino acids – Taurine – Heart rate – Catecholamines – Performance

Introduction

Many athletes use energy-drink "Red Bull" because of its highly stimulating effect to improve physical and psychological performance.

"Red Bull" consists of many different substances of which the following seem to be remarkable:

1. Saccharose and glucose: it is well known that the application of carbohydrates before or during exercise improves physical performance (Sherman et al.,

1989; Geiß et al., 1993). Higher concentrations of blood glucose lead to an increased intracellular utilisation of glucose and higher rates of carbohydrate oxidation.

2. Caffeine: sportmedical examinations showed a positive effect on endurance performance but it has to be mentioned that the increase of performance could be caused by psychological effects too (Costill et al., 1978).

3. Taurine: the effect of taurine on performance was explored by M. Ono et al. (Ono et al., 1987). Their studies demonstrated many indirect, metabolic effects of taurine, which were held responsible for improving performance. But all exercises were made under low-carbohydrate diets, which contradicts a regular sports nutrition.

4. Glucuronolactone: the effect of glucuronolactone on biochemical and physiological processes has yet to be achieved. A detoxification of performance decreasing substrates of metabolism might be possible.

The purpose of this study, therefore, was to examine whether a taurine-containing drink, ingested during exercise, influences performance and the metabolic responses to prolonged exercise.

Methods

Subjects

Ten endurance trained male athletes served as subjects. Their mean age was 24,5 ± 3,5 years, the mean body mass 78,8 ± 8,3 kg and mean height 186 ± 15 cm. They trained for 10–15 hours per week. All subjects were informed about the nature of the experimental procedures and of the possible risks involved before giving their voluntary consent to participate. The protocol was approved by the Medical Ethical Review Committee of the Landesärztekammer Hessen/Germany.

Study design

The experiments were conducted during a three-week period when the subjects' training schedules were held constant. The volunteers followed a regular meal plan and exercise regimen so that no variation in either pre-exercise food consumption or exercise occurred (Geiß et al., 1991). The subjects were required to fast for at least 2 hours prior to each exercise trial. Prior to exercise a venous cannula was inserted into an antecubital vein in the right forearm for blood sampling. Each subject completed three trials in which they performed 60 min. of cycling at approximately 70% VO_2 max on cycle ergometer. After this period of submaximal cycling the workload on the ergometer was increased every three minutes for 50 Watts until the subject was unable to continue. The end point was defined as the point at which pedalling dropped below 10% of the set rate because of muscle soreness or fatigue.

24 hours later the subjects had to perform a cycling exercise starting with 50 Watts. Again the workload on the ergometer was increased every three minutes for 50 Watts until subjects' exhaustion (Fig. 1).

The drinks were consumed after 30 min. submaximal cycling:

U1 = "Red Bull" without taurine, without glucuronolacton, with caffeine (160 mg), with glucose (10,5 g), with saccharose (43 g) (500 ml)

U2 = "Red Bull" without taurine, without glucuronolacton, without caffeine, with glucose (10,5 g), with saccharose (43 g) (500 ml)

U3 = "Red Bull" original drink containing taurine (2 g), glucuronolacton (1,2 g), caffeine (160 mg), with glucose (10,5 g), with saccharose (43 g) (500 ml)

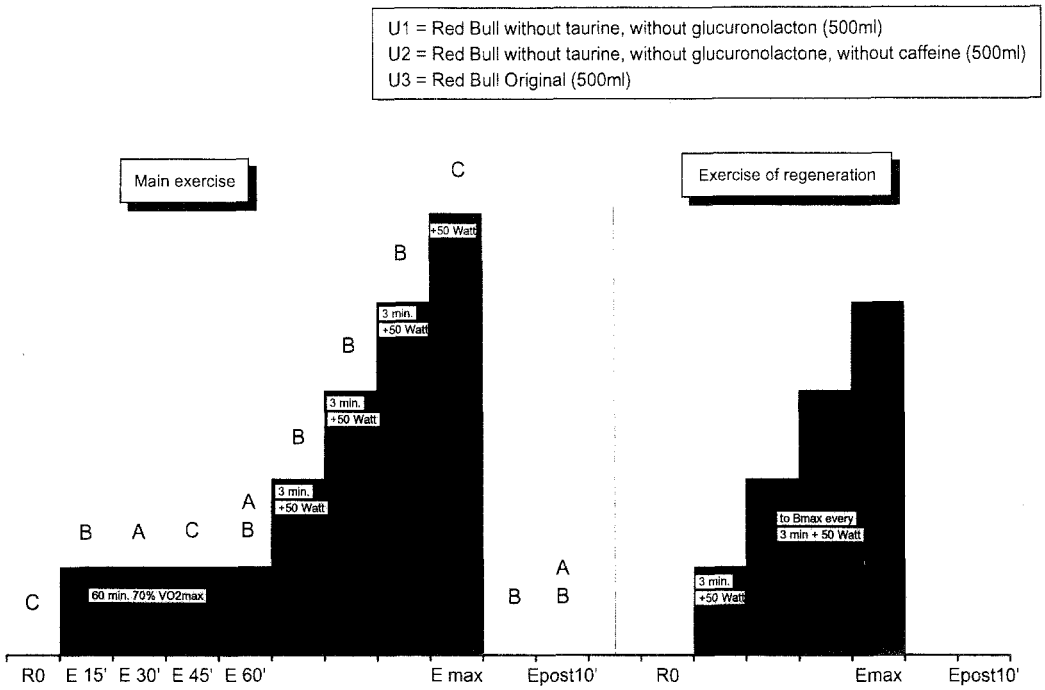

Fig. 1. Study design. Determination of: *A* glucose; *B* lactate; *C* glucose, lactate, insulin, free fatty acids, epinephrine, norepinephrine, human growth hormone; *R* rest; *E* effort

The effect was examined using double-blind test method, that means in this case none of the subjects and examinator knew what kind of drink was applied at which number of trial.

Equipment

All the experimental trials were conducted using an Ergo-Fit 777® cycle ergometer. The heart rate using a Sport Tester™ (Polar Elektro, Finland) was recorded continuously throughout the exercise.

Blood collection and analysis

Blood samples were taken just before the start of exercise and then after every 15 min. throughout the experiment, after every workload-increase, at Emax, 4 and 10 min. after exercise (Fig. 1). Serum glucose was analysed by the glucose hexokinase method (Reflotron®, Boehringer Mannheim, Germany) and blood lactate by enzymatic colorimetric method (Miniphotometer 8®, Fa. Dr. Lange, Germany). Plasma insulin was analysed by an enzymeimmunoassay (MEIA) (IM®x, Abbott GmbH, Germany) (Jacobs, 1988), plasma free fatty acids (FFA) by an automated enzymatic colorimetric method (NEFA C®, Wako Chemicals GmbH, Germany) (Trout et al., 1969), catecholamines epinephrine and norepinephrine were analysed by a radioimmunoassay (Amicyl-Test™ Katcombi, IBL, Germany) (IBL, 1993) and human growth hormone (hGH) was analysed by a radioimmunoassay (hGH RIA®, Kabi Pharmacia Diagnostics, Sweden) (Livesey et al., 1980). Blood for immunoassays was drawn into an EDTA coated blood collection tube and the sample spun at 4000 rpm for 5 minutes. Plasma was taken off, divided into two storage tubes and frozen at −20°C until analysed.

Statistics

Results are expressed as mean ± standard deviation (SD). Significant differences between means were determined by the Student-t-test for paired data and crosschecked using two-way analysis of variance for repeated measures. The probability level for significance was set at $p < 0{,}05$.

Results

Heart rate

After start of exercise heart rate levels rose rapidly and plateaued to E 60′ (U1: 119,0 ± 4,58; U2: 117,6 ± 4,01; U3: 114,5 ± 4,06 sec) during cycling at 70% VO_2 max. Throughout increasing workload the heart rate had risen gradually and reached a peak at Emax (U1: 187,8 ± 3,09; U2: 188,7 ± 3,03; U3: 190,3 ± 1,83 sec). The heart rate level in U3 was significantly lower at E 45′ (113,1 ± 4,98 sec; $p = 0{,}029$), at E 60′ (114,5 ± 4,06 sec; $p = 0{,}0031$) and E 3′ (132,7 ± 3,87 sec; $p = 0{,}0295$) than in U1 (117,6 ± 4,98; 119,0 ± 4,58; 137,1 ± 3,78) (Fig. 2).

In the regeneration trials heart rate rose with increasing workload and no statistically differences between the three treatments were observed.

Blood glucose

Blood glucose levels were in all three trials very similar and no significant differences could be found. The blood glucose level showed from R0 (U1: 92,6 ± 5,85; U2: 91,6 ± 6,36; U3: 94,2 ± 8,50 mg/dl) to E 30′ (U1: 83,2 ± 3,38; U2: 82,4 ± 5,60; U3: 84,4 ± 9,21 mg/dl) a light decrease after 30 min. cycling at

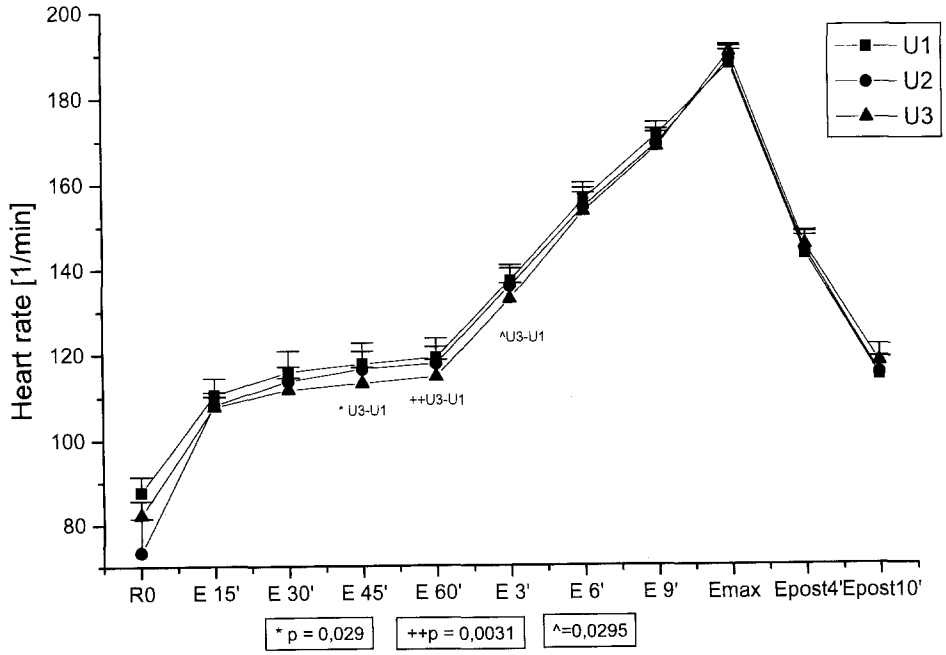

Fig. 2. Heart rate

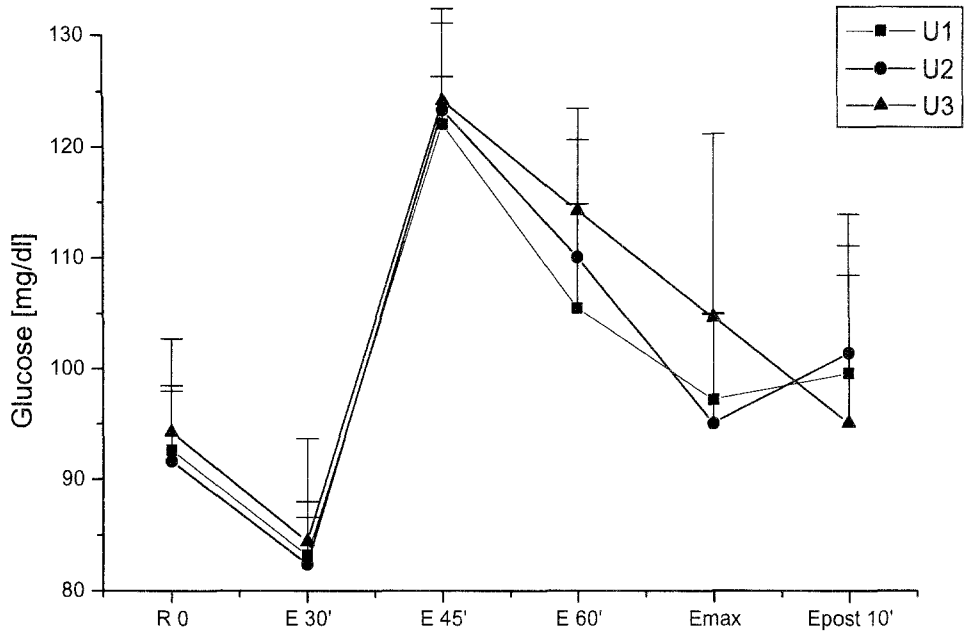

Fig. 3. Blood glucose

70% VO_2 max. After drink application the blood glucose concentration rose rapidly, reaching a peak at E 45′ (U1: 122,0 ± 4,33; U2: 123,3 ± 8,99; U3: 124,1 ± 6,92 mg/dl) and then decreased during exercise to Epost 10′ (U1: 99,5 ± 8,86; U2: 101,3 ± 12,48; U3: 95,0 ± 16,01 mg/dl) in all three trials (Fig. 3).

Blood lactate

In all trials the blood lactate concentration was throughout the period of submaximal cycling lower than 4 mmol/l (U1: 1,82 ± 0,21; U2: 1,58 ± 0,37; U3: 1,50 ± 0,31 mmol/l). With increasing workload blood lactate levels rose gradually and reached a maximum concentration at highest load-intensity (Emax) (U1: 14,50 ± 1,36; U2: 14,68 ± 1,98; U3: 16,30 ± 1,94 mmol/l). After exercise lactate decreased at Epost 10′ (U1: 10,31 ± 1,03; U2: 11,17 ± 2,54; U3: 11,80 ± 2,15 mmol/l). No significant differences in blood lactate levels of U1, U2 and U3 could be observed (Fig. 4).

Plasma insulin

15 min after drink application, plasma insulin concentration in U3 (15,07 ± 4,41 μU/ml) was significantly lower than in U1 (26,39 ± 10,99 μU/ml) ($p = 0{,}0172$). No differences were observed between exercises at Emax (Fig. 5).

Plasma FFA

After ingestion of the test drinks, plasma FFA means increased from resting value R0 (U1: 3,61 ± 2,35; U2: 3,04 ± 1,95; U3: 6,47 ± 6,36 mg/dl) to E 45′ (U1:

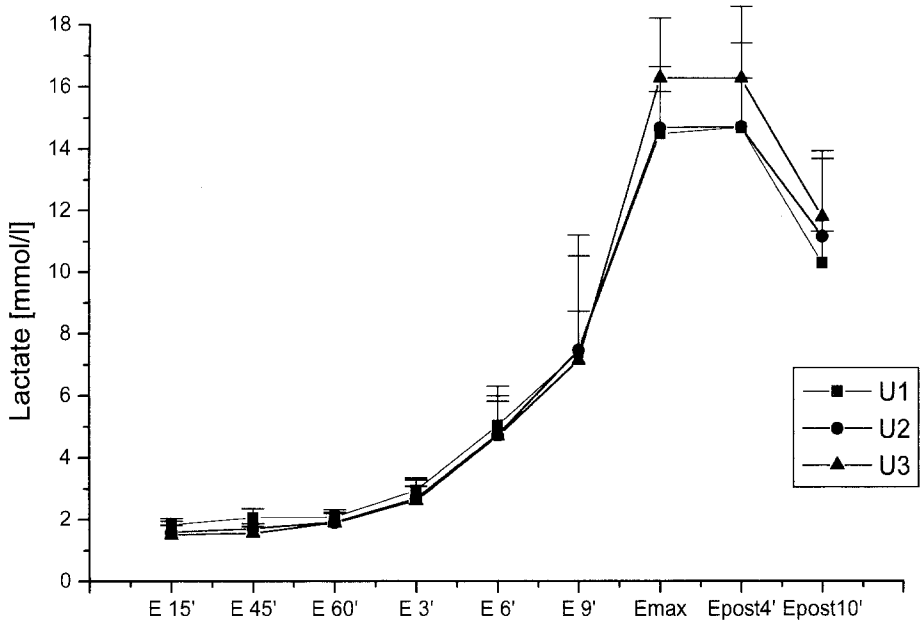

Fig. 4. Blood lactate

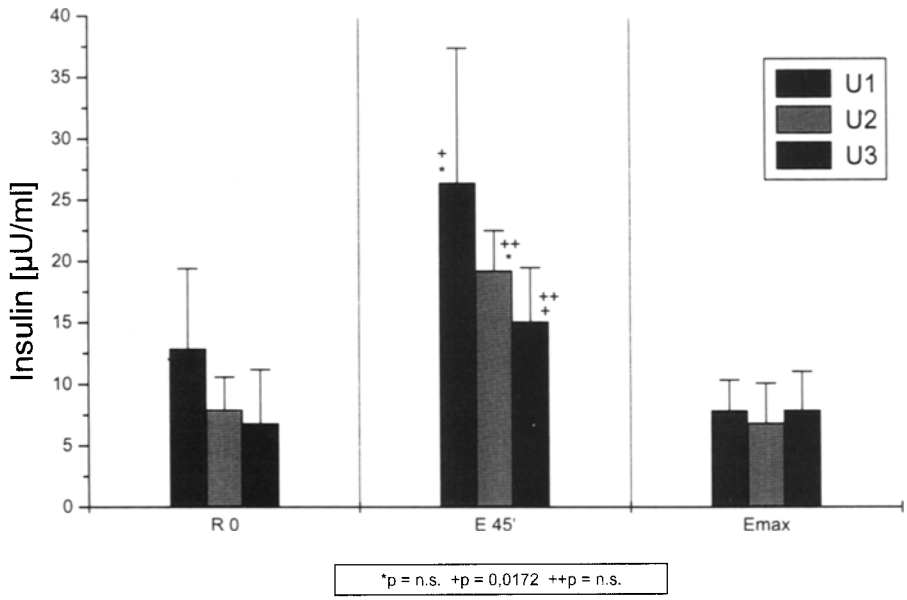

Fig. 5. Insulin

5,12 ± 3,85; U2: 4,80 ± 2,03; U3: 6,92 ± 6,41 mg/dl) and declined during exercise with high intensity at Emax (U1: 0,97 ± 0,75; U2: 1,06 ± 0,82, U3:2,00 ± 1,61 mg/dl). But no statistically significant difference between the three treatments for FFA concentration could be found (Fig. 6).

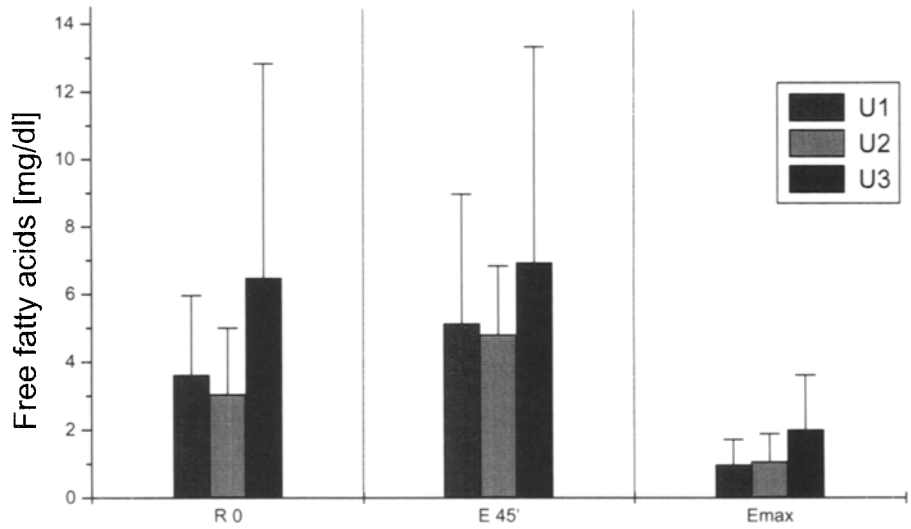

Fig. 6. Free fatty acids

Plasma hGH

During exercise plasma hGH levels rose gradually from R0 to Emax. Because of high SD, no significant differences between the trials were observed.

Catecholamines

Epinephrine

Plasma epinephrine concentration increased slightly form start of exercise (R0) (U1: 31,1 ± 3,01; U2: 23,7 ± 8,52; U3: 20,2 ± 5,65 pg/ml) to E 45′ (U1: 44,8 ± 10,81; U2: 30,2 ± 12,56; U3: 26,4 ± 8,29 pg/ml) in all trials. The epinephrine level in U3 remained significantly lower than in U1 ($p = 0{,}0011$). During the late phase of exercise the level of plasma epinephrine increased in all treatments, but at Emax plasma epinephrine had risen significantly lower in U3 (U3: 267,0 ± 125,88 pg/ml) than in U2 (U2: 449,5 ± 196,56 pg/ml) (Fig. 7a).

Norepinephrine

During exercise the plasma norepinephrine level increased slightly from R0 (U1: 197,9 ± 18,99; U2: 186,8 ± 51,00; U3: 159,5 ± 21,55 pg/ml) to E 45′ (U1: 415,2 ± 80,21; U2: 261,2 ± 84,15; U3: 211,5 ± 32,73 pg/ml) and rose rapidly to Emax (U1: 2929 ± 995,98; U2: 2439 ± 744,20; U3: 1499 ± 393,75 pg/ml). At E 45′ and Emax the norepinephrine concentration in U1 had risen significantly higher than in U3 ($p = 0{,}0003$ at E 45′ and $p = 0{,}0023$ at Emax) (Fig. 7b).

Endurance

Endurance time on the individual maximal intensity level was significantly longer in U3 (857,8 ± 236,4 sec; $p = 0{,}0115$) and in U2 (791,8 ± 188,52 sec;

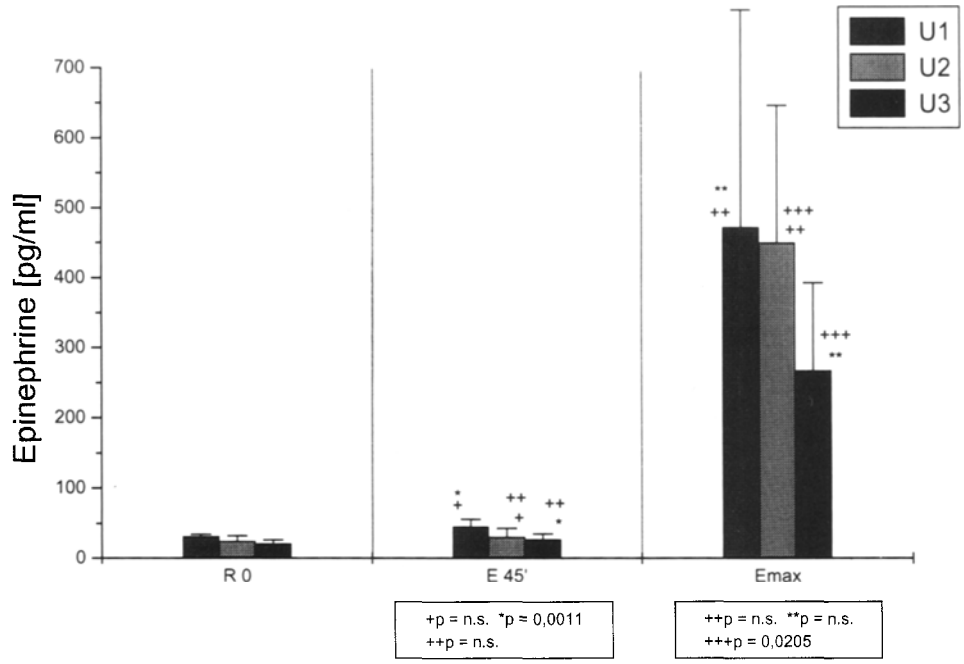

Fig. 7a. Epinephrine

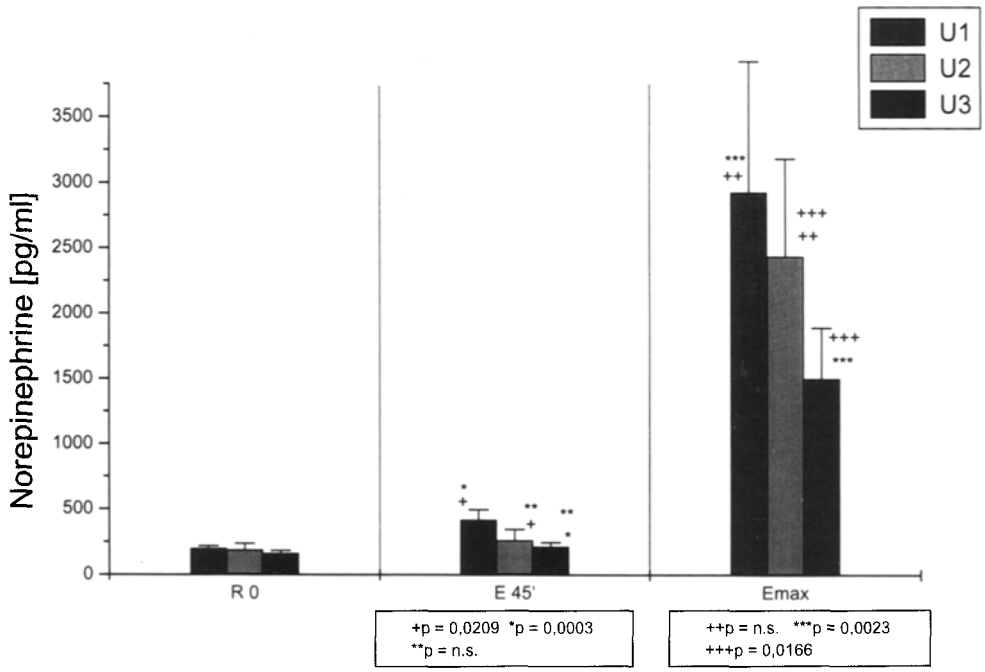

Fig. 7b. Norepinephrine

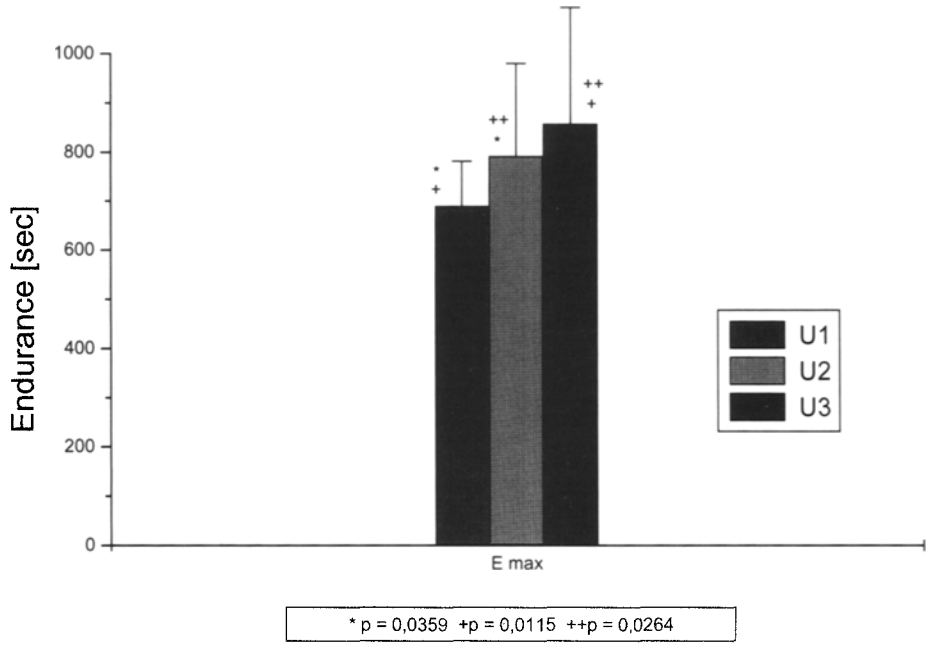

Fig. 8a. Endurance

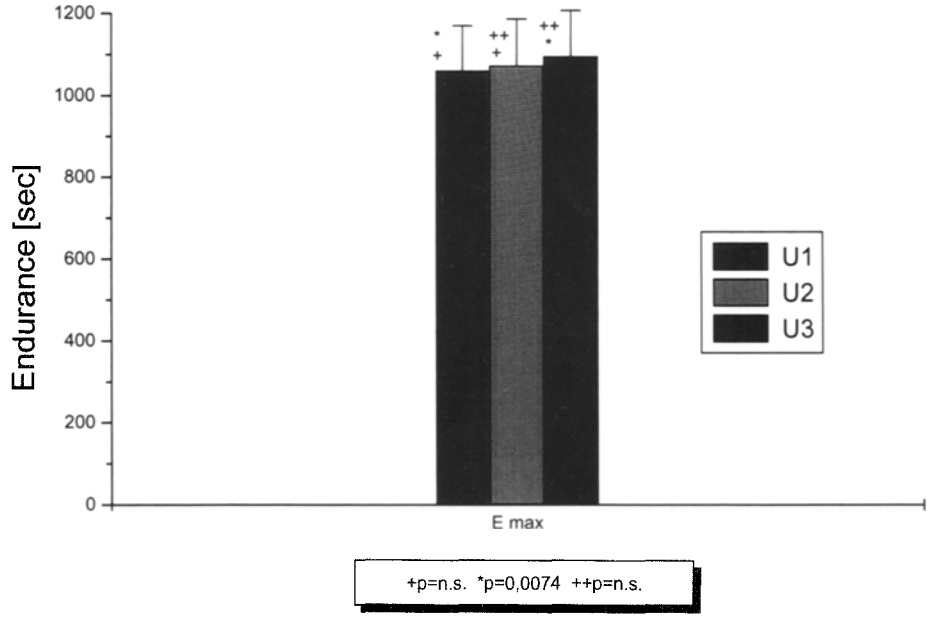

Fig. 8b. Endurance-regeneration

$p = 0{,}0359$) than in U1 ($689{,}3 \pm 92{,}35$ sec). Endurance time in U3 was also significantly longer than in U2 ($p = 0{,}0264$) (Fig. 8a).

In the exercise of regeneration endurance time was again significantly longer in U3 (1095 sec) than in U1 (1060 sec) ($p = 0{,}0074$) (Fig. 8b).

Discussion

The major result of this study was a significant increase in performance during exercise after an intake of the original taurine-containing drink "Red Bull". It has been demonstrated, that the heart rate and catecholamines concentrations reached a lower level with original "Red Bull", than with the two drinks in U1 and U2, which did not contain taurine. This effect led to a significantly prolonged endurance ($p = 0{,}0115$), which had been found even in the regeneration exercise 24 hours later ($p = 0{,}0074$). A conclusion to be drawn of this result needs a view on the effects of taurine:

Studies of M. Ono et al. (1987) have demonstrated a cardiac effect of taurine during exercise. He observed, that an increase of serum creatine kinase (CK) and isoenzyme CK-MB before to 24 hours after exercise was prevented by taurine. Taurine administration proved to inhibit the degree of increase in heart rate and increased the maximal degree of pulse pressure in response. The result of this study goes along with previous studies, demonstrating an interrelationship between taurine and the sympathetic system in the periphery. In the heart taurine antagonizes the stress-induced increase in cyclic nucleotide content (Mal'Chikova et al., 1979). Taurine increases the turnover of cAMP in the heart, stimulating both adenylate cyclase and phosphodiesterase (Mal'Chikova and Flizarova, 1981).

On the one hand taurine decreases the norepinephrine levels in blood, which was observed in our study in U3 on highest intensity level during exercise, on the other hand it minimizes the binding of catecholamines on heart muscle cells, examined by Franconi et al. (1983). These effects protect the heart of stress caused by high release of catecholamines. This taurine-induced mechanism goes along with a more economic cardiac action, reduces heart rate and improves performance, which was observed in our study; while endurance was prolonged, heart rate had been at a lower level during exercise.

Another important biochemical action of taurine is on calcium movements. Taurine modulates the Ca^{++} storage capacity of the sarcoplasmic reticulum (Huxtable and Bressler, 1973) and stimulates the pumping rate of Ca^{++}-activated ATPase pumps (Pasantes-Morales, 1982; Pasantes-Morales et al., 1982). These pharmacological and physiological actions of taurine are very similar to the characteristics of digitalis. The inotropic functions of digitalis are due to an increased rate and amount of Ca^{++} to the myofibrillar contractile proteins. Furthermore, taurine exerts a heart protective effect and improves the symptoms of congestive heart failure (Azuma et al., 1985) and acute myocardial infarction (Chazov et al., 1974).

Along with other inhibitory amino acids, taurine blocks the elevation in cAMP concentrations in brain slices induced by cysteine sulfinate and antagonizes the stimulatory effects of norepinephrine, adenosine and histamine on hippocampal cAMP concentrations (Baba et al., 1982). Central administration of taurine decreases heart rate and sympathetic nerve activity (Bousquet et al., 1981; Inoue et al., 1985). Taurine suppresses the K^{+}-evoked release of norepinephrine from the superior cervical ganglion and from cerebral cortical slices

(Muramatsu et al., 1978) and inhibits the release of preloaded norepinephrine from synaptosomes (Huxtable, 1992).

These effects were not examined in our study but would be compatible with our findings.

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