

Table 1

Patient	Plasma $\mu\text{mol/l}$		Muscle $\mu\text{mol/l}$ intracellular $\text{H}_2\text{O}$	
	Before	After 10 weeks	Before	After 10 weeks
1	45	2481	15 039	36 053
2	96	–	5600	–
3	40	712	9150	32 174
4	41	–	9771	–
27 controls		49 (3) <sup>a</sup>		19 194 (3378) <sup>a</sup>

<sup>a</sup>Mean (SD).

### Accumulation of taurine in patients with renal failure

Sir,

The amino acid taurine (2-aminoethanesulphonic acid) is present in high concentrations in mammalian tissues, especially skeletal muscle, heart and the central nervous system. Taurine has several beneficial physiological and biochemical effects *in vitro* and *in vivo* in experimental animals. It has cardiogenic actions, participates in osmoregulation, stabilizes the membrane potential in skeletal muscle, affects calcium ion kinetics, has antioxidant and anti-inflammatory properties and acts as a neurotransmitter [1]. Clinical studies suggest that oral treatment with taurine improves symptoms and cardiac performance in humans with congestive heart failure [2].

Taurine is an ingredient in some so-called energy drinks, which also contain caffeine, carbohydrate and B vitamins. Such drinks are taken to improve physical performance, although there is little evidence that taurine *per se* exerts any beneficial effects in healthy individuals or animals without taurine depletion. It has been suggested that daily intake for 3 weeks of 0.5 l Red Bull<sup>®</sup>, providing 2 g of taurine and 1.2 g of glucuronolacton, increases endurance time slightly at maximum intensive exercise level, compared with drinks containing the same ingredients as Red Bull<sup>®</sup> but without glucuronolacton and taurine [3]. Hence, the effect might have been due to either of these components.

Patients with end-stage renal disease are reported to be taurine depleted with low plasma and muscle intracellular concentrations of taurine [4]. Since taurine depletion is potentially harmful for these patients, who frequently have heart failure, muscular fatigue and neurological symptoms, we decided to make an open, non-randomized trial in ten chronic haemodialysis patients on the effect of daily oral taurine substitution for 10 weeks on various neurophysiological parameters, and plasma and muscle levels of taurine. The dose of taurine, 100 mg/kg/day, was similar to that previously used in human clinical trials [2]. The protocol was approved by the Ethics Committee of Karolinska Institutet at Huddinge University Hospital.

One patient completed the study with no symptoms or side effects. The second patient underwent kidney transplantation after 7 weeks of taurine, which was withdrawn immediately before transplantation. No symptoms that could be related to the intake of taurine were reported. The third patient completed the study but complained of dizziness at the end of it, which disappeared within 24 h after stopping the taurine intake. The fourth patient reported increasing dizziness and non-rotatory vertigo and withdrew from the study after 2.5 weeks. The symptoms resolved within 24 h after discontinuing taurine. This patient was rechallenged after 3 days with half the dose of taurine (50 mg/kg/day) and the same

symptoms recurred. The study was then immediately stopped before any new patient had been recruited. Results of taurine analyses of plasma and muscle before and at the end of the study were available in patients 1 and 3 but only initial values in patients 2 and 4, who withdrew from the study (Table 1).

Three of the patients were taurine-depleted with markedly reduced muscle taurine concentrations and one patient (patient 1) had a marginally low level compared to the controls. After taurine treatment, the plasma levels in patients 1 and 3 had increased by 5513% and 1780%, respectively, and the muscle intracellular concentrations by 239% and 351%, respectively, i.e. far above the normal ranges. The accumulation of taurine was presumably due to lack of renal excretion, which in normal persons accounts for the excretion of excess taurine. The removal by haemodialysis was apparently insufficiently effective to control the body content of taurine. We conclude that the symptoms reported were caused by excessive extra- and intracellular accumulation of taurine. In keeping with this conclusion is the observation that withdrawal of taurine caused a rapid disappearance of the symptoms, which reappeared when one patient was rechallenged with taurine after a symptom-free interval.

With this report we want to call attention to the risk of taurine administration to patients with renal failure and specially warn against the use of energy drinks such as Red Bull<sup>®</sup> and +Battery<sup>®</sup>, of which three cans of 33 ml/day provide 4 g of taurine (i.e. half the dose that caused excessive accumulation of taurine and neurological symptoms in our patients). Although the symptoms were relatively mild and rapidly disappeared after stopping the taurine intake, long-term risks of excessive taurine accumulation cannot be ruled out. We strongly suggest that cans or bottles of energy drinks containing taurine should have a label, which warns against their use by patients with kidney failure.

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