Another „soberade“ on the market: does OUTOX keep its promise?

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The fructose effect

Fructose (laevulose) is known to possibly increase the ethanol (alcohol) metabolism. Alcohol elimination in the human body is mainly performed by the alcohol dehydrogenase (ADH) in the liver. The first step is the oxidation of ethanol by ADH in the presence of the coenzyme NAD+, resulting in acetaldehyde, NADH and H^+. The (re)oxidation of NADH, respective the dissociation of the ADH-NADH complex, is considered to be the rate limiting step here. Different metabolic pathways were proposed to explain how fructose is involved. Most of them have in common that during fructose metabolism NAD+ is provided in different ways that accelerates the ethanol elimination. This is called the “fructose effect”.

Using fructose for merchandising purposes

The fructose effect was repeatedly used for merchandising of different products, claiming to reduce alcohol levels and to prevent hangover. However, all of them vanished from the market soon. In the forensic community the fructose effect was severely doubted *in vivo*, and also *in vitro* investigations failed to prove the fructose effect.

OUTOX

A new fructose containing soft drink was put on the market, OUTOX. This drink is promoted as “soberade”, promising accelerated alcohol reducing and hangover preventing effects.
Ingredients according to the manufacturer: carbonated water (CO_2: 6 g/l), fructose, citric acid, malic acid, ascorbic acid, “tutti frutti” flavour and carminic acid (E120).
Nutrition values per 100 ml: energy 345.1 kJ (81.2 kcal); carbohydrates: 20.3 g; sodium: 3 g; fat and protein: 0 g.

The present study

The present **study** was carried out as randomised, placebo controlled, double blind study in cross-over design. Thirty healthy volunteers (20 to 40 years) participated in two absolutely identical drinking sessions concerning time, alcohol consumption, physical activity, food and beverage uptake. The participants drank alcoholic beverages of their own choice during 2 hours. Within 15 min after drinking end, either 250 ml of OUTOX or 250 ml of a placebo drink, a sugarless carbonated soft drink, was consumed. Starting 30 min after the end of alcohol consumption, every 30 min BrAC was measured and a blood sample was drawn.
Measurement of blood alcohol concentration (**BAC**) was conducted according to Austrian forensic standards with headspace gas chromatography and flame ionisation detection (FID), using t-butanol as internal standard. The average value of 4 measurement results was calculated. Per mille is defined as g/l in Austria.
Measurement of breath alcohol concentration (**BrAC**) was performed using a calibrated Alcotest 7110 MK III A (Dräger Austria, Vienna). For a valid result two independent breath samples are necessary. BrAC values are given as mg/l.

Results

The mean peak BAC level was already achieved at the first measurement point in time, i.e. in average 36 min after drinking end. It was 1.010 ± 0.286 g/l, varying between 0.486 g/l and 1.675 g/l, in the placebo experiment, and 0.976 ± 0.286 g/l, varying between 0.579 g/l and 1.543 g/l, in the OUTOX experiment. The difference between the BAC and BrAC values in both experiments were statistically significant ($p < 0.0001$). Mean BAC and BrAC values for a mean individual regarding age and body weight to a mean point in time were then calculated (**Table 1**).

BAC	age (years)	weight (kg)	time (min)		BAC [g/l] (95% CI)	difference (95% CI)	rel. diff. (%)
total	29.2	68.9	163.7	Placebo	0.748 (0.719–0.776)	0.077 (0.037–0.117)	10.28
				OUTOX	0.671 (0.642–0.700)		
male	30.3	76.4	172.9	Placebo	0.814 (0.789–0.839)	0.098 (0.063–0.132)	12.04
				OUTOX	0.716 (0.692–0.741)		
female	27.7	58.4	151	Placebo	0.706 (0.672–0.740)	0.05 (0.016–0.084)	7.08
				OUTOX	0.656 (0.653–0.660)		
BrAC	age (years)	weight (kg)	time (min)		BrAC [mg/l] (95% CI)	difference (95% CI)	rel. diff. (%)
total	29.2	68.9	168.1	Placebo	0.314 (0.301–0.327)	0.045 (0.027–0.063)	14.3
				OUTOX	0.269 (0.256–0.282)		
male	30.3	76.4	176.6	Placebo	0.332 (0.322–0.342)	0.048 (0.033–0.062)	14.5
				OUTOX	0.284 (0.273–0.295)		
female	27.7	58.4	156.1	Placebo	0.300 (0.282–0.319)	0.030 (0.011–0.049)	10.0
				OUTOX	0.270 (0.262–0.279)		

Table 1

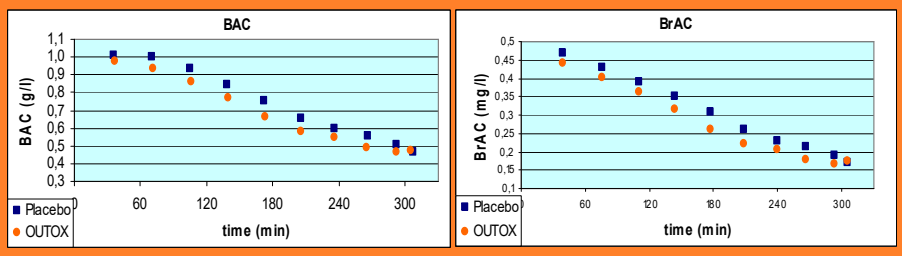

Figure 1

The mean values of BAC and BrAC at any measurement point in time are depicted as curves in **Figure 1**, showing a nearly linear alcohol elimination rate. The slopes of the linear regression showed no statistically significant difference ($p = 0.15$ for BAC and $p = 0.06$ for BrAC). This means that OUTOX did not increase the elimination rate of alcohol.

Conclusion

The mean relative difference in BAC values of the OUTOX and the placebo experiment was 10.28%, for BrAC values 14.3%. So in total an effect could be proved, meaning that OUTOX has a statistically significant effect on peak alcohol concentrations if consumed in close connection with alcoholic beverages. The alcohol elimination rate did not change statistically significantly, so maybe an increased “absorption deficiency” did also play a role.
However, the mean absolute decrease of 0.077 g/l for BAC and 0.045 mg/l for BrAC is much too low to reduce the alcohol induced impairment of persons remarkably, especially concerning road traffic. In addition, considerable variances, up to an inverse effect, were observed within the volunteers. Consequently OUTOX cannot be called a “soberade” in scientific respects.