

Competing interests None declared.

REFERENCE

1. **Solaymani-Dodaran M**, Card TR, Aithal GP, *et al*. Fracture risk in people with primary biliary cirrhosis: a population-based cohort study. *Gastroenterology* 2006;**131**:1752.

PWE-278 DOES N-ACETYL CYSTEINE MODULATE RENAL FUNCTION AFTER MAJOR HEPATECTOMY?

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Introduction Acute kidney injury or deterioration in pre-existing renal dysfunction following major hepatectomy is an important cause of morbidity and mortality. N-acetyl cysteine has been extensively utilised as a reno-protective agent to ameliorate contrast induced nephropathy. The aim of this study was to investigate its potential use for preserving renal function following major hepatectomy.

Methods A prospective study to compare the impact of N-Acetyl cysteine (NAC) on liver function after hepatectomy was undertaken in our Unit from 2004 to 2010. A cohort of 44 patients received peri-operative NAC (10 g/24 h) for 5 days and were compared to a further cohort of 44 patients (matched for the extent of liver resection—all more than three segments), gender, age and chemotherapy use) who did not. Post-operative renal function was evaluated. Other variables known to influence renal function included associated co-morbidity, drugs, intra-operative parameters (eg, CVP and blood loss). In addition to calculating the renal risk index as described by Clavien *et al*. We also examined other outcome measures such as duration of renal support and long-term renal specific outcomes.

Results There were three patients with known CKD in the NAC group as compared to one in the control group. Mean (SD) pre-operative creatinine levels [93.9 (20.5), 92.5 (19.3) p=0.7], BMI [26.9 (14.5), 28.9 (13.1) p=0.3], operating time (h) [6 (1.9), 5.5 (1.7), p=0.9]. The mean renal risk index were also similar between the NAC and control group. Mean (SD) creatinine rise (delta creatinine) between days 1 and 5 in the post-operative period, were also similar between the 2 groups [NAC 43.8 (82.9) vs Control 49.2 (63.5), p=0.72]. Renal support in the form of CVVH was needed in three patients in the study group vs one in the control group (all known to have pre-existing CKD). All these patients went back to their pre-operative renal status at 3-months follow-up.

Conclusion In this study NAC does not appear to be reno-protective in patients with known renal impairment who require a major liver resection. Its presumed renoprotective role in those at “high risk” needs to be further assessed in an adequately powered RCT.

Competing interests None declared.

PWE-279 THE HEPATIC EFFECTS OF A HIGH FRUCTOSE VS A HIGH GLUCOSE DIET IN HEALTHY OVERWEIGHT MEN

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Introduction A high intake of fructose has been implicated in NAFLD aetiology. As the majority of dietary fructose originates from sucrose it remains uncertain if these observations are fructose-specific.

Infrequent and inconsistent differences in hepatic metabolism have been shown with a high intake of fructose or glucose, the constituents of sucrose.^{1,2} There is no prior comparative data in healthy overweight men, and none in the isoenergetic state.

Methods 32 healthy, centrally overweight males were randomised to two periods each of 2 weeks of either a high fructose or glucose intake in a non-crossover fashion. Isoenergetic status was maintained by providing foodstuffs during the first period, followed by a 6-week washout and then a second period of *ad libitum* overfeeding. The sugars contributed 25% of predicted total energy requirements, and were consumed 4 times a day dissolved in water. The primary outcome was hepatic triglyceride content (¹H MRS), with further assessments of calf lipid (¹H MRS), deuterated glucose hyperinsulinaemic euglycaemic clamps, and indirect calorimetry. Outcomes assessed by Student t test.

Results The groups were well matched at study entry. Overall the subjects' mean age was 34 years, BMI 29.4 kg/m² and daily dose of sugars was 217 g. The changes in the primary measures are shown in the Abstract PWE-279 table 1 below. There were no changes during the energy balanced period. With energy and monosaccharide overfeeding weight, serum triglycerides, liver lipid and biochemistry increased significantly, but to a similar extent in both groups. Further to this the groups did not differ in terms of satiety, whole body oxidative profile, hepatic insulin resistance, calf lipid, or renal function.

Abstract PWE-279 Table 1

	Energy balance	Fructose, mean	SD	Glucose, mean	SD	Sig between, groups
Weight (kg)	Iso	-0.26	0.92	0.12	0.70	0.62
	Excess	1.03*	1.37	0.57*	1.00	0.29
Hepatic lipid (%)	Iso	0.30	2.20	-0.05	2.13	0.65
	Excess	1.70*	2.60	2.05*	2.89	0.73
ALT (U/l)	Iso	-4.0	7.9	-2.9	6.5	0.67
	Excess	5.8*	8.7	4.1	9.8	0.62
Triglyceride (mmol/l)	Iso	-0.07	0.35	0.13	0.74	0.35
	Excess	0.36	0.75	0.33**	0.39	0.91

Absolute change in parameters during intervention (*p<0.05, **p<0.01 as compared to baseline values in that group).

Conclusion There were no differences between a high fructose and glucose diet in relation to hepatic lipids or biochemistry. The changes during the overfeeding period were strongly associated with changes in weight, reinforcing the interpretation that these were an energy, as opposed to a nutrient, specific effect.

Clinical trial registration number NCT01050140.

Competing interests None declared.

REFERENCES

1. **Stanhope KL**, Schwarz JM, Keim NL, *et al*. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. *J Clin Invest* 2009;**119**:1322–34.
2. **Ngo Sock ET**, Lê KA, Ith M, *et al*. Effects of a short-term overfeeding with fructose or glucose in healthy young males. *Br J Nutr* 2010;**103**:939–43.

PWE-280 FERROPORTIN DISEASE—AN IMPORTANT CAUSE OF IRON OVERLOAD

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Introduction Ferroportin disease is a recently characterised cause of iron overload. Unlike hereditary haemochromatosis it involves