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Abstracts of Original Communications

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Effect of vitamin and mineral supplementation on cognitive function in men and women aged 65 years and over. By A.C. MILNE[1], A. AVENELL[1] and G. McNEILL[2] for the MAVIS TRIAL GROUP, [1]*Health Services Research Unit and* [2]*Department of Environmental and Occupational Medicine, University of Aberdeen, Foresterhill, Aberdeen, UK, AB25 2ZD*

Cross-sectional studies of older individuals have reported positive associations between vitamin or mineral intake or blood levels and cognitive performance, but supplementation studies with individual vitamins and minerals have, however, mostly failed to show a beneficial effect.

In a randomised double-blind placebo-controlled trial of the effect of a commonly used over the counter multivitamin and mineral preparation on infection in older individuals (MAVIS trial group, unpublished results) we also collected data on cognitive function. A total of 910 men and women aged 65 years and over who had not taken vitamin or mineral supplements in the preceding 3 months (or 1 month for water-soluble vitamins) were recruited between February and December 2002. Four hundred and fifty-six participants (239 men and 217 women) were randomly allocated to the active group and received a daily multivitamin and mineral supplement for 12 months, and 454 participants (240 men and 214 women) received a daily placebo tablet for the same period. The median age was 72 (interquartile range 68, 76) years in the supplemented group and 71 (interquartile range 68, 76) years in the placebo group. The active treatment was a daily tablet containing 800 µg vitamin A, 60 mg vitamin C, 5 µg vitamin D, 10 mg vitamin E, 1.4 mg thiamine, 1.6 mg riboflavin, 18 mg niacin, 6 mg pantothenic acid, 2 mg pyridoxine, 1 µg vitamin B_{12}, 200 µg folic acid, 14 mg Fe, 150 µg iodine, 0.75 mg Cu, 15 mg Zn and 1 mg Mn. Cognitive function was assessed in all participants by digit span forward and verbal fluency tests carried out face-to-face at recruitment and over the telephone at the end of the 12-month intervention period. The analysis was carried out on an intention-to-treat basis in all participants and in a sub-group of those 75 years or over, with adjustment for baseline test values and trial minimisation factors (age, sex and place of residence). Statistical significance was sought at $P<0.05$ for the whole group and $P<0.01$ for the older sub-group.

The Table shows the baseline and 12-month scores for the two tests in all participants and the older sub-group. There was strong evidence of an improvement in the digit span forward test (change +0.4; 95% CI 0.2, 0.6) but weaker evidence for an improvement in the verbal fluency test (change +0.7; 95% CI −0.2, 1.7). There was no evidence for a difference in the change in the digit span forward score between active and placebo groups in all participants ($P=0.537$) or in the older sub-group ($P=0.606$). There was also no evidence for a difference between active and placebo groups in the verbal fluency scores in all participants ($P=0.163$), but in those 75 years or over there was weak evidence for a beneficial effect of supplementation ($P=0.037$).

		Digit span forward scores						Verbal fluency scores					
		Baseline		12 months		Difference		Baseline		12 months		Difference	
Participants		Mean	SD	Mean	SD	Mean	CI*	Mean	SD	Mean	SD	Mean	CI*
All participants	Active	11.0	2.2	11.5	2.3	−0.1	−0.3, 0.2	32.1	12.0	33.8	12.8	0.8	−0.3, 2.0
	Placebo	11.2	2.2	11.7	2.1			31.8	12.8	33.0	13.3		
75 years or over	Active	10.8	2.2	11.1	2.3	−0.1	−0.8, 0.6	30.0	12.7	32.4	13.0	2.8	−0.6, 6.2
	Placebo	11.0	2.1	11.3	2.2			29.6	12.5	30.0	13.4		

*95% CI for all participants and 99% CI for those 75 years or over.

These results provide no evidence for a beneficial effect of multivitamin supplementation on cognitive function in community-living men and women 65 years or over. We cannot exclude the possibility of effects on other domains of cognitive function, or of small benefits of supplementation in older subjects.

The present study was funded by The Health Foundation (formerly PPP Healthcare Medical Trust).

Childhood and adult dietary vitamins B_1 and B_{12} intake and psychiatric symptom frequency at age 43 among women in the 1946 British Birth Cohort. By G.D. MISHRA[1], M.A. O'CONNELL[2], C.J. PRYNNE[2], S.A. McNAUGHTON[2] and D. KUH[1], [1]*MRC National Survey of Health and Development, University College and Royal Free Medical School, 1-19 Torrington Place, London, UK, WC1E 6BT and* [2]*MRC Human Nutrition Research, Elsie Widdowson Laboratory, Fulbourn Road, Cambridge, UK, CB1 9NL*

B vitamins may be beneficial in a number of neuropsychiatric disorders including depression, due to their role as coenzymes in the synthesis of neurotransmitters and in one-carbon metabolism (Malouf *et al.* 2003). There are few longitudinal studies that have investigated the cumulative effects of nutrition over the life-course or the importance of childhood diet on neuropsychiatric disorders.

The present study examined the effect of dietary B vitamin intake (B_1, B_2, nicotinic acid, B_6, folic acid, B_{12}) during childhood (age 4) and adulthood (ages 36 and 43) on psychiatric symptom frequency (PSF) score at age 43 years in the MRC National Survey of Health and Development (1946 Birth Cohort). This cohort is a social-class-stratified random sample of 5362 male and female singleton births in Britain during the first week of March 1946. Trained research nurses collected the PSF information (Lindelow *et al.* 1997), an eighteen-item scale measuring symptoms of anxiety and depression in the year before the interview. Responses to each item were summed to provide a total score ranging from 0 (no disturbances) to 90 (severe disturbances).

This analysis is based on female participants who provided dietary and socio-economic information at the three time-points and PSF score at 43 years (n 1047). A 24 h recall was obtained at interview from the child's carer, usually the mother, at age 4 years. A 48 h recall of all food and drink recorded in household measures was obtained at interview at ages 36 and 43 years. Intakes were expressed as proportion of total energy (per MJ). Long-term intake during adulthood was calculated as the average intake over the two adult time-points. Combinations of groups defined by the tertiles of energy-adjusted vitamins were used to create six categories to describe change in intake from childhood to adulthood. In childhood, the distributions of intakes showed lower variation, therefore the lowest and middle thirds of intake were combined. Linear regression was used to model the relationships between nutrient intake groups and PSF score. PSF score (at age 43 years) by lifetime dietary vitamin B_1, and vitamin B_{12} intake is presented.

		Vitamin B_1			Vitamin B_{12}†		
Childhood (at age 4)	Adulthood (average intake at ages 36 and 43)	Adjusted regression coefficient*	95% CI	P value	Adjusted regression coefficient*	95% CI	P value
Low or middle	Low	0.21	−0.04, 0.52	0.08	−0.28	−0.42, −0.1	0.08
High	Low	0.09	−0.16, 0.4		−0.2	−0.38, 0.04	
Low or middle	Middle	0.36	0.08, 0.71		−0.25	−0.4, −0.07	
High	Middle	0.35	0.05, 0.73		−0.18	−0.37, 0.06	
Low or middle	High	0.27	0.02, 0.59		−0.25	−0.4, −0.06	
High	High	Reference			Reference		

*Confounders for vitamin B_1 were smoking status, physical activity, BMI, mean alcohol consumption at ages 36 and 43; confounders for vitamin B_{12} were social class, education, BMI, mean alcohol consumption at ages 36 and 43.
†n 1009 due to missing values.

Vitamin B_1 intake was marginally associated with the PSF score at 43 years, with women in the high childhood–high adulthood intake category being at least risk. In marked contrast, those in the high childhood–high adulthood category for vitamin B_{12} intake had a higher PSF score. In a subsequent analysis, childhood vitamin B_{12} intake, rather than in adulthood, was associated with psychiatric symptoms frequency, with those in the low and middle thirds of intake having lower scores that those in the highest third. The food source for vitamin B_{12} intake was animal products, including meat products, red meat, fish, eggs and dairy products. Thus, the contrary direction of the positive association between childhood vitamin B_{12} intake and PSF at 43 years may reflect other social or lifestyle factors not fully taken into account. There remains the possibility of the results being due to type 1 errors, and the associations being found arising purely by chance. For the remaining B vitamins, there were no significant relationships with psychiatric symptom frequency score.

The present study was funded by the Food Standards Agency (project no. N05048).

Lindelow M, Hardy R & Rodgers B (1997) *Journal of Epidemiology and Community Health* **51**, 549–557.
Malouf M, Grimley EJ & Areosa SA (2003) Cochrane Database of Systematic Reviews CD004514.

Intakes of antioxidants in childhood and the risk of psychiatric disorder in females between ages 15 to 32 in the 1946 British Birth Cohort. By M.A. O'CONNELL[1], G.D. MISHRA[2], S.A. McNAUGHTON[1], C.J. PRYNNE[1] and D. KUH[2], [1]*MRC Human Nutrition Research, Elsie Widdowson Laboratory, Fulbourn Road, Cambridge, UK, CB1 9NL and* [2]*MRC National Survey of Health and Development, University College and Royal Free Medical School, 1–19 Torrington Place, London, UK, WC1E 6BT*

As antioxidants are thought to afford protection against neurodegenerative diseases by decreasing oxidative damage, they may also play a beneficial role in psychiatric disorder. Significant positive correlations have been found between depressive symptoms and markers of oxidative stress such as serum lipid peroxides (Tsuboi *et al.* 2004). Measures of psychological stress have been shown to be associated with increased oxidative stress and antioxidant intake and status have also been associated with cognitive function and decline (Rogers, 2001; Irie *et al.* 2003). A number of important dietary components exhibit antioxidant activity including vitamin C, vitamin E and carotenoids. However, there is limited research investigating their role in the development of psychiatric disorder.

The present study examined the effect of high and low dietary intake of antioxidants (vitamin C, vitamin E and total carotene equivalents) during childhood (age 4) on the risk of psychiatric disorder in females between the ages of 15–32 years in the MRC National Survey of Health and Development (1946 Birth Cohort), a social class-stratified, random sample of 5362 singleton births in Britain during the first week of March 1946.

A 24 h dietary recall was obtained at interview from the child's carer, usually the mother, at age 4 years. Analysis is based on women survey members for whom information was available on diet at age 4 years, socio-economic circumstances, and psychiatric disorder between ages 15–32 years (*n* 2084). The measure of psychiatric health used distinguished those who had been admitted to psychiatric hospital (*n* 211; 9.6%) from those reporting minor nervous illness (*n* 698; 31.6%) or no such illnesses (*n* 1298; 58.8%) between ages 15–32 years. Odds ratios (OR) and 95% CI for the risk of psychiatric disorder between ages 15 and 32 years by thirds of dietary antioxidants intake at age 4 years are presented.

Childhood dietary intake (thirds)	*n*	%	Cases*	OR	95% CI	*P* value†	Adjusted OR‡	95% CI	*P* value†
Carotene (μg/MJ)						0.05			0.03
Low (5–70)	695	45.3	315	1.17	0.96, 1.40		1.17	0.92, 1.47	
Middle (71–158)	698	38.4	268	0.91	0.73, 1.12		0.83	0.69, 1.09	
High (159–1466)	691	40.7	281	Reference			Reference		
Vitamin E (mg/MJ)						0.02			0.07
Low (0.08–0.36)	698	41.8	698	1.17	0.95, 1.44		1.14	0.91, 1.38	
Middle (0.36–0.47)	692	44.8	310	1.34	1.08, 1.66		1.26	1.04, 1.63	
High (0.47–2.22)	695	37.8	695	Reference			Reference		
Vitamin C (mg/MJ)						0.5			0.5
Low (0.3–4.5)	690	42.8	295	1.1	0.91, 1.38		1.14	0.91, 1.43	
Middle (4.5–7.4)	692	41.2	285	1.06	0.86, 1.31		1.10	0.88, 1.37	
High (7.4–49.7)	702	40.5	284	Reference			Reference		

* Cases of minor nervous illness or admitted to psychiatric hospital.
† Test for trend.
‡ Adjusted for social class at age 26 years and education level.

Low carotene and vitamin E intakes in childhood were associated with psychiatric disorder in young adult life. After adjustment for confounders (social class at age 26, and education level), only carotene intake remained significant, with the severity of psychological health increasing for women with lower carotene intake. Until there is further evidence for a relationship between carotene and psychiatric disorder, it is important to consider that the protective action (in the middle and highest thirds) attributed to carotene may be due to some other dietary component that is closely associated with carotene, for example non-provitamin A carotenoids, or other phytochemicals found in fruit and vegetables (Steinmetz & Potter, 1991), or some other social or lifestyle factors not fully taken into account. Further research is required to confirm these results in other populations and among men.

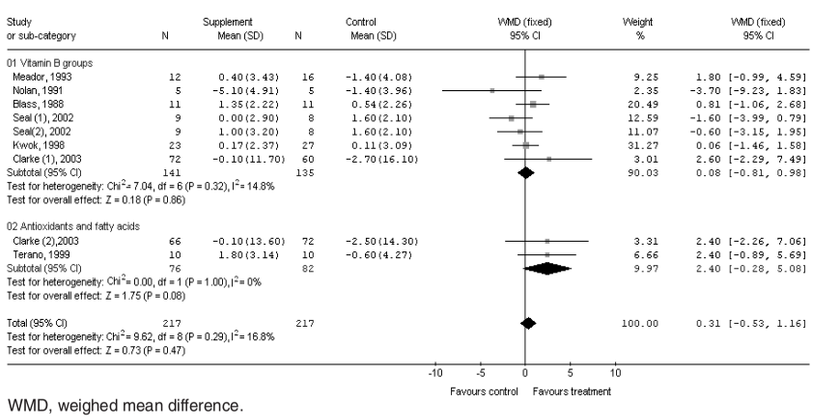
The present study was funded by the Food Standards Agency (project no. N05048).

Irie M, Asami S, Ikeda M & Kasai H (2003) *Biochemical and Biophysical Research Communications* **311**, 1014–1018.
Khanzode SD, Dakhale GN, Khanzode SS, Saoji A & Palasodkar R (2003) *Redox Reports* **8**, 365–370.
Rogers PJ (2001) *Proceedings of the Nutrition Society* **60**, 135–143.
Steinmetz KA & Potter JD (1991) *Cancer Causes and Control* **2**, 427–442.
Tsuboi H, Shimoi K, Kinae N, Oguni I, Hori R & Kobayashi F (2004) *Journal of Psychosomatic Research* **56**, 53–58.

The effect of micronutrients and fatty acids on age-associated cognitive decline in the elderly: a systematic review. By X. JIA[1], G. McNEILL[1] and A. AVENELL[2], [1]*Department of Environmental and Occupational Medicine, University of Aberdeen, Foresterhill Road, Aberdeen, UK, AB25 2ZP and* [2]*Health Services Research Unit, University of Aberdeen, Foresterhill, Aberdeen, UK, AB25 2ZD*

Observational studies frequently show a positive association between cognitive performance and micronutrient intake or status in the elderly, but it is difficult to exclude the effects of confounding variables such as health-seeking behaviour. The aim of the present systematic review was to collate the available randomised controlled trials on the effectiveness of micronutrients and fatty acids in the prevention of age-associated cognitive disorder, cognitive impairment and degenerative dementia. MEDLINE, EMBASE, the Cochrane Central Register of Controlled Trials and three websites of registered trials in the UK, Europe and the USA respectively up to March 2005 were searched. Studies were excluded if the supplements contained any macronutrients, or if the cognitive changes were due to head injury, vascular accidents or other non-age-associated causes.

A total of 4229 articles were found on the initial searches and fifty were reviewed in detail. After the critical assessment of quality, twenty completed studies were found to be eligible for the review, while four did not have complete results, and twenty-six were excluded. The included studies involved twenty-three interventions and 2251 participants. In four studies the participants had low folate levels. In eight studies the participants were mentally healthy; in the others the subjects had cognitive decline with ranged from mild cognitive decline through to severe dementia. The nutrient content, dose and duration of supplement use varied widely between studies, as did the measure of cognitive performance. Of the twenty studies, five reported a significant difference between intervention and control groups in one or more tests. In nine studies the Mini-Mental State Examination (MMSE) was used, so the results of these studies were pooled in a meta-analysis, which is shown below. For B vitamins, antioxidant, fatty acid and for all studies the results favoured the treatment group but were not statistically significant. One study of multivitamins and minerals, which has been the subject of debate, found a clear beneficial effect of supplementation in the MMSE and other tests. This study is not included in the meta-analysis.



WMD, weighed mean difference.

Overall there is weak beneficial effect of micronutrient and fatty acid supplements on cognitive decline in the elderly. Trials with larger sample size, longer duration, and using same outcome measurements are expected for the future review.

Socio-economic factors affecting the quantity and quality of dietary iron among rural women. By A. DJAZAYERY[1], S.A. KESHAVARZ[1], F. ANSARI[2] and M. MAHMOUDI[2], [1]*School of Public Health and Institute of Public Health Research, Tehran University of Medical Sciences, Tehran, Iran and* [2]*Institue of Standard and Industrial Research of Iran, Karaj, Iran*

A prerequisite for planning and selecting appropriate strategies to promote iron (Fe) status and alleviate anaemia, a common public health and nutritional problem in many communities, is a knowledge of its exact causes and contributing factors. The objective of the present study was to determine the Fe status of married women and the factors influencing their dietary Fe intake in Lorestan, a province in Iran with a medium economic status.

The Fe intake and status were investigated in 998 married women, 20–56 years old, selected by two-stage random sampling, in Lorestan rural areas. Interviews were conducted by nutrition MSc students to obtain information on socio-economic status, and a validated, pre-tested 24 h dietary recall questionnaire was used to obtain data on nutrient intakes. Fasting blood samples were collected to assess Festatus. Fe deficiency anaemia was defined as a value below 12.6 g/dl, 410.0 µg/dl, 50.0 µg/dl, 37.8%, 33.3%, or 15.0% for haemoglobin, total iron binding capacity, serum Fe, haematocrit, mean corpuscular haemoglobin concentration, and transferrin saturation, respectively, and low Fe-status was defined as a value of ferritin below 10 µg/L or of transferrin saturation less than 15%.

Depending on the parameter used, 7.1–27.5% of the women were assessed as having a Fe deficiency anaemia. Low Fe-status in 27.0%, the corresponding national proportion being 30% (Djazayery & Marchesich, 2001). The average daily Fe intake was about 90% recommended dietary allowance. Some key socio-economic variables were significantly associated with Fe intake in univariate analysis.

	Education level					Occupation					Family size				
Fe intake	≤6 years		>6 years			Employed		Housewife			<6		≥6		
(mg/d)	Mean	SD	Mean	SD	*P*<	Mean	SD	Mean	SD	*P*<	Mean	SD	Mean	SD	*P*<
Total	13.9	5.9	16.5	8.5	0.001	14.9	5.9	14.9	8.7	NS	19.1	9.8	14.0	8.8	0.0001
Animal	1.0	0.9	1.5	0.1	0.001	2.2	1.5	1.2	1.0	0.01	1.7	1.5	1.1	1.1	0.0002
Plant	13.0	8.9	15.5	8.9	0.001	13.0	5.5	14.1	9.0	NS	17.0	9.9	13.1	8.0	0.0001

As shown in the Table, the total and plant Fe intakes were influenced positively by a higher education level and a smaller family size. Employment of the women (indicative of a higher family income) did not affect the total or plant Fe intake; it did, however, increase the proportion supplied by animal foods–although the proportion (2.2%) was still much below 8%, the corresponding national figure (National Nutrition and Food Technology Research Institute, 2003). Further analysis is required to elucidate which of the socio-economic variables are the most important. It is concluded that employment opportunities, education and small family size are important determinates of Fe intake and status among women in rural community settings. These should, therefore, receive particular attention in national health and nutrition planning.

Djazayery A & Marchesich R (2001) Nutrition Country Profiles – Iran. www.fao.org
National Nutrition and Food Technology Research Institute (2003) *Report on the National Food Consumption Patterns and Nutrition Situation 2000–2002*. Tehran: National Nutrition and Food Technology Research Institute.

Zinc status and immune function in a healthy older population aged 55–70 years: the Zenith study. By C.F. HODKINSON[1], V.C. JEWELL[1], M. KELLY[1], C. COUDRAY[2], W.S. GILMORE[1], J.M. O'CONNOR[1], J.J. STRAIN[1] and J.M.W. WALLACE[1], [1]*Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, UK, BT52 1SA,* [2]*Centre de Recherche en Nutrition Humaine d'Auvergne, Unité Maladies Métaboliques et Micro-nutriments INRA, Centre de Recherche de Clermont-Ferrand/Theix 63122 Saint Genès Champanelle, France*

Zn plays a vital role in the maintenance and function of the immune system. Approximately 50% of adults aged 50–70 years may have sub-optimal Zn status (Briefel *et al.* 2000). The present study aims to evaluate Zn status, and examine the effect of Zn status on immune function in ninety-three apparently healthy late middle-aged men and women (aged 55–70 years). Multiple biochemical measures were used to assess Zn status, while flow cytometry was used to determine immune function. Serum and erythrocyte Zn concentrations were 13.0 (SD 1.4) µM/l and 222 (SD 48.2) µM/l, respectively. Plasma alkaline phosphatase concentration was 76.8 (SD 16.1) U/l. Significant associations between serum and erythrocyte Zn concentration and, a number of immune function parameters were observed (see Table).

			Serum Zn (µM/l)		Erythrocyte Zn (µM/l)	
Parameter	Median	Interval*	*r*	*P* value†	*r*	*P* value†
CRP (mg/l)	0.10	0.00–1.36	−0.267	0.005‖	0.002	0.494
CD3+ T-cells ($\times 10^9$/l)	1.12	0.43–2.03	0.168	0.055	0.234	0.013‡
CD3+/CD16+/CD56+ NKT cells ($\times 10^9$/l)	0.06	0.01–0.65	0.113	0.139	0.244	0.010‖
CD3+/CD25+ T-cells ($\times 10^9$/l)	0.29	0.11–0.66	0.180	0.040‡	0.218	0.020‡
IL-2 receptor density (MFI)	68.8	38.1–110	0.198	0.030‡	0.049	0.323

MFI, mean fluorescence intensity.
* Intervals given as 2.5 and 97.5 percentiles.
† Data were normalised as appropriate and analysed by Pearson correlation.
Significant levels: ‡$P \le 0.05$; ‖$P \le 0.01$.

In addition, sex-associations were also apparent. Serum Zn was negatively correlated with C-reactive protein (CRP) in women only ($P=0.008$), while serum Zn was positively correlated with CD3+ T-cells and CD3+/CD16+/CD56+ NKT cells ($P=0.038$ and $P=0.040$, respectively) in men only. Erythrocyte Zn was positively correlated with CD3+ T-cells and CD3+/CD16+/CD56+ NKT cells in men only ($P=0.011$ and $P=0.030$, respectively) and positively correlated with early-activated CD3+/CD25+ T-cells in women only ($P=0.035$). These findings indicate that increased concentrations of serum and erythrocyte Zn may promote enhanced cell-mediated immunity, and may also lower the production of inflammatory proteins such as CRP. However, effects of Zn status on immune function appear to be sex-specific. Zn parameters for all subjects were within the normal adult ranges. Further investigation is required to determine if enhanced Zn status would improve cell-mediated immunity in these individuals, and whether it would be beneficial in the protection against age-related inflammatory disorders common to this age group.

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Briefel RR, Bialostosky K, Kennedy-Stephenson J, McDowell MA, Erwin RBR & Wright JD (2000) *Journal of Nutrition* **130**, 1367S–1373S.

High- *v.* low-sucrose diet: evaluation of effects on insulin sensitivity. By M. SPENCE[1], R.N.A. BLACK[2], G.J. CUSKELLY[1], C.N. ENNIS[2], D.R. McCANCE[2], I.S. YOUNG[1], P.M. BELL[2] and S.J. HUNTER[2], [1]*Nutrition and Metabolism Group, Queen's University Belfast, UK, BT12 6BJ and* [2]*Regional Centre for Endocrinology and Diabetes, Royal Victoria Hospital, Grosvenor Road, Belfast, UK, BT12 6BA*

The long-term impact of dietary carbohydrate, in particular sucrose, on insulin action and the development of diabetes and atherosclerosis is not established. Current guidelines for those with type 2 diabetes advise restriction of sucrose intake to 10% of total energy intake (Connor *et al.* 2003).

Thirteen healthy male volunteers (mean age 33 (SE 3.0) years and BMI 26.6 (SE 0.9) kg/m^2) with normal glucose tolerance were assigned to a randomised cross-over trial, consisting of a 6-week period of either low or high sucrose intake followed by a second 6-week period on the complementary diet. The respective dietary interventions provided 10% (low) *v.* 25% (high) of their total energy as sucrose and were separated by a 4-week wash-out phase.

Diets were designed using nutritional analysis software, WISP© (Tinuviel Software, Warrington, UK). During interventions, individual volunteer menus were altered, where appropriate, to ensure that body weight was maintained constant throughout. This was achieved whilst retaining fixed macronutrient profiles (55% energy from carbohydrate; 30–35% energy from fat; 10–15% energy from protein) and dietary fibre intake (18 g/d). Strategies were adopted to optimise compliance including regular (three to five per week) meetings with volunteers and all appropriately weighed foodstuffs were supplied. Insulin resistance was assessed using a two-step euglycaemic clamp performed at the end of each arm of the cross-over trial.

There was no change in weight or physical activity during the study period. At an insulin infusion rate of 2 mU/kg per min, the glucose infusion rate required to maintain euglycaemia, an index of insulin sensitivity, was 47.5 (SE 3.5) μmol/kg per min compared with 42.5 (SE 3.0) μmol/kg per min in response to 25% and 10% sucrose regimens, respectively (95% CI for the increase in insulin sensitivity was −3.5 to 14.1; P=0.2).

	25% sucrose diet	10% sucrose diet
Glucose infusion rate (μmol/kg per min)	47.5 (3.5)	42.5 (3.0)

Values in the table are mean (SE).

In conclusion, as part of an isoenergetic diet, low and high sucrose intakes do not differ significantly in their effects on insulin sensitivity in healthy non-diabetic subjects.

Connor H, Annan F, Bunn E, Frost G, McGough N, Sarwar T & Thomas B (2003) *Diabetic Medicine* **20**, 786–807.

A randomised controlled trial of a pragmatic nutrition education intervention in primary care. By S.M. MADIGAN[1], M. STEVENSON[2], M.E. WRIGHT[1], P. FLEMING[1], F. DOBBS[1] and D. McAULEY[3], [1]*Institute for Postgraduate Medicine and Primary Care, University of Ulster, Jordanstown, UK,* [2]*Clinical Research Support Centre, Royal Group of Hospital, Belfast, UK and* [3]*Department of Epidemiology, The Queens University Belfast, UK*

Enteral tube feeding in the community is a treatment commonly used for adult patients with a wide range of chronic diseases where dysphasia is present. Its management is complicated by patients with varying types of disease, types of equipment, there being no uniform support to patients across the UK (L'Estrange, 1997; Mensforth, 1999) and by the fact that patient numbers have grown significantly in the last decade. There are now approximately 19 500 adult patients in the UK (Glencourse *et al.* 2003) and it is reported that 200 000 tubes are placed annually in the USA (Roche, 2003). There is little information available on the effect of educational interventions with primary care health professionals on the topic of enteral tube feeding and in general health professionals in primary care perceive that their access to training is poor. The objective of the present study was to compare the efficacy of a community-based detailing intervention on the pre- and post-intervention scores of a sample of primary care health professionals using a cluster randomised pre–post test trial with waiting list control. The control group were offered the intervention after they had completed the two questionnaires. An educational intervention was delivered to twenty-two clusters including physicians and nurses over a period of 13 months. It was delivered in the work place, was of short duration and involved either teaching face-to-face or in small groups. The main outcome measure was the change in score along with the change in score of the intervention group at 6 months. Subjects in the treatment clusters given the educational intervention had significantly better score changes than those in the control group (P<0.001); however, the improvement was not maintained over time.

The Table shows the change in questionnaire scores by professional group at intervention.

Change in score*	*n*	Mean	SD	SEM	*t*	df	*P*
GPs							
Treatment	20	4.25	1.97	0.44	9.82	62	<0.001
Control	44	0.52	1.07	0.16			
District nurses							
Treatment	65	4.2	1.86	0.23	11.1	20	<0.001
Control	48	0.38	0.89	0.26			
Private nursing home nurses							
Treatment	36	3.19	1.69	0.28	6.20	54	<0.001
Control	20	0.45	1.39	0.31			

* Differences between pre- and post-test were measured using an independent *t* test.

It appears that a short, work-based targeted educational intervention resulted in improvements in knowledge scores but ongoing training will be required to maintain this effect.

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The effects of commercial weight-loss diets on body weight and body composition during weight-loss and follow-up phases. By C.M. LOGAN, J.M.W. WALLACE, P.J. ROBSON, M.P. BONHAM, K.L. RENNIE and M.B.E. LIVINGSTONE, *Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, UK, BT52 1SA*

As the prevalence of obesity continues to rise, so too are strategies aimed at reducing body weight. In particular, there has been an increase in the popularity of commercial weight-loss programmes. However, the efficacy of such programmes for promoting weight loss and subsequent weight maintenance remains limited (Hamilton & Greenway, 2004).

The aim of the present study was to compare the effects of four commercial weight-loss programmes on body weight and body composition during a 6-month weight-loss intervention and subsequent 1-year follow-up period. Forty-eight subjects were recruited throughout Northern Ireland and were randomised to one of four commercial weight-loss programmes; Dr Atkins' New Diet Revolution, Weight-Watchers Pure Points Programme, the Slim-Fast Plan and Rosemary Conley's 'Eat Yourself Slim' Diet and Fitness Plan. Body weight and body composition were measured at baseline, at 2 months and 6 months during the dietary intervention, and at 6 months and 12 months during the period. The effects of diet group on changes in body weight and body composition were analysed using a general linear repeated measures model follow-up adjusted for gender. Paired-sample *t* tests were used to compare measurements at baseline with 12 months follow-up. Regression analysis was used to investigate the relationship between the changes in body weight and body composition during dieting and follow-up.

Neither weight loss nor changes in body composition differed significantly between diet group during the weight-loss intervention. In the whole group, body weight ($P<0.001$), fat mass ($P<0.001$) and lean mass ($P<0.001$) decreased significantly between baseline and 2 months of the intervention. During the remainder of the intervention, body weight ($P=0.007$) and fat mass ($P<0.001$) decreased further, with no further change in lean mass. The weight-loss diet followed during the intervention had no effect on the changes in body weight or body composition during the follow-up period. In the whole group, body weight increased significantly between the end of the intervention and 6 months follow-up ($P=0.02$), with a further increase between 6 months and 12 months ($P<0.001$). Fat mass did not change significantly between the end of the intervention and 6 month follow-up but increased between 6 months and 12 months ($P<0.001$). Lean mass did not change significantly during follow-up nevertheless body weight remained lower at 12 month follow-up compared with baseline prior to the intervention ($P=0.002$). The Table presents the changes in body weight and body composition from baseline to 12 months follow-up. Additionally, changes in body weight ($P=0.028$), fat mass ($P=0.018$) during the intervention were negatively associated with corresponding changes during the follow-up period.

	Baseline		6-month intervention			12-month follow-up			Mean % change	
	Mean	SD	Mean	SD	*P**	Mean	SD	*P*†	Mean	*P*‡
Body weight	90.1	13.0	81.9	10.9	<0.001	88.0	12.9	0.018	−4.9	0.002
Fat mass	33.9	5.1	27.8	7.3	<0.001	31.2	5.4	0.005	−8.8	0.001
Lean mass	52.6	13.4	51.3	12.7	<0.001	53.7	13.7	0.215	−3.1	0.007

* Change during intervention (general linear model).
† Change during follow-up (general linear model).
‡ Baseline v. 12 month follow-up (paired-sample t tests).

In conclusion, the commercial weight-loss porgrammes assessed in the present study were equally effective in assisting short-term weight loss. Additionally body weight remained significantly lower 12 months after completing the programme. However the key to successful slimming appears to be largely dependent on selecting a diet that is suitable to each individual's lifestyle and eating habits.

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Prevalence of obesity in Irish children. By J.L. O'NEILL, S.N. McCARTHY, S.J. BURKE and M.J. GIBNEY, *Department of Clinical Medicine, Trinity College Dublin, Republic of Ireland*

Childhood obesity is a growing problem worldwide (World Health Organization, 1998) and, therefore, a major public health issue that needs to be addressed. In adults, the BMI cut-offs of ≥25 and 30 kg/m^2 are widely accepted definitions of overweight and obesity, respectively. However, in children there is no single accepted standard, thus making it difficult to estimate the prevalence of childhood obesity. The present study uses four different methods to define overweight and obesity in children. Data for this investigation are from the National Children's Food Survey (NCFS), for which 596 children (295 boys and 301 girls), aged 5–12 years, were randomly selected throughout the Republic of Ireland (Irish Universities Nutrition Alliance, 2005). Anthropometric data were collected for all children and BMI was determined by weight (kg) divided by height (m) squared.

The US Centers for Disease Control and Prevention (CDC) (Kuczmarski *et al.* 2000) and the UK 1990 (Cole *et al.* 1995) BMI-for-age growth charts were used to identify overweight and obesity. As there are no BMI-for-age charts available for an Irish reference population (Griffin *et al.* 2004), the current Irish weight and height centile charts (Hoey *et al.* 1987) were used to calculate actual relative weight (ARW). The International Obesity Task Force (IOTF) age- and sex-specific BMI cut-offs that are linked to the adult cut-offs for overweight and obesity (≥25 and 30 kg/m^2) at age 18 years (Cole *et al.* 2000) were also applied to these data.

	Boys (%) (*n* 295)				Girls (%) (*n* 301)			
	Normal	Overweight	Obese	Fold Increase in Obesity 1990–2005	Normal	Overweight	Obese	Fold Increase in Obesity 1990–2005
ARW*†	65.4	12.9	11.2		54.8	15.9	16.3	
CDC*‡	75.3	14.9	9.2	1.6	68.4	15.9	14.3	4.4
UK 1990§	80.3	10.5	9.2	1.4	75.4	11.6	13	3.0
IOTF¶	80.7	15.3	4.1	1.8	71.1	19.6	9.3	6.4

* Underweight was analysed but not presented.
† Overweight 111–120% and obesity >120%.
‡ BMI-for-age charts for boys and girls (overweight ≥85th and obesity ≥95th percentile).
§ BMI reference curves for the UK 1990 (overweight ≥91st and obesity ≥98th percentile).
¶ Age- and sex-specific BMI cut-offs for 2–18 years (Cole *et al.* 2000).

It is evident from the Table that the prevalence of overweight and obesity in Irish children is high. However, the values vary considerably with each method. The prevalence of obesity in boys ranged from 4.1 to 11.2% and in girls from 9.3 to 16.3%, depending on the method used.

The trend in childhood obesity in Ireland over the last decade was assessed by comparing the NCFS data for 8–12-year-olds to that on 8–12-year-olds from the 1990 Irish National Nutrition Survey (Lee & Cunningham, 1990), using the CDC, UK 1990 and IOTF methods to define obesity. Between the 1990 and the 2005 survey, depending on the method used, up to a 2-fold increase in obesity was seen in boys and up to a 6-fold increase in obesity was found in girls.

The need for a single definition to identify the overweight and obese child is evident given the variation displayed here in the prevalence of overweight and obesity when using the different methods. The findings show a high prevalence of overweight and obesity in Irish school children and the increase in the prevalence of obesity over the last 15 years highlights this growing public health issue. In an attempt to develop effective treatment and prevention programmes, additional work will be carried out to identify the underlying factors associated with the aetiology of obesity in Irish children.

This project was funded by the Irish Government under the National Development Plan 2000–2006.

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Where are Irish children eating? By S.J. BURKE, S.N. McCARTHY, J.L. ONEILL and M.J. GIBNEY, *Department of Clinical Medicine, Trinity College Dublin, Republic of Ireland*

The food service sector has been shown to play a major role in the Irish diet, contributing to 24% of energy consumed by Irish adults (Irish Universities Nutrition Alliance, 2001). Foods eaten outside the home are also important in the diets of children (Lin *et al.* 1999), although there has been no information available on Irish children until now.

The National Children's Food Survey which was carried out by the Irish Universities Nutrition Alliance estimated habitual food and drink consumption in a nationally representative sample of 594 Irish children aged 5–12 years using a 7 d semi-weighed record. A number of locations were coded, based on where the foods were prepared rather than consumed. Location was categorised into home, other home (friend's home, relative's home or child minder) and out (restaurant, coffee shop, takeaway, shop, pub, cinema, fast-food chain). The percentage of consumers at each location was calculated along with the breakdown for age and sex. The mean number and range of eating occasions is given for the week, whereas intakes for the percentage contribution to energy and percentage of energy from macronutrients are mean daily intakes for consumers only.

	At home	In other people's houses	Outside the home
Percentage of consumers	100.0	58.8	77.3
% Male	49.0	26.8	35.7
% Female	51.0	32.0	41.6
% 5–8 years	49.8	32.2	36.9
% 9–12 years	50.2	26.6	40.4
Mean number of eating occasions per week	29.6	3.1	2.5
Range of eating occasions	13–54	1–16	1–11
% Contribution to energy intakes for the total population at each location	85.1	6.3	8.6
% Contribution to energy intakes for consumers only at each location	85.1	10.7	11.2
% Energy from protein for consumers only at each location	13.8	12.9	10.9
% Energy from fat for consumers only at each location	33.4	35.7	36.9
% Energy from carbohydrate for consumers only at each location	52.3	51.3	51.2

All children consumed food at home over the course of the survey, with an average of 29.6 eating occasions occurring there over the 7 d (4.2 per d). Almost 60% consumed foods at least once in somebody else's home, with a mean number of eating occasions per week of 3.1. Over 77% of the children consumed foods outside the home, with the mean number of eating occasions outside the home equal to 2.5 (with values ranging from 1 to 11).

Foods consumed at home made the greatest contribution to energy intake at 85.1%. Foods outside the home contributed to 8.6% of energy in the total population, and 11.2% in those who were consumers of foods outside the home. The percentage energy from fat was below the recommended 35% of food energy at home (Department of Health, 1991). However, at other people's homes, and particularly outside the home, the percentage of energy from fat was higher than these recommended levels. The percentage energy from carbohydrate was higher than the recommended 50% energy from food at each location.

Food based dietary guidelines could focus on the reduction of fat at locations within the food service sector. However as only 9% of energy comes from foods consumed outside the home, healthy eating guidelines for Irish children should focus on improving the diet in the home environment.

This project was funded by the Irish Government under the National Development Plan 2000–2006.

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Effects of varying the volume of bread in a breakfast meal on satiety and subsequent intakes. By P.A. IRVINE, R.W. WELCH, M.B.E. LIVINGSTONE, P.J. ROBSON, M. McDWYER and G.H. O'CONNOR, *Northern Ireland Centre for Food and Health (NICHE), School of Biomedical Sciences, University of Ulster, Coleraine, UK, BT52 1SA*

Obesity and overweight are reaching epidemic proportions in the UK. Habitual intake of foodstuffs that induce high levels of satiety may be a strategy to help prevent or alleviate overweight and obesity. Previous research has shown that increasing the total volume of pre-loads (yoghurt-based milkshakes), by incorporating different amounts of air, results in a reduction in both appetite and energy intake at the next meal (Rolls *et al*. 2000). The aim of the present study was to evaluate the effects of varying the gas contents, and hence total volumes, of bread rolls given as part of breakfast meal on satiety and subsequent intakes.

The bread rolls were made from white flour and the gas contents (and volumes) of the products were varied by proofing the dough for different times. The present study was conducted using a repeated-measures, randomised, within-subjects cross-over design. Healthy female subjects (n 30; mean age 28.5 (SD 6.3) years; BMI 20–30 kg/m^2) participated on two occasions, 1 week apart. On each occasion subjects consumed the breakfast meal (1897 kJ) at 09.00 hours consisting of bread rolls (90 g fresh weight) with different volumes (233 *v.* 372 ml total roll volume), spreading fat (15 g), preserve (30 g) and orange juice (200 ml). Satiety was measured using visual analogue scales before and after breakfast and every 30 min until 12.00 hours when subjects were offered an *ad libitum* lunch consisting of cottage pie, peaches, pears and fresh cream and drinking water. Lunch intakes were covertly weighed. Following lunch subjects completed food diaries for the remainder of the day. Data were analysed by ANOVA.

Compared with the breakfasts with the smaller-volume rolls, the breakfasts with the larger-volume rolls resulted in significantly (P<0.05) greater fullness and lower hunger for up to 120 min post-consumption. At the *ad libitum* lunch meal, the breakfasts with the larger-volume rolls led to significantly (P<0.05) lower intakes of energy and weight of food (see Table). There were no significant differences in intake for the remainder of the day.

	Small-volume rolls (n 30)		Large-volume rolls (n 30)		
	Mean	SE	Mean	SE	% Difference*
Total energy (kJ)	2778[a]	108	2647[b]	109	−4.9
Weight of food (g)	702[a]	26	668[b]	27	−5.1
Weight of drink (g)	402[a]	24	450[a]	34	10.7
Weight of food and drink (g)	1104[a]	33	1118[a]	34	1.3

[a,b] Mean values within a row with unlike subscript letters are significantly different (P<0.05).
* 100×((Large–small)/large)).

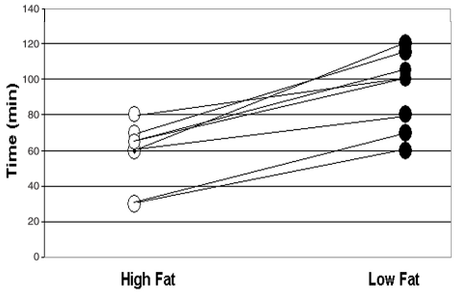
The results obtained in the present study support the concept that foodstuffs with greater gas contents, and hence increased volumes, may lead to enhanced satiety and decreased energy intakes at least in the short term. However, further work is required to evaluate if these effects would persist in the medium-to-longer term.

Rolls BJ, Bell EA & Waugh BA (2000) *American Journal of Clinical Nutrition* **72**, 361–368.

Gastrointestinal transit of high-fat meals in obese humans. By M. CLEGG and A. SHAFAT, *Department of Physical Education and Sport Sciences, University of Limerick, Republic of Ireland*

Obesity is a pandemic with costly co-morbidities and debilitating consequences. The relationship between gastrointestinal transit, satiety and obesity is still unclear. Several studies have tried to determine if there is a difference in gastric emptying and mouth-to-caecum transit time (MCTT) between obese and normal-weight individuals. Current literature demonstrates either accelerated or delayed food transit. The data are confounded by use of high- or low-fat test meals. French *et al.* (1993) found no differences in MCTT in obese and control groups using high- and low-fat meals. The purpose of the present study was to investigate if there was a difference in MCTT and satiation of isoenergetic high- and low-fat meals in obese individuals.

Eight obese (BMI 35.2 (SD 3.6) kg/m^2), male volunteers (31.3 (SD 8.9) years, 113.3 (SD 14.2) kg, 179.4 (SD 4) cm) consented to participate in the present study, approved by the University of Limerick Ethics Committee. All volunteers were asked to record their diet for 7 d using a weighed food diary. On the eighth day, following a 12 h overnight fast, they attended the laboratory and consumed either a high- or low-fat meal, of equal protein and energy (2301 kJ (550 kcal)) content, randomly assigned. The meal was supplemented with 15 g lactulose. Breath H_2 was tested every 5 min using Micro Medical H_2 meter. MCTT was defined as an increase in breath H_2 of 3 parts per million for three consecutive readings. Volunteers were asked to rate their feelings of hunger, thirst, desire to eat, tiredness, fullness and cold using a visual analogue scale every 15 min. Volunteers were instructed to use the 7 d food diary to repeat their diet for the following 7 d. This was analysed using the Compeat Pro nutrition software program. On the 15th day the procedure was repeated using the alternative meal type. Statistical significance ($P<0.05$) was examined with SPSS (version 11.0) using paired *t* tests.



Results indicated that obese individuals had faster MCTT of high-fat food (57.5 (SD 18.1) min) in comparison with a low-fat food transit time of 95.6 (SD 21.5) min ($P=0.000$) (see Fig. 1 which shows MCTT of high- and low fat meals in obese). There were differences in fullness in the visual analogue scores for the two meals over a 90 min period ($P=0.036$). A similar but non-significant trend existed for hunger and desire to eat. Dietary intake results indicated that the volunteers consumed a high-fat diet (38% of total energy intake) compared with that recommended by World Health Organization (2003) guidelines (15–30%).

Castiglione *et al.* (2002) reported that a high-fat diet can cause nutrient-specific changes in gastric emptying. The present results illustrate that high-fat food results in shorter MCTT in obese individuals in comparison with low-fat food. This contradicts work by French *et al.* (1993) yet they did not use isoenergetic meals. Consumption of a high-fat diet may result in faster gastric emptying, intestinal transit, and reduced satiety that in turn may contribute to increased food intake observed in obesity. Further work is required to confirm the differences in gastrointestinal transit between low-fat and high-fat meals in lean individuals.

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Effect of vitamin D supplementation on vitamin D status and bone turnover in young adults. By M.S. BARNES, P.J. ROBSON, M.P. BONHAM, J.J. STRAIN and J.M.W. WALLACE, *Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, UK, BT52 1SA*

Sub-optimal vitamin D status is thought to be prevalent in individuals living at high geographical latitudes, primarily owing to their reduced capacity to synthesise vitamin D during winter months (Calvo *et al.* 2005). As vitamin D plays a central role in bone mineralisation, it is possible that sub-optimal vitamin D status in younger individuals could impact negatively on achievement of their potential peak bone mass (Lehtonen-Veromaa *et al.* 2002). However, few studies have investigated the effects of vitamin D supplementation on vitamin D status and markers of bone metabolism in younger individuals. The aim of the present study was to assess the effects of vitamin D supplementation on 25-hydroxyvitamin D (25(OH)D) levels and bone turnover markers in healthy young individuals living in Northern Ireland (latitude of 55°N).

Thirty individuals (18–27 years) were recruited and randomised to receive either 1500 mg Ca and 15 µg vitamin D3/d (in the form of Calcichew® D_3) (vitamin D group) or 1500 mg Ca/d (Calcichew®) (control group) for 8 weeks during wintertime. At baseline (January) and following intervention (March), plasma 25(OH)D, bone-specific alkaline phosphatase (BAP) and serum cross-laps (CTx) were measured using commercial ELISA kits. Serum Ca and plasma parathyroid hormone (PTH) concentrations were also measured. There were no significant differences in baseline characteristics between the two groups. Mean baseline plasma 25(OH)D concentrations were low in the vitamin D (47.9 (SD 16.0) nmol/l) and control groups (55.5 (SD 18.6) nmol/l). Following supplementation, 25(OH)D concentrations were significantly higher in the vitamin D group (86.5 (SD 24.5) nmol/l) compared with the control group (48.3 (SD 16.8) nmol/l) ($P<0.0001$). There was no significant effect of supplementation on bone turnover markers or PTH concentrations.

	Control group (males *n* 8, females *n* 7)				Vitamin D group (males *n* 7, females *n* 5)				
	Baseline		Post-intervention		Baseline		Post-intervention		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P**
Ca (mmol/l)	2.37	0.13	2.63	0.12	2.34	0.17	2.65	0.10	0.578
25(OH)D (nmol/l)	55.5	18.6	48.3	16.8	47.9	16.0	86.5	24.5	0.000
PTH (pg/ml)	28.6	10.8	25.1	12.8	29.2	16.7	30.6	9.56	0.230
BAP (U/l)	25.3	8.04	25.5	7.13	27.2	8.51	27.2	8.97	0.988
CTx (ng/ml)	0.63	0.26	0.53	0.21	0.59	0.21	0.50	0.29	0.959

* Difference between baseline and post-intervention assessed using analysis of covariance.

These results, albeit based on a small sample, suggest that young adults in Northern Ireland may not consume enough vitamin D to prevent sub-optimal vitamin D status in the wintertime. Although there was no short-term effect of supplementation on bone turnover in the present study, we cannot rule out a long-term effect on bone mineralisation by another mechanism. If further research confirms that vitamin D insufficiency is widespread among young individuals, it is likely that public health policies concerning vitamin D fortification and supplementation may have to be revised.

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Vitamin D status of adolescents from the Northern Ireland Young Hearts Project: effects of age, sex and season. By T.R. HILL[1], A.A. COTTER[1], J. WALLACE[3], P.J. ROBSON[3], C. BOREHAM[3], W. DUBITZKY[3], L. MURRAY[4], A. FLYNN[1], M. KIELY[1] and K.D. CASHMAN[1,2], [1]*Department of Food and Nutritional Sciences,* [2]*Department of Medicine, University College, Cork, Republic of Ireland,* [3]*Northern Ireland Centre for Food and Health, University of Ulster, Coleraine, UK and* [4]*Department of Epidemiology and Public Health, Queens University, Belfast, UK*

Sub-optimal vitamin D status, as measured by serum or plasma 25-hydroxyvitamin D (25 (OH) D), is considered to be an important determinant of peak bone mass in adolescents. A high prevalence of sub-optimal vitamin D status has been reported among adolescents from several European countries (Andersen *et al.* 2005). The objective of the present study was to assess serum 25 (OH) D levels among a representative sample of adolescents from Northern Ireland and to determine the impact of season, age and gender on serum 25 (OH) D levels.

Serum samples for the present study were obtained from the Northern Ireland Young Heart's Project (Gallagher *et al.* 2002). In total, 997 adolescents (500 boys and 497 girls) were studied. All subjects were aged either 12 or 15 years at the time of blood sampling, which was conducted throughout the year. Serum was analysed for 25 (OH) D by enzyme-immunoassay (IDS Ltd, Boldon, Tyne and Wear, UK). The quality of our serum 25 (OH) D analysis is assured on an ongoing basis by participation in the DEQAS external quality assurance scheme (London, UK).

Two-way ANOVA showed that serum 25 (OH) D levels were not affected by age (12- or 15-year-olds) or sex and there was no significant interaction between sex and age. Therefore, vitamin D status is presented for the entire group (*n* 997) according to percentiles.

Percentile ...	5th	10th	25th	50th	75th	90th	95th
Serum 25 (OH) D (nmol/l)	28.1	32.7	44.1	61.1	81.2	100.0	116.5

There is no international consensus on serum 25 (OH) D cut-off levels for defining vitamin D insufficiency (McKenna & Freaney, 1998). Therefore, percentages of adolescents whose vitamin D status falls below several cut-offs are presented.

Serum 25 (OH) D (nmol/l) ...	<10	<20	<30	<40	<50	<60	<70	<80
Percentage of subjects	<1	1	7	20	36	49	62	74

Using a serum 25 (OH) D cut-off value of 50 nmol/l for defining vitamin D insufficiency (Lips, 2004), about 36% of subjects were insufficient. The prevalence of vitamin D insufficiency during summer (June–August), autumn (September–November), winter (December–February) and spring (March–May) was 8% (*n* 66), 13% (*n* 321), 52% (*n* 368) and 48% (*n* 242), respectively.

In conclusion, a significant number of Northern Irish adolescents are at risk of sub-optimal vitamin D status, especially during winter and spring. This low vitamin D status may limit the achievement of peak bone mass.

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Vitamin D intakes in Irish 5–12-year-old schoolchildren. By M. KIELY, E.M. HANNON, J. WALTON and A. FLYNN, *Department of Food and Nutritional Sciences, University College Cork, Republic of Ireland*

At high (or low) latitudes, dermal synthesis of vitamin D does not occur during winter. Global awareness of the risks of UVB-mediated skin damage has prompted the extensive use of sunscreen, which blocks dermal synthesis of vitamin D during summer. The half-life of serum 25-hydroxy vitamin D (25-OH D), the standard biomarker of vitamin D status, is only 2–3 weeks. Thus, individuals are largely and increasingly dependent on dietary vitamin D to maintain vitamin D status (Holick, 2004).

Evidence is accumulating that sub-optimal vitamin D status is endemic in the Irish population. Low serum concentrations of 25-OH D have been reported in Irish postmenopausal women (Hill *et al.* 2005*a*), elderly women, girls aged 11–13 years (Andersen *et al.* 2005) and boys and girls, aged 12 and 15 years (Hill *et al.* 2005*b*). Sources of vitamin D are scarce in the typical Irish diet and 74% of a representative sample of adults had a mean vitamin D intake <5 μg/d (Hill *et al.* 2004).

The National Children's Food Survey was carried out by the Irish Universities Nutrition Alliance at University College Cork and Trinity College Dublin from April 2003 to April 2004. A representative sample of 596 children aged 5 to 12 years was obtained by random selection from the national primary schools database, taking geography, school size and socio-economic factors into account. Food and beverage intakes were measured using a weighed 7 d food diary that included details on the use of nutritional supplements. The 6th Edition of *The Composition of Foods* (Food Standards Agency, 2002) was adjusted to incorporate data on supplements and fortified, composite and novel foods. Vitamin D intakes in 594 children (293 boys and 301 girls) are reported.

Mean intakes of vitamin D were 2.3 (SD 1.5) μg/d and there was no effect of age, sex or season. The main determinant of vitamin D intake was the use of nutritional supplements. One hundred and eight children (18%) consumed vitamin D in supplemental form and the mean intake of total vitamin D intake in these children was 5.6 (SD 2.8) μg/d, which was almost four times higher than the intake of 1.5 (SD 1.2) μg/d in non-users. In the absence of an reference nutrient intake (RNI) for vitamin D for this age group, the Table shows the numbers and percentages of children (classified as users and non-users of vitamin D-containing supplements) that have mean daily vitamin D intakes below 10, 5 and 1 μg/d.

	All (*n* 594)		Non-users (*n* 486)		Users (*n* 108)	
Daily vitamin D intakes (μg/d)	*n*	%	*n*	%	*n*	%
<10	585	98	486	100	99	92
<5	524	88	476	98	48	44
<1	203	34	202	42	1	1

These data show that Irish schoolchildren, particularly those that do not use vitamin D-containing supplements, have very low intakes of vitamin D, which places them at risk of hypovitaminosis D. An evidence basis for the establishment of an RNI for vitamin D in children is urgently required.

The present study was supported by the Irish Government Department of Agriculture and Food through the Food Institutional Research Measure as part of the National Development Plan 2000–2006.

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Investigation of B vitamin status in lean and obese women: a pilot study. By M.A. KERR[1], H. McNULTY[1], J.M. SCOTT[2], A.M. MOLLOY[2], M. WARD[1], N. SULLIVAN[1], C. PUTLAND[1] and M.B.E. LIVINGSTONE[1], [1]*Northern Ireland Centre for Diet and Health (NICHE), University of Ulster, Coleraine, UK, BT52 1SA and* [2]*Department of Biochemistry, Trinity College, Dublin, Republic of Ireland*

Obese women compared with non-obese women are reported to have an up to 3-fold greater risk of a pregnancy affected by a neural tube defect (NTD) (Shaw *et al.* 1996; Werler *et al.* 1996). The basis for this effect is poorly understood, but it is not explained by lower dietary folate intake or non-use of folic acid supplementation. However, it is possible that impaired status of folate and/or of a metabolically related B vitamin may be implicated in the excess risk of NTD among obese women. We have recently reported significantly lower vitamin B_{12} status among obese British children aged 4–18 years, an effect not explained by age, sex or dietary B_{12} intake (Kerr *et al.* 2004). Lower vitamin B_{12} status has, in turn, been associated with a higher risk of NTD, independent of folate status (Kirke *et al.* 1993; Suarez *et al.* 2003). In the present study our aim was to investigate vitamin B_{12} status in obese and lean women. From a larger screening sample of healthy women (*n* 146), we recruited twenty-five obese (BMI>30 kg/m^2) women and twenty-three lean (BMI 20–24.9 kg/m^2) age-matched controls. Baseline blood samples were analysed for plasma B_{12}, plasma folate and plasma homocysteine. Weight (kg) and height (m) measurements were taken to determine BMI (wt/ht^2). Body composition was assessed using air displacement plethysmography (BOD POD; Life measurement, Inc, Concord, CA, USA).

	Lean			Obese			
	n	Mean	SD	*n*	Mean	SD	*P**
BMI (kg/m^2)	23	22.9	1.1	25	34.5	5.0	0.000
Body fat (%)[2]	17	29.7	5.7	14	47.8	5.0	0.000
Age (years)	23	30.9	10.3	25	30.3	9.2	0.847
Plasma B_{12} (pg/ml)	23	469	153	25	378	135	0.035
Plasma folate (ng/ml)	23	9.0	7.5	25	5.9	4.1	0.079
Homocysteine (μmol/l)	23	7.8	1.8	25	8.9	3.3	0.189

*Values were compared using an independent *t* test. Data were log-transformed for normalisation purposes as appropriate.

Vitamin B_{12} status was significantly lower among obese compared with lean women (see Table). In addition, obese women tended to have lower folate concentrations, and higher homocysteine, however, not significantly so in either case. BMI was found to be significantly correlated with both vitamin B_{12} (r −0.340; P=0.019) and folate (r −0.293; P=0.044), but not with homocysteine (r 0.192; P=0.192). Importantly, we observed a strong correlation between vitamin B_{12} and percentage body fat (r −0.547; P=0.002); however, no significant correlation was found between percentage body fat and either folate (r −0.292; P=0.111) or homocysteine (r 0.202; P=0.276). The finding that vitamin B_{12} was much more strongly correlated with percentage body fat than with BMI suggests that excess adiposity *per se* is associated with an impaired vitamin B_{12} status in obese women. In conclusion, obese women appear to have lower vitamin B_{12} status, which may in turn be associated with an increased risk of NTD. Further research is warranted to examine the underlying mechanism.

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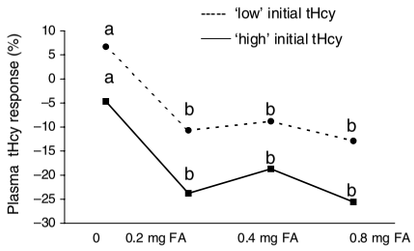
The impact of initial vitamin B_{12} status on the extent of plasma homocysteine lowering with folic acid. By P. TIGHE[1], M. WARD[1], H. McNULTY[1], O. FINNEGAN[2], J.J. STRAIN[1], A. DUNNE[3], A.M. MOLLOY[4] and J.M. SCOTT[4], [1]*Northern Ireland Centre for Diet and Health, University of Ulster, Coleraine, UK, BT52 1SA,* [2]*Coronary Care Unit, Causeway Hospital, Coleraine, UK, BT52 1HS,* [3]*Department of Statistics, University College Dublin, Belfield, Dublin 4, Republic of Ireland and* [4]*Department of Biochemistry, Trinity College, Dublin, Republic of Ireland*

Food fortification with folic acid (FA), although primarily aimed at reducing neural tube defects (NTD), is expected to have some benefit in terms of the primary and secondary prevention of CVD via a homocysteine (tHcy)-lowering effect. Vitamin B_{12} is also essential for tHcy metabolism but is currently overlooked in food fortification programmes. Recent reports indicate that in the face of optimised folate status, vitamin B_{12} becomes the major determinant of tHcy concentration (Liaugaudas *et al.* 2001; Quinlivan *et al.* 2002). We previously reported that 0.2 mg/d FA administered for 26 wks maximally lowered tHcy with no additional lowering achieved at higher doses (0.4, 0.8 mg/d; Tighe *et al.* 2004). The aim of the present investigation was to examine the influence of baseline tHcy and its determinants on the extent of the response of tHcy to FA. We re-analysed our data by splitting the sample into 'high' or 'low' tHcy (i.e. above or below the median tHcy for each treatment group) at baseline.

Results showed that 0.2 mg/d FA was the optimal homocysteine-lowering dose irrespective of initial tHcy concentration, ANOVA with bonferroni post-hoc test (Fig). The extent of response to FA was limited in those with a higher baseline tHcy, in that even after 6 months of 0.8 mg/d FA, tHcy concentrations were 3 μmol/l higher in those classed as 'high' v 'low' at baseline (not shown). We then investigated the characteristics at baseline of the 'high' group and found they were significantly older and had lower status of folate (as expected) and vitamin B_{12}. In a subset of age-matched volunteers, serum vitamin B_{12} was also significantly lower in the 'high' v 'low' group (Table).



	'Low' (*n*=70)	'High' (*n*=67)	*P*
Age (y)	60.4 (7.3)	62.9 (8.9)	0.114
tHcy (μmol/l)	10.1 (1.9)	16.6 (6.7)	<0.0001
Serum folate (μg/l)	12.2 (7.2)	7.9 (4.5)	<0.0001
Vitamin B12 (ng/l)	319 (100)	254 (100)	<0.0001
Plasma PLP (nmol/l)	76.9 (42.0)	74.8 (75.4)	0.478
Riboflavin (EGRac)	1.28 (0.15)	1.31 (0.14)	0.217

Values are presented as mean (SD).

Although FA was highly effective in reducing tHcy concentrations in both groups, it appears that in volunteers with a suboptimal vitamin B_{12} status at baseline, folic acid alone will not lower tHcy to desirable levels (i.e.<10 μmol/l). Given that the relationship between tHcy and CVD is graded, any small but significant decrease in tHcy (over and above that achieved with folic acid alone) could be predicted to confer an additional benefit in terms of CVD risk. Consideration, therefore, should be given to vitamin B_{12}, in addition to FA, in future fortification strategies.

This research was supported by the Northern Ireland Chest, Heart and Stroke Association.

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Planned pregnancy is no guarantee of folic acid compliance. By U.B. FALLON[1], G. BURY[1], S. DALY[1], F. HANNON[2], B.G. LOFTUS[2], J. MORRISON[2], A.W. MURPHY[2], C.M. MURRIN[1,3], G. NOLAN[3], D. O'MAHONY[2] and C.C. KELLEHER[1,3], [1]*Health Research Board Unit for Health Status and Health Gain, University College Dublin, Republic of Ireland,* [2]*National University of Ireland, Galway, Republic of Ireland and* [3]*National Nutrition Surveillance Centre, University College Dublin, Republic of Ireland*

Randomised controlled trials have demonstrated that folic acid consumed in the pre-conception period prevents both primary and recurrent neural tube defects (MRC Vitamin Study Research Group, 1991). 'Folic Acid – One of Life's Essentials' is a cross-border health promotion campaign, encouraging women who are likely to become pregnant to take 400 µg folic acid daily as well as consuming a diet rich in folate. The Food Safety Authority of Ireland is presently (March to June 2005) engaged in public consultation on folic acid fortification. Despite high awareness of the benefits of taking folic acid this is not matched by pre-conception intake (Ward *et al.* 2004). The aim of this analysis is to examine factors, which are associated with the use of folic acid before conception including age, education, medical card status, planned pregnancy and martial status.

The Lifeways study is a longitudinal population-based, cross-generational cohort study comprising of 1120 mothers recruited during their first antenatal visit between October 2001 and January 2003 in Galway and Dublin. In addition to the routine EuroKing ante-natal and birth record, the mothers self-completed a questionnaire providing information on their health, socio-demographic status, pregnancy, diet and family.

Women in Lifeways cohort who consumed Folic Acid supplements during the three months before conception

Folic acid supplement use	Yes % (*n*)	No % (*n*)	Total *n* (%)
Total	45.0 (388)	54.9 (472)	860 (100)
GMS card	25.6 (34)	74.4 (99)	133 (15.5)
No GMS card	48.6 (351)	51.5 (372)	723 (84.5)
			856 (100) ($P=0.001$).
Planned	61.0 (291)	39.0 (186)	477 (59.8)
Not Planned	8.5 (23)	91.5 (248)	271 (36.2)
			748 (100) ($P=0.0001$)
Third level education	49.2 (216)	50.8 (223)	439 (51.0)
Completed secondary education	44.5 (121)	55.5 (151)	272 (31.6)
< completed secondary education	33.0 (43)	66.9 (87)	150 (17.4)
			861 (100) ($P=0.005$).
Married	58.5 (336)	41.5 (238)	574 (68.4)
Single	12.7 (20)	87.3 (138)	158 (18.8)
Co-habitating	29.2 (31)	70.8 (75)	106 (12.6)
			838 (100)
Mean age	31.7 (4.4)	28.0 (6.2)	855 (100) ($P=0.0001$)

GMS, eligible for medical card.

Overall pre-conception folic acid consumption of 45% is high relative to other Irish studies. This may be due to a positive reporting bias as 23% of women omitted to answer the folic acid questions. Despite possible over-reporting, we have demonstrated that even when the pregnancy is planned, 39% of women did not take folic acid during this critical time. We have also demonstrated significant health inequality in that younger, poorer, less educated women are less likely to take folic acid. Health promotion campaigns to date have not been effective. Compulsory fortification of flour with folic acid should be considered.

MRC Vitamin Study Research Group (1991) *Lancet* **338**, 131–137.
Ward M, Hutton J, McDonnell DR, Bachir N, Scallan E, O'Leary M, Hoey J, Doyle A, Delany V & Sayers G (2004) *Irish Medical Journal* **97**, 274–276.

Folic acid consumption in pregnancy leads to the appearance of unmetabolised folic acid in cord-blood and in infants. By M.R. SWEENEY[1,3], J. McPARTLIN[1], D.G. WEIR[1], S. DALY[4], K. PENTIEVA[5], L. DALY[3] and J.M. SCOTT[2], *Departments of* [1]*Clinical Medicine and* [2]*Biochemistry, University of Dublin, Trinity College, Republic of Ireland,* [3]*Department of Public Health Medicine and Epidemiology, University College Dublin, Republic of Ireland,* [4]*Coombe Woman's Hospital, Dublin, Republic of Ireland and* [5]*Northern Ireland Centre for Diet and Health, University of Ulster, Coleraine, UK, BT52 ISA*

Oral folic acid above certain threshold doses results in unmetabolised folic acid in serum. This raises a number of public health safety issues, principally the potential to mask pernicious anaemia (Scott & Weir, 1998), and more recently the theoretical potential for high-dose folic acid to promote cancer has been highlighted (Charles *et al.* 2004; Young-In, 2004).

In the present paper we set out to examine the appearance of unmetabolised folic acid both in cord-blood and in infants post-formula feeding. The study was conducted in the Coombe Woman's Hospital, Dublin. Blood was collected from the umbilical cord of eleven infants in the delivery room immediately after birth and serum was separated. A follow-up serum sample (*n* 9) was collected 4 d later from infants post-formula feeding.

We detected unmetabolised folic acid in cord-blood from all infants at birth. Statistical analysis was based on the Wilcoxon signed rank test and exact 95% confidence limits for a proportion. In addition unmetabolised folic acid was present in serum of seven infants post-formula feeding, six of which had increased from birth.

Table 1. Unmetabolised folic acid (µg/L) in serum of premature and full term infants pre and post formula feeding.

	Sample 1. Baseline cord-blood (serum)	Sample 2. Day 4 (venopuncture)
Minimum	0.08	0.00
Maximum	0.47	1.30
Mean	0.185	0.468
Standard Deviation	0.12233	0.43144

Our results imply that over 70% of infants in Ireland, which does not yet have mandatory fortification, could have circulatory unmetabolised folic acid at the time of birth. We do not know if the presence of folic acid in cord-blood will have any adverse consequences. However, if theoretical safety concerns are borne out by future research, the likelihood is that the longer the exposure the more likely the potential for harm. This would also be the case in infants exposed to unmetabolised folic acid as a result of formula feeding.

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Young-In K (2004) *American Journal of Clinical Nutrition* **80**, 1123–1128.

Phytosterol, tocopherol and squalene content of five edible nuts (Brazil, pecan, pine, pistachio, cashew). By E. RYAN[1], K. GALVIN[1], T.P. O'CONNOR[1], A.R. MAGUIRE[2], F. McCARTHY[2] and N.M. O'BRIEN[1], [1]*Department of Food and Nutritional Sciences and* [2]*Department of Chemistry and School of Pharmacy, Analytical and Biological Chemistry Research Facility, University College Cork, Republic of Ireland*

Nuts are typically high in fat content but have a fatty acid profile that may be beneficial in relation to risk of CHD. In addition, nuts contain bioactive constituents that elicit cardio-protective effects including phytosterols, tocopherols and squalene. Phytosterols are membrane constituents of plants that have been shown to reduce absorption of dietary cholesterol. β-Sitosterol, campesterol and stigmasterol represent the most abundant phytosterols. Tocopherols are powerful antioxidants and in high doses have been shown to lower the risk of CHD. Squalene, a biosynthetic precursor to all steroids in plants and animals, has been reported to decrease the risk for various cancers and reduce serum cholesterol levels. The objective of the present study was to determine and compare the total oil content and also the levels of phytosterols, tocopherols and squalene in five edible nuts. Oil was extracted from freshly ground Brazil, pecan, pine, pistachio and cashew nuts based on a procedure previously outlined by Maguire *et al.* (2004). The extracted oil was evaluated by HPLC for levels of phytosterols, tocopherols and squalene.

	% Yield		β-Sitosterol (μg/g oil)		Campesterol (μg/g oil)		Stigmasterol (μg/g oil)		α-Tocopherol (μg/g oil)		γ-Tocopherol (μg/g oil)		Squalene (μg/g oil)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Brazil	61	1	1325	68	27	4	578	34	83	10	116	5	1378	8
Pecan	58	3	1572	41	52	7	340	30	12	3	169	16	152	11
Pine	59	2	1842	125	215	14	681	46	124	9	105	7	40	8
Pistachio	51	1	4686	154	237	25	663	61	16	1	275	20	91	19
Cashew	40	2	1768	211	105	16	115	13	4	1	57	6	89	10

Result are the mean value for at last three independent experiments.

The total oil content of the nuts ranged from 40 to 61% (w/w), with the Brazil nut yielding the greatest percentage of oil. β-Sitosterol was the most prevalent phytosterol, ranging in concentration from 325 to 4686 μg/g oil. Stigmasterol and campesterol were also present in significant amounts in all five nuts. The order of decreasing total phytosterol content was pistachio >pine >Brazil >pecan >cashew. γ-Tocopherol was the most abundant tocopherol in all nuts except the pine nut. Levels ranged from 57 to 245 μg/g oil with the pistachio nut yielding the greatest concentration. α-Tocopherol was measured in concentrations ranging from 4 μg/g oil in the cashew nut to 124 μg/g oil in pine nuts. The order of decreasing total tocopherol content was pistachio >pine >Brazil >pecan >cashew. The levels of squalene detected ranged from 4 μg/g oil in the pine nut to 1378 μg/g oil in the Brazil nut. In conclusion, the present data indicate that whilst nuts contain appreciable amounts of phytosterols, tocopherols and squalene, levels vary greatly between nut types. Cashews nuts had the least oil, phytosterol and tocopherol content whilst pistachio nuts had the greatest phytosterol and tocopherol content.

Maguire LS, O'Sullivan MS, Galvin K, O'Connor TP & O'Brien NM (2004) *International Journal of Food Sciences and Nutrition* **55**, 171–178.

Effects of watercress on biomarkers for colon cancer. By A. BOYD, M. McCANN, Y. HASHIM, S. HALDAR, C. GILL and I. ROWLAND, *Northern Ireland Centre for Diet and Health, University of Ulster, Coleraine, UK, BT52 1SA*

Cruciferous vegetables contain an important group of sulfur-containing, water-soluble phytochemicals known as glucosinolates. Hydrolyses of glucosinolates to aglucone, glucose and sulfate via the enzyme Myrosinase leads to the production of isothiocyanates (ITCs), such as phenethyl isothiocyanate, sulforaphane and indole 3-carbinol, which are thought to modulate the activity of phase I and phase II enzymes that are responsible for the bio-activation and detoxification of carcinogens (Chiao *et al.* 2004).

The aim of the present *in vitro* study was to examine the effects of watercress crude extract (Kassie *et al.* 1996) on a selection of biomarkers biologically relevant to colorectal cancer. These included: (1) initiation, in which the protective effects were observed against genotoxin-induced DNA damage; (2) proliferation, in which the effects on the progression of the cell cycle were investigated; (3) metastasis, in which the protective effects of the extract were investigated against invasion of colon cancer cells through a Matrigel coated porous insert. HT29 cells were used to investigate the protective effects of the extract on DNA damage and the cell cycle. DNA damage was induced using a range of genotoxins, namely H_2O_2, faecal water and 4-hydroxy nonenal and the extent of damage to the DNA was measured using the single-cell gel electrophoresis assay. Statistical analysis was performed in both assays using a one-way ANOVA test (n 3; $P=0.05$). HT115 cells were used to examine the effects of the extract on invasion. Statistical analysis was carried out using Dunnett's *t* test (*n* 3; $P=0.05$).

Watercress Conc (μl/ml)	% Tail DNA+ H_2O_2	% Tail DNA+ Faecal water	Cells in S phase of Cell Cycle	% Invasion
0	49	49	12.8575	100
5	43	49	19.4375	30.518*
10	40	45	19.8525	19.8476*
20	N/A	N/A	20.1375*	5.2337*
50	36**	40*	25.385*	1.96526*

Mean values were significantly different to control. *$P<0.05$, **$P<0.01$.

In each of the three experiments, the watercress proved to be significantly protective against the three stages of the carcinogenesis process investigated. The extract proved to inhibit DNA damage against two of the three genotoxins used. The anti-genotoxic activity observed in the present study is approaching previously recorded ranges for watercress and other cruciferous vegetable extracts (Kassie *et al.* 2003; Gill *et al.* 2004). Watercress extract caused an accumulation of cells in the S phase of the cell cycle during the present study, indicating possible cell cycle arrest at this stage. A significant reduction in invasion rates was also observed in response to the watercress extract. The protective effects of the extract on invasion and adhesion observed during the present study occurred without a change in total cell numbers or cell viability at any of the concentrations tested. This suggests that the protective effects observed were not linked to a decrease in cell number through cytotoxicity of the extract itself.

The chemoprotective effects observed in response to watercress extract during the present study, could explain the anticarcinogenic effect noted in epidemiological studies demonstrating a link between an increase in consumption of cruciferous vegetables and a decreased risk of colorectal cancer.

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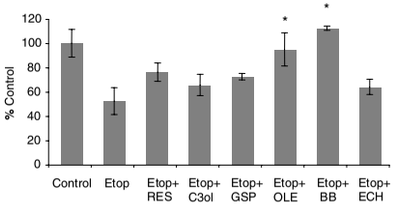
Kassie F, Laky B, Gminski R, Mersch-Saundermann V, Scharf G, Lhoste E & Knassmuller S (2003) *Chemico-Biological Interactions* **142**, 285–296.
Kassie F, Parzefall W, Musk S, Johnson I, Lamprecht G, Sontag G & Knassmeller S (1996) *Chemico-Biological Interactions* **102**, 1–16.

Modulatory effects of resveratol, citroflavan-3-ol and plant-derived extracts on oxidative stress in U937 cells. By R. CARPENTER, Y.C. O'CALLAGHAN, M. O'GRADY, J.P. KERRY and N.M. O'BRIEN, *Department of Food and Nutritional Sciences, University College Cork, Republic of Ireland*

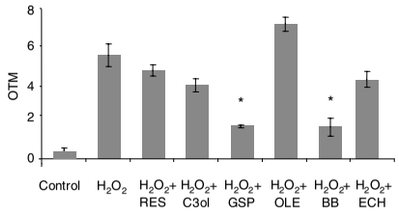
Phytochemicals and plant extracts, present in fruit, vegetables, plants, herbs and beverages, have been shown to have antioxidant potential which may modulate the aetiology of certain chronic diseases (Duthie *et al.* 2003). Whilst the biological activities of flavonoids and carotenoids are well documented, little is known about other phytochemicals and plant extracts. The objective of the present study was to determine the IC_{50} value of a range of phytochemicals and plant extracts and to investigate their antioxidant and genoprotective effects under conditions of oxidative stress towards U937 cells, a human monocytic cell line.

U937 cells were grown in RPMI 1640 medium supplemented with 2.5% fetal bovine serum. Two phytochemicals, resveratrol (RES) and citroflavan-3-ol (C3ol) and four plant extracts, grapeseed polyphenols (GSP), olive leaf extract (OLE), bearberry (BB) and echinacea purpurea (ECH) were examined. Compounds were administered in methanol except ECH, which was administered in distilled water. Control cells were treated with an equal volume of carrier vehicle and incubated for 24 h (37 °C; 5% CO_2). Viability was assessed by the fluorescein diacetate–ethidium bromide assay. Concentration of compound that inhibited cell growth by 50% (IC_{50}) was calculated. To examine their antioxidant and genoprotective effects, cells were pretreated at levels below the IC_{50} resulting in greater than 95% cellular viability of RES (2 μg/ml), C3ol (100 μg/ml), GSP (50 μg/ml), OLE (50 μg/ml), BB (10 μg/ml) or ECH (1 mg/ml). U937 cells were then exposed to oxidants: 0.5 μM-etoposide (Etop) or 100 μM-H_2O_2 or 400 μM-*tert*-butylhydroperoxide (tBOOH). Cellular glutathione (GSH) levels were measured as an indicator of oxidative stress. DNA damage was assessed by the alkaline single-cell gel electrophoresis (ASCGE) assay or comet assay and results expressed as olive tail moment (OTM), an arbituary unit of DNA damage.



n 3, * $P<0.05$, ANOVA followed by Dunnett's test.

Fig. 1. GSH levels in U937 cells.



n 3, * $P<0.05$, ANOVA followed by Dunnett's test.

Fig. 2. DNA damage in U937 cells.

Exposure of U937 cells to phytochemicals and plant extracts decreased cell viability in a dose-dependent manner. RES demonstrated the highest IC_{50} value of 14 μg/ml with ECH the lowest at 9 μg/ml. Etop-induced oxidative stress was strongly reduced by BB and OLE, evident by an increase in GSH levels to 112% and 94% of control respectively (Fig. 1). GSP and BB decreased H_2O_2-induced DNA damage leading to a 3-fold reduction in OTM (Fig. 2). Similar trends were seen with tBOOH (data not shown). In conclusion, these results provide evidence that non-nutrient dietary constituents may act as significant bioactive compounds and that plant extracts, such as BB, GSP and OLE strongly protect against oxidative stress, a contributory factor in the pathogenesis of human diseases such as cancer and CVD.

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An investigation of the anti-apoptotic efficiency of apigenin, astaxanthin and lycopene in cholesterol oxide-treated human monocytic blood cells. By S. LORDAN, Y.C. O'CALLAGHAN and N.M. O'BRIEN, *Department of Food and Nutritional Sciences, University College Cork, Republic of Ireland*

Cholesterol oxidation products (oxysterols) arise from the enzymic or non-enzymic oxidation of cholesterol. Although cholesterol itself is not cytotoxic, a number of potential biological activities of oxysterols have been reported. The oxysterol 7β-hydroxycholesterol (7β-OH) has been shown to induce oxidative stress in U937 cells leading to apoptotic cell death (O'Callaghan *et al.* 2001). Agents or antioxidants that can inhibit the production of reactive oxygen species can prevent apoptosis (Choi *et al.* 2003). Due to the fundamental importance of apoptosis in pathological processes, the identification of substances capable of modulating this form of cell death is now actively researched. Flavonoids and carotenoids are important antioxidant phytochemicals found in a large number of fruits and vegetables. The objective of the present study was to investigate if the flavonoid apigenin, and the carotenoids astaxanthin and lycopene, could inhibit 7β-OH-induced apoptosis in U937 cells.

U937 cells were adjusted to a density of 1×10^5 cells/ml in RPMI 1640 medium supplemented with fetal bovine serum (25 ml/l). Cells were pretreated with the antioxidants for 1 h followed by treatment with 30 μM-7β-OH. After 24 h, cell viability was assessed by the fluorescein diacetate–ethidium bromide assay and apoptotic nuclei were quantified following staining with Hoechst 33342.

	Control		7β-OH (30 μM)		α-TOC (10 μM)		Apigenin 0.5 μM		2 μM		5 μM		10 μM		50 μM	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
% Viable	98	1	70	2	81*	1	65	5	78	4	80	2	70	1	25	4
% Apoptotic	3	1	19	1	13*	1	16	2	15	2	17	2	29	6	68	11

Table 1: values represent the mean of three independent experiments (*$P<0.05$, paired *t* test).

	Control		7β-OH (30 μM)		α-TOC (10 μM)		Carotenoids 0.1 μM		0.5 μM		1 μM	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Lycopene												
% Viable	97	1	67	3	83	5	72	3	65	6	66	3
% Apoptotic	4	0	12	2	7	1	13	1	13	1	16	2
Astaxanthin												
% Viable	95	1	85	2	92	2	88	0	79	2	51	7
% Apoptotic	6	0	12	2	8	1	10	2	16	0	49	2

Table 2: values represent the mean of three independent experiments.

The antioxidant α-tocopherol (α-TOC) has previously been shown to decrease the toxicity of 7β-OH to U937 cells (Lyons *et al.* 2001). Similarly, in the present study, 10 μM-α-TOC increased the viability and decreased the apoptotic death of cells exposed to 7β-OH (Table 1). Apigenin at low concentrations (2 and 5 μM) partially protected against oxysterol-induced cell death (Table 1). However, at higher concentrations (50 μM), apigenin intensified cell death and did not protect the cells from 7β-OH-induced apoptosis (Table 1). Neither of the carotenoids, at the concentrations tested, opposed the adverse effects of 7β-OH in U937 cells (Table 2). Higher concentrations of astaxanthin potentiate the toxicity of 7β-OH. In conclusion, our data suggest that the flavone apigenin offers some protection against oxysterol-induced cell death and this protection is concentration dependent. On the other hand, the carotenoids astaxanthin and lycopene offered no protection. These findings warrant further studies of flavonoids for their protection against oxysterol-induced toxicities.

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Anti-inflammatory effects of eicosapentaenoic and docosahexaenoic acid on classically activated human THP1 monocyte-derived macrophages. By A. MULLEN, S.M. WELDON, C.E. LOSCHER and H.M. ROCHE, *Nutrigenomics Research Group, Institute for Molecular Medicine, Trinity Centre for Health Sciences, St. James' Hospital, Dublin 8, Republic of Ireland*

Atherosclerotic plaque contains leukcocytes, of which approximately 80% are monocyte-derived macrophages. The monocyte-derived macrophage can exacerbate a pro-inflammatory milieu by secretion of cytokines such as IL-6, IL-1β and TNF-α. NF-κB p65 activates transcription of these, and other, pro-inflammatory mediators. IκBα maintains NFκB p65 in the cytoplasm, preventing its nuclear translocation, or may shuttle it out of the nucleus. Fish oil, rich in the n-3 PUFA eicosapentaenoic (EPA) and docosahexaenoic (DHA) acids, is reported to ameliorate inflammation and has been associated with reduced pro-inflammatory phenotypes in human and animal supplementation and feeding trials (Pischon *et al.* 2003; Trebble *et al.* 2003). The aim of the present study was to investigate the effects of EPA and DHA on pro-inflammatory mediators of classically activated human macrophages.

THP1 human leukaemia monocytes were differentiated to macrophages and starved of serum for 24 h before a 48 h treatment with EPA, DHA or dimethyl sulfoxide (DMSO) control. Lipopolysaccharide (LPS) was subsequently used to stimulate macrophages. IL-6, IL-1β and TNF-α secretion were measured by ELISA and transcription by real time RT-PCR. Nuclear and cytosolic NF-κB p65 and IκBα expression were investigated by Western immunoblotting. ANOVA from DataDesk® 6.0 (Data Description Inc., Ithaca, USA) were performed and a p value ≤0.05 was considered statistically significant.

EPA significantly reduced IL-6, IL-1β and TNF-α secretion ($P \leq 0.02$) and IL-6 and TNF-α transcriptionally ($P \leq 0.04$). DHA significantly reduced IL-6 and IL-1β secretion ($P \leq 0.01$) and IL-6 and IL-1β transcriptionally ($P \leq 0.01$). At 1 h of LPS stimulation, DHA reduced expression of nuclear NF-κB p65 and IκBα ($P \leq 0.04$) and increased both in the cytoplasm ($P \leq 0.02$). EPA increased cytoplasmic expression of p65 (I<0.0001). At 5 h of LPS stimulation DHA increased cytoplasmic p65 and IκBα ($P \leq 0.03$). EPA increased cytoplasmic IκBα ($P = 0.03$). Both n-3 PUFA decreased nuclear IκBα ($P \leq 0.009$) compared to DMSO control.

Human and animal feeding studies of n-3 PUFA mixes have indicated their anti-inflammatory potential. The present study has elucidated the effects of the individual component fatty acids in human macrophages. EPA significantly reduced IL-6, IL-1β and TNF-α secretion and IL-6 and TNF-α transcription. DHA significantly reduced IL-6 and IL-1β secretion and transcription. Inhibition of NF-κB phosphorylation by n-3 fatty acid emulsions has recently been demonstrated (Novak *et al.* 2003). We have shown that both EPA and DHA limit NF-κB p65 translocation to the nucleus, and DHA appears to be most potent in this regard. It has recently been reported that both EPA and DHA down regulate LPS-induced NF-κB:DNA binding in THP-1 macrophage (Weldon *et al.* 2005).

Macrophages are the predominant inflammatory cell in the atherosclerotic plaque. The present study has demonstrated the anti-inflammatory effects of the n-3 PUFA EPA and DHA in a human macrophage model. The secretion and transcription of the pro-inflammatory cytokines IL-6, IL-1β and TNF-α were decreased by EPA and/or DHA. The nuclear translocation of the transcription factor NF-κB p65 was generally inhibited by treatment with the n-3 PUFA in this classically activated macrophage model.

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Effect of conjugated linoleic acid on global gene expression in human intestinal-like Caco-2 cells. By E.F. MURPHY[1], G.J.E.J. HOOIVELD[2], M. MULLER[2] and K.D. CASHMAN[1,3], [1]*Department of Food and Nutritional Sciences, University College, Cork, Republic of Ireland,* [2]*Nutrition, Metabolism and Genomics Group, Division of Human Nutrition, Wageningen University, The Netherlands and* [3]*Department of Medicine, University College, Cork, Republic of Ireland*

Conjugated linoleic acid (CLA), especially the *trans*-10, *cis*-12 (*t*10, *c*12) isomer, has been shown to enhance transepithelial Ca transport in human intestinal-like Caco-2 cells via an increased transcellular and paracellular rate of transport (Jewell *et al.* 2005). However, the molecular mechanisms of action are still unclear. Furthermore, little is known about the effects of CLA on gut cell function beyond the process of Ca transport and epithelial permeability (Roche *et al.* 2001). Therefore, the objective of the present study is to use a transcriptomic approach to help elucidate the molecular mechanisms underlying the CLA-mediated increase in Ca absorption and, in addition, to investigate the effects of CLA on global gene expression profiles in these cells.

Caco-2 cells were treated with 80 μM-linoleic acid (LA; the parent fatty acid) or 80 μM-*t*10, *c*12 CLA for 14 d after initial seeding of the cells. RNA was isolated from the cells, labelled and hybridised to the Affymetrix U133 2.0 Plus arrays (54 645 sequences). Three separate RNA preparations, each from an independent dish of cells, were analysed for each treatment. Selected genes were verified with quantitative reverse-transcriptase PCR (qRT-PCR). Data analysis was preformed using Bioconductor (GCRMA algorithm, regularized *t* test, multiple test correction) and functional analysis was performed using DAVID (gene ontology tool).

Using a minimum change criterion of 1.5-fold, 1940 transcripts (1113 increased and 828 decreased) were differentially expressed (adjusted $P<0.05$) between the LA- and *t*10, *c*12 CLA-treated cells. In relation to transepithelial Ca transport, regulated genes involved in paracellular and transcellular Ca transport are listed in the Table.

	LA		*t*10, *c*12		Fold change		
Gene	Mean	SD	Mean	SD	Increase	Decrease	*P* value
Transcellular transport							
Calbindin 9Dk	229	106	1343	905	5.7	–	<0.001
Paracellular transport							
Claudin 1	913	285	513	69	–	1.7	0.004
Claudin 2	21	12	49	6	3.2	–	<0.001
Claudin 4	555	87	926	135	1.7	–	0.004
Claudin 7	466	58	716	50	1.6	–	0.007

The differentially expressed genes were categorised according to gene ontology, which revealed that the cellular and physiological processes most modified by CLA were cell proliferation, lipid metabolism, steroid metabolism and DNA replication.

The present study shows that the *t*10, *c*12 CLA isomer specifically regulates a number of key genes involved in both transcellular and paracellular Ca transport in Caco-2 cells. These molecular changes help explain the mechanisms by which CLA modulates Ca absorption. In addition, the present study provides evidence of gene-regulatory effects of the *t*10, *c*12 CLA isomer on a number of key processes and pathways, which require further work to understand implications on gut cell function.

This research was part funded by *Safe*food – The Food Safety Promotion Board, Ireland and by the EU-funded European Nutrigenomics Organisation (NuGO) Network of Excellence.

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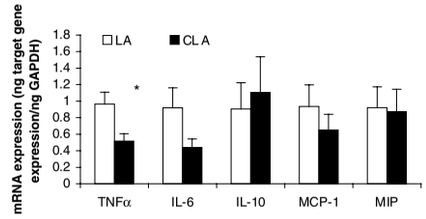
Improvement in insulin sensitivity following a *cis*-9, *trans*-11 conjugated linoleic acid (CLA)-enriched diet is associated with an improvement in inflammatory profile in adipose tissue. By F. MOLONEY, E. NOONE, M.J. GIBNEY and H.M. ROCHE, *Nutrigenomics Research Group, Institute of Molecular Medicine, Trinity Centre for Health Sciences, St James's Hospital, Dublin 8, Republic of Ireland*

Conjugated dieonic derivatives of linoleic acid are a mixture of geometric and positional isomers of linoleic acid (LA). CLA is a natural food component found in the lipid fraction of meat, milk and other dairy products. Our total daily intake of CLA is estimated to be approximately 200 mg/d, with the *cis*-9, *trans*-11 isomer (*c*9, *t*11-CLA) accounting for >90% of the total CLA intake. Evidence has emerged that the *trans*-10, *cis*-12 CLA isomer is pro-diabetic (Risérus *et al.* 2002; Roche *et al.* 2002). However, we have shown evidence that *c*9, *t*11-CLA improves metabolic and molecular markers of insulin sensitivity in adipose tissue and liver (Moloney *et al.* 2004). Feeding a CLA-enriched diet significantly reduced both plasma glucose and insulin concentrations ($P<0.05$). IRS-1 and Glut 4, key targets involved in glucose uptake in adipose tissue, were increased by 1.4-fold and 1.6-fold respectively.

Eleven 12-week-old male ob/ob mice were randomly allocated to receive either a high-fat diet supplemented with *c*9, *t*11-CLA or LA for 5 weeks. Tissue samples were homogenised in lysis buffer to yield the plasma membrane fraction or the membrane and cytosolic fraction. RNA was extracted from white adipose tissue (WAT) and gene expression was investigated using real-time quantitative PCR. Statistical analysis was completed using a pooled *t* test.

Feeding a diet enriched with *c*9, *t*11-CLA increased the expression of the insulin receptor in the membrane and cytosolic fraction by 23%, and the expression of Glut 4 at the plasma membrane by 17% compared with the control diet. In the positive controls, animals treated with thiazolidinedione (pharmacological agent which increases insulin sensitivity), the expression of the insulin receptor increased by 29.5% in the membrane and cytosolic fraction and Glut 4 expression increased by 15% at the plasma membrane. This increased expression of the insulin receptor and Glut 4 is likely to promote glucose uptake and storage in cells, thereby contributing to the lower circulating glucose and insulin concentrations observed in the biochemical assays.

Since inflammatory mediators are now known to significantly impact on insulin sensitivity, we investigated the mRNA expression profile of inflammatory markers in adipose tissue, as shown in the Figure. Values represent group means and SEM.



* Mean value was significantly different from LA ($P<0.05$).

The altered expression profile indicates an attenuation of inflammatory mediators. TNF-α is a key molecular link between obesity and insulin resistance, and represents a potential mechanism for the observed improvement in insulin sensitivity. Several mechanisms have been proposed explaining how TNF-α might induce insulin resistance in adipocytes as well as systemically, including increased serine phosphorylation of IRS-1 and down regulation of protein levels of IRS-1 and Glut 4. Further work should investigate whether the improvement in insulin sensitivity following *c*9, *t*11-CLA is mediated through the decreased expression of TNF-α.

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High-concentration sports drinks aid performance. By J. O'HALLORAN, M. CLEGG, G. BRENNAN, M. FOLEY, B. MURPHY, A. O'SULLIVAN and A. SHAFAT, *Department of Physical Education and Sport Sciences, University of Limerick, Republic of Ireland*

Research has shown that reduced blood glucose concentration may be a limiting factor for performance (Mitchell *et al.* 1989). Increasing carbohydrate concentration leads to reduced gastric emptying rates, compromising glucose delivery (Mitchell *et al.* 1989; Vist & Maughan, 1994). It is a common practice among athletes that sports drinks should be consumed in weaker concentrations than those commercially available. The present study examines these concepts. The purpose of the present study was to investigate the effects of three different carbohydrate sports drink solutions on mouth to caecum transit time (MCTT), concentration of blood glucose and distance achieved in a cycle performance test.

Eight healthy, male volunteers (24.9 (SD 1.3) years, 84.0 (SD 15.3) kg, 177.5 (SD 5.3) cm) consented to take part in the present study, approved by the University of Limerick ethics committee. Three carbohydrate solutions, 6.8, 5.1 and 3.4%, were administered on three separate test days, randomly assigned under double-blind conditions. Participants fasted 12 h overnight before performing a 90 min intermittent cycle on a Monark cycle ergometer at 85%$VO_{2\ max}$ (7 min on; 2 min rest). MCTT was tested every 9 min using a Micro Medical H_2 meter (Ternent *et al.* 2001). MCTT was defined as an increase in breath hydrogen of 3 parts per million for three consecutive readings. Capillary blood samples were taken every 10 min and analysed for blood glucose concentration using an Analox Glucometer. Subsequently, a 10 min maximum effort cycle test was carried out in which the distance travelled was recorded. The blood glucose area under the curve (AUC) was calculated from the baseline level until 90 min. Statistical significance ($P<0.05$) using repeated measures ANOVA was tested with SPSS (version 11.0).

Results indicate MCTT was significantly longer for 6.8% (79 (SD 27) min) ($P=0.048$) and 5.1% (75 (SD 26) min) ($P=0.014$) compared with 3.4% (62 (SD 35) min). AUC increased with successively stronger drink concentrations (see Fig. 1). The 6.8% solution had a significantly greater AUC (800 (SD 68) mmol×min/l) than both the 5.1% (409 (SD 46) mmol×min/l) ($P=0.001$) and the 3.4% (382 (SD 35) mmol×min/l) ($P=0.001$) solution. The greatest performance was found with the 6.8% solution (5.63 (SD 0.53) km), which was significantly greater than 5.1% (5.16 (SD 0.35) km) ($P=0.040$) but not 3.4% (5.30 (SD 0.56) km).

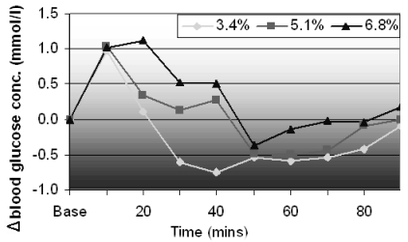

Fig. 1. Blood Glucose Area Under Curve.

To our knowledge, this is the first study to examine MCTT, blood glucose levels, and performance in one testing procedure. Carbohydrate feeding using a 6.8% solution delayed gastrointestinal transit as previously shown in the extant literature (Vist & Maughan, 1994; Murray *et al.* 1999). Yet, the more concentrated solution achieved the most favourable balance of MCTT and blood glucose to aid performance during a medium intensity and duration exercise (for example, soccer, hockey, rugby). Further research is required using concentrations greater than 6.8% to reveal the optimal balance between carbohydrate-electrolyte drink, gastrointestinal transit and performance enhancement. The results imply that the common practice of diluting carbohydrate drinks may be detrimental to performance.

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The consumption of a commercially available sports drink on the effectiveness of intermittent exercise. By S.M. MADIGAN[1], D. GAMBLE[1], C. McCLEAN[1], J. BROWN[1], T. TRINICK[2], E. DULY[2] and G.W. DAVISON[2], [1]*School of Health Sciences, University of Ulster, Belfast, UK and* [2]*Ulster Hospital, Dundonald, Belfast, UK*

It is well established that the ingestion of a carbohydrate–electrolyte solution immediately before, and at frequent intervals during exercise, delays fatigue and improves exercise performance (Tsintzas *et al.* 1993). There is a paucity of research, however, on the efficacy of ingesting a carbohydrate–electrolyte solution immediately before but not during intermittent exercise. Thus the aim of the present study was to examine the effect of ingesting a commercially available carbohydrate–electrolyte solution (sports drink) before exercise performance.

Ten healthy recreational male volunteers (age 20 (SD 2) years; height 178 (SD 7) cm; mass 77 (SD 10) kg; estimated VO_{2max} 56 (SD 3) ml/kg per min) completed the present study, which had local ethics committee approval (University of Ulster). All subjects completed two experimental trials in random order separated by a minimum of 7 d. For 24 h before each experimental trial, subjects standardised their diet and activities. For each trial, subjects consumed (8 ml/kg body mass) a carbohydrate–electrolyte solution (PowerAde, Coca-Cola; 6% carbohydrate, 250 mg Na/500 ml) 15 min before exercise or no fluid. The exercise involved intermittent shuttle (20 m apart) running for 4×15 min blocks. Each block consisted of ten 90 s segments: 3×20 m walking; 1×20 m maximum sprint; 3×20 m jogging; 3×20 m fast running. On completion of the final block, subjects commenced the incremental shuttle running to exhaustion test (Ramsbottom *et al.* 1998). Before and following exercise, measures of body mass and specific gravity of urine were obtained. Pre- and post-exercise blood samples were also obtained from a prominent forearm ante-cubital vein for the determination of glucose, triacylglycerols, cortisol, K and Na concentration. Heart rate was measured throughout exercise using standard telemetry. Post-exercise blood samples were corrected for plasma volume shifts using the equations of Dill & Costill (1974).

When comparing the two exercise trials, subjects had the capacity to exercise for longer when the sports drink was ingested in comparison with the no-fluid trial (exercise time to exhaustion in the carbohydrate trial was 649 (SD 95) s compared with no fluid, 593 (SD 107) s; $P<0.05$). There was a main effect for time for specific gravity of urine ($P<0.05$ *v.* post-exercise; pooled data), body mass ($P<0.05$ *v.* post-exercise; pooled data) and heart rate ($P<0.05$; pooled data).

The main finding from this investigation shows that drinking a carbohydrate–electrolyte solution before exercise improves performance. The present study has practical implications for those sports where drinking during activity is restricted.

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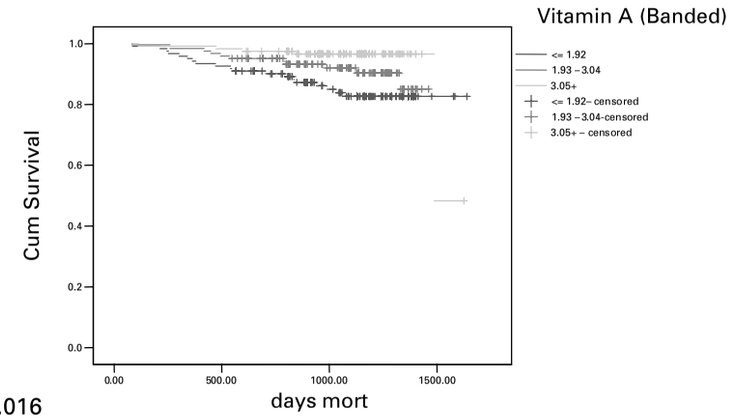
Tsintzas K, Liu R, Williams C, Campbell I & Gaitanos G (1993) *International Journal of Sports Nutrition* **3**, 127–139.

Antioxidant vitamin concentrations and survival in a renal transplant population. By G. CONNOLLY[1], R. CUNNINGHAM[2], J.V. WOODSIDE[1], I.S. YOUNG[1] and A.P. MAXWELL[2], [1]*School of Clinical Medicine, Queen's University Belfast, Belfast, UK, BT12 6BJ and* [2]*Department of Nephrology, Belfast City Hospital, Belfast, UK, BT9 7AB*

CVD is the leading cause of death in patients with chronic kidney disease (Foley & Parfrey, 1998). Exaggerated oxidative stress (Loughrey *et al.* 1994) may play a major role in the 'accelerated' atherosclerosis observed in this population. As antioxidants protect against the toxic effect of excess production of oxidative species, they may have a protective role in the pathogenesis of atherosclerosis. However, although experimental evidence supports these effects for the antioxidant vitamins A, C, and E, results from clinical trials are not consistent (Tribble, 1999).

The aim of the present study was therefore to examine the relationship between survival and concentrations of the antioxidants vitamins A, C and E in a renal transplant population. Three hundred and seventy-nine renal transplant recipients were enrolled in the present prospective cohort study between June 2000 and December 2002. Vitamin A (*n* 365) and vitamin E (*n* 368) were analysed in serum using HPLC. Vitamin E was adjusted for total cholesterol (i.e. vitamin E ratio = vitamin E:total cholesterol). Vitamin C (*n* 182) was analysed in plasma using a fluorometric assay. Participants were followed up for a median of 1080 d and a mean of 1038 d. The concentrations for each vitamin were banded into thirds and Kaplan–Meier analysis with log rank test was used for cumulative survival.

As shown in the Figure, higher vitamin A concentration was associated with lower mortality ($P=0.016$). However, neither vitamin C, vitamin E nor vitamin E ratio was associated with mortality ($P=0.40$, $P=0.70$ and $P=0.28$ respectively).



* $P=0.016$

In univariate analysis in the local renal transplant population, lower vitamin A status was associated with increased mortality. However, there was no significant association between vitamin C, vitamin E nor vitamin E ratio and mortality in the enrolled population.

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Tribble L (1999) *Circulation* **99**, 591–595.

Adhesion molecules and age-related macular degeneration. By R. GRAYDON, J.V. WOODSIDE, I.S. YOUNG and U. CHAKRAVARTHY, *School of Clinical Medicine, Queen's University Belfast, Belfast, UK, BT12 6BJ*

Age-related macular degeneration (AMD) is a leading cause of blindness. It is estimated that between 20 and 25 million individuals worldwide are affected (Chopdar *et al.* 2003). Early-stage disease is characterised by small drusen (acellular debris), which may develop into late stage disease. Local inflammation has been shown to play a role in drusen formation. Indeed, altered levels of immunoglobulins, caeruloplasmin and fibrinogen have been noted in AMD subjects (Penfold *et al.* 2001). Also, a recent study found that elevated levels of an inflammatory biomarker, C-reactive protein, was an independent risk factor for AMD (Seddon *et al.* 2004). Cell adhesion molecules are involved in protecting the body against infection and are well-known markers of inflammation. Intracellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1) have both been shown to be elevated in various inflammation-associated diseases. It is not known, however, if ICAM-1 and VCAM-1 levels are altered in AMD patients.

The present study was a case–control study in 226 AMD cases and 208 healthy controls. Non-fasting blood samples were collected. Details of CHD history and smoking status were collected and body weight and blood pressure (BP) were measured. Serum samples were analysed for ICAM-1 and VCAM-1 (ELISA kits obtained from Immunodiagnostic Systems Ltd, Boldon, Tyne and Wear, UK).

The Table shows a comparison of cases and controls.

	Cases		Controls		
	Mean	SD	Mean	SD	*P* value
Age (years)	76.6	7.3	73.3	5.8	<0.001
Weight (kg)	72.4	17.0	73.6	12.7	NS
Systolic BP (mmHg)	152.8	22.5	144.3	19.7	<0.001
Diastolic BP (mmHg)	79.9	11.7	81.1	14.4	NS
ICAM-1 (ng/ml)	711.2	209	892.2	302	<0.001
VCAM-1 (ng/ml)	1439	490	1716	873	<0.001

AMD cases were more likely to smoke than controls (21 *v.* 15%; $P<0.05$). There was no effect of smoking on ICAM-1 or VCAM-1, but VCAM-1 was positively associated with age (r 0.24; $P<0.001$) and ICAM-1 with diastolic BP (r 0.18; $P=0.001$).

The present study has shown that ICAM-1 and VCAM-1 are significantly lower in AMD cases than controls, which is unexpected. The role of inflammation in AMD pathogenesis therefore requires further elucidation.

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Frequency of fruit and vegetable consumption and carotenoid status in the EUREYE study. By S.E.C.M. GILCHRIST, J.V. WOODSIDE, U. CHAKRAVARTHY and I.S. YOUNG, on behalf of the EUREYE investigators, *School of Clinical Medicine, Queen's University Belfast, Belfast, UK, BT12 6BJ*

It is widely accepted that fruit and vegetables are healthy foods and the consumption of five portions per d is recommended by both national and international authorities. However, the active components in fruit and vegetables responsible for their health-promoting effects have not been conclusively determined. Interest has focused on the carotenoids, primarily because of their antioxidant activity. Fruit and vegetables are rich in carotenoids, but little information is available on the fruits and vegetables contributing most to carotenoid status in the elderly population, who are most at risk of eye disease.

The EUREYE study is a multi-centre, population-based, cross-sectional study aimed at evaluating the prevalence of age-related macular degeneration (AMD) in elderly European populations, and to investigate risk factors for AMD. Belfast was one of the centres for the EUREYE study, and 684 subjects aged >65 years were recruited from this location. Subjects completed lifestyle questionnaires, including a detailed food-frequency questionnaire which included questions on forty-eight different fruits and vegetables, and a non-fasting blood sample was collected. Concentrations of lutein, zeaxanthin, β-cryptoxanthin, α-carotene, β-carotene and lycopene were assessed in serum by HPLC according to Craft *et al.* (1992).

Frequency of fruit and vegetable consumption was compared with carotenoid status using Pearson correlation coefficients. The Table shows the fruit and vegetables correlating most strongly with the carotenoids measured. Correlation coefficients are statistically significant, although low.

Carotenoid ...	Lutein		Zeaxanthin		β-Cryptoxanthin		α-Carotene		β-Carotene		Lycopene	
Food item	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*
Beetroot	0.20	<0.001	0.20	<0.001								
Apples	0.18	<0.001	0.19	<0.001	0.20	<0.001						
Marrow	0.16	<0.001									0.10	0.001
Broccoli	0.14	0.001	0.14	0.001			0.13	0.001	0.09	0.04		
Cauliflower	0.13	0.002					0.13	0.001	0.11	0.01		
Sweetcorn			0.17	0.001								
Cabbage			0.13	0.001								
Oranges					0.20	<0.001						
Clementines					0.16	<0.001			−0.11	0.008	−0.107	0.009
Melon					0.15	<0.001	0.13	0.001				
Kiwi					0.15	<0.001						
Carrots							0.17	<0.001	0.08	0.05		
Peppers*							0.14	0.001				
Coleslaw									0.11	0.005	0.10	0.012
Parsnips											−0.09	0.02
Pears											−0.10	0.01

* Red and orange peppers.

The present study shows that carotenoid status in the elderly is associated with frequency of fruit and vegetable consumption.

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B vitamin and antioxidant supplementation has no effect on C-reactive protein concentration in healthy male volunteers. By G.C. McKEEMAN, J.V. WOODSIDE, J.W.G. YARNELL, I.S. YOUNG and A. EVANS, *School of Clinical Medicine, Queen's University Belfast, Belfast, UK, BT12 6BJ*

Vitamin supplementation has been associated with a reduced risk of CVD. B vitamin supplementation has been proposed to reduce cardiovascular risk through lowering homocysteine concentrations, while antioxidant vitamins have been proposed to reduce cardiovascular risk through reduction in the susceptibility of LDL to oxidation (Woodside *et al*. 1998; Young & Woodside, 2001). C-reactive protein (CRP) is an inflammatory marker which has been shown to be a marker of CVD (Blake & Ridker, 2002). The effect of vitamin supplementation on CRP levels in healthy subjects has yet to be determined. The aim of the present pilot study was to assess the effect of B group and antioxidant vitamin supplementation, either singly or in combination, on CRP concentrations.

Healthy male subjects (30–49 years; *n* 765) were screened for plasma homocysteine concentration. Those with elevated homocysteine (>8.34 μmol/l; *n* 132) in the screening study were randomised to one of four possible micronutrient treatments using a factorial design; either B vitamins alone (1 mg folic acid, 7.2 mg pyridoxine, 0.02 mg cyanocobalamin daily), antioxidants alone (150 mg ascorbic acid, 67 mg α-tocopherol, 9 mg β-carotene daily), B vitamins with antioxidant vitamins, or placebo for an 8-week period. CRP concentrations were assessed in a random sample of the intervention subjects (*n* 45) by an automated immunoturbidimetric assay.

CRP concentration at baseline was significantly associated with vitamin B_{12} concentration (*r* 0.3; $P<0.05$) and antibody to LDL oxidation titre (*r* 0.39; $P<0.01$). There were no differences between the groups in baseline vitamin status. The change in CRP concentration was compared between the four intervention groups by a non-parametric Kruskal–Wallis test. There was no significant difference in change in CRP (CRP concentration week 8 − CRP concentration week 0) between the four intervention groups (see Table).

The Table shows the median change in CRP concentrations (mg/l) in each of the four intervention groups.

Placebo (*n* 11)		B vitamins (*n* 11)		Antioxidants (*n* 10)		Antioxidants and B vitamins (*n* 13)	
Median	IQ range	Median	IQ range	Median	IQ range	Median	IQ range
−0.10	−0.40, 0.60	−0.20	−0.80, 0.00	0.10	−0.03, 0.43	0.20	−0.15, 1.60

IQ, interquartile range.
All changes calculated as week 8 – week 0 were NS ($P=0.08$).

The present study shows that B vitamin and antioxidant vitamin supplementation over 8 weeks do not affect CRP concentration in healthy male subjects. These findings could be related to the short study duration, small sample numbers and the fact that the B vitamin and antioxidant supplements contained more than one vitamin type. Larger, well-designed studies using individual micronutrients are required to determine if micronutrient supplementation has a significant effect on CRP status.

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The effect of folate, pyridoxine and riboflavin depletion on plasma homocysteine. By K.M. MOONEY, I.S. YOUNG and G.J. CUSKELLY, *Nutrition and Metabolism Research Group, Centre for Clinical and Population Sciences, Queen's University Belfast, Grosvenor Road, Belfast, UK, BT12 6BJ*

Homocysteine (hcy) is an independent risk factor for CVD. Four B vitamins are required for hcy metabolism; folate, vitamin B_{12}, pyridoxine and riboflavin. Numerous studies have assessed the relationship between these four B vitamins and hcy, but none have examined the independent roles of depletion of each B vitamin alone. Therefore, the relative effects of low levels of the four B vitamins on hcy are unknown. The aim of the present study was to investigate the impact of marginal deficiency of folate or pyridoxine or riboflavin on hcy while controlling for intakes of all other B vitamins.

Low-folate (0.55 μg/d), low-pyridoxine (0.36 mg/d) and low-riboflavin (0.55 mg/d) diets were designed (7 d cycles). Volunteers were randomised to one of the three depletion diet groups; low folate (6 weeks; *n* 5), low pyridoxine (4 weeks; *n* 5), low riboflavin (6 weeks; *n* 6) or control group (6 weeks; *n* 6). These periods of depletion are required to induce marginal deficiency of respective B vitamins. Those in the control group consumed their normal diet and a multivitamin containing all vitamins and minerals. Volunteers on each of the low B vitamin diets consumed a deplete diet and in addition a multivitamin and multimineral tablet (devoid of the respective B vitamin). In order to optimise compliance, all meals and snacks were prepared and given to volunteers on weekdays. Supplies of food were weighed, packaged and given to volunteers on Friday for the weekend period. Responses to depletion were monitored by assessing changes in plasma or serum B vitamin status, in the respective depletion regimens (one-way ANOVA and Duncan *post hoc* test). Plasma hcy was measured weekly using HPLC (see Table). Changes in hcy (calculated as the slope of the intervention time line) were compared between groups.

Responses in plasma hcy of the low folate group were not significantly different from the control group. Neither were hcy responses of the riboflavin group significantly different to any of the groups. The low pyridoxine group responses were significantly different from both the low folate and control groups ($P=0.019$). The Table shows plasma tHcy concentrations per group (μmol/l).

	Folate group		Pyridoxine group		Riboflavin group		Control group	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Baseline	10.2	2.3	9.4	1.7	10.4	1.4	10.3	0.7
End of week 1	9.4	2.1	5.0	1.6	10.4	1.2	7.2	2.0
End of week 2	9.0	2.3	5.4	1.4	9.2	1.9	9.4	1.5
End of week 3	10.6	2.6	5.4	1.4	9.9	1.1	9.1	1.1
End of week 4	14.6	2.6	6.8	1.3	10.8	1.6	10.3	2.2
End of week 5	14.4	2.5			10.3	1.8	10.1	1.0
End of week 6	14.7	2.0			11.4	1.0	10.4	1.1

Although the folate and riboflavin depletions induced an elevation in hcy concentrations, responses were not significantly different from the control group. The pyridoxine-deplete group was the only group found to be significantly different from the control group.

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Effect of γ-tocopherol supplementation on γ-tocopherol status in healthy adults. By J.M.W. WALLACE[1], E. KEAVENEY[2], P.J. ROBSON[1], A.J. SINCLAIR[3] and M. KIELY[2], [1]*Northern Ireland Centre for Food and Health, University of Ulster, Coleraine, UK, BT52 1SA,* [2]*Department of Food and Nutritional Sciences, University College Cork, Republic of Ireland and* [3]*Department of Food Science, Royal Melbourne Institute of Technology University, Melbourne, Victoria, Australia*

Epidemiological studies have associated higher intakes of vitamin E with reduced risks for CHD (Rimm *et al.* 1993). However, intervention studies with α-tocopherol (α-T), the main vitamin E isomer in plasma, have shown little benefit (Knekt *et al.* 2004). Evidence is now emerging to suggest that γ-tocopherol (γ-T), the second most predominant vitamin E isomer in plasma, may have important physiological effects that complement the functions of α-T (Jiang & Ames, 2003; Liu *et al.* 2003). To date, only one study, using a mixed-tocopherol supplement, has evaluated the effects of γ-T *per se* in human subjects (Liu *et al.* 2003). The present study examined the impact of supplementation with γ-T on α- and γ-T status in apparently healthy adults.

Men (*n* 15) and women (*n* 15), aged 20–36 years, were randomised to receive placebo, or 100 or 200 mg γ-T/d contained in a mixed tocopherol preparation (active capsules contained 62% γ-T, 26% δ-T and 13% α-T; Cognis Nutrition and Health, Australia) for a 4-week period. Blood samples were collected at baseline, and at week 2 and week 4. Tocopherol concentrations in plasma and platelet-rich plasma (PRP) were determined by HPLC.

There were no differences between the groups at baseline and no sex differences were observed. Supplementation with 100 or 200 mg γ-T increased plasma and PRP γ-T concentrations ($P<0.001$), but had no significant effect on α-T concentration. Both plasma and PRP α-T:γ-T ratios decreased following supplementation ($P<0.001$). There was no significant difference between supplementation with 100 and 200 mg of γ-T on tocopherol status.

	Placebo (*n* 10)						100 mg γ-tocopherol (*n* 10)						200 mg γ-tocopherol (*n* 10)					
	Baseline		Week 2		Week 4		Baseline		Week 2		Week 4		Baseline		Week 2		Week 4	
μmol/l	Mn	SD	Mn	SD	Mn	SD	Mn	SD	Mn	SD	Mn	SD	Mn	SD	Mn	SD	Mn	SD
Plasma γ-T	0.8	0.5	0.9	0.3	0.9	0.4	1.1[a]	1.0	5.7[b]	3.1	3.5[b]	2.1	0.8[a]	0.4	5.7[b]	3.6	4.2[b]	1.9
PRP γ-T	1.2	0.3	1.2	0.4	1.4	0.9	1.3[a]	0.3	7.3[b]	3.8	4.2[b]	1.6	1.2[a]	0.4	7.2[b]	4.1	6.4[b]	3.3
Plasma α-T	12.5	8	17.3	8	12.2	3	16.2	10	20.4	7	18.4	7	15.5	6	17.4	4	12.5	4
PRP α-T	15.3	2	17.9	4	19.7	9	19.4	5	26.3	10	20.4	3	18.0	4	20.6	3	20.9	3
Plasma α:γ-T	17.1	3	18.4	3	14.1	3	17.2[a]	5	4.3[b]	1.7	6.2[b]	2.8	23.8[a]	11	4.4[b]	2.9	3.5[b]	1.8
PRP α:γ-T	12.8	1	16.2	2	16.3	6	15.0[a]	4	4.0[b]	1.3	5.6[b]	2.6	17.0[a]	8.9	3.9[b]	2.5	3.9[b]	1.8

Mn, mean. Data were analysed using repeated measures ANOVA ($P<0.05$).
[a,b] Mean values within a row with unlike superscript letters are significantly different.

The results of the present study show that γ-T, when administered at a relatively high dose in the short term, is expressed in plasma and PRP. Furthermore, a plateau in γ-T concentration in plasma and PRP was observed following administration of 100 mg γ-T, with no additional effect observed in individuals who received 200 mg of γ-T. The effects of lower dose and longer-term supplementation with γ-T on tocopherol status requires further investigation.

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The effect of the high- and low-glycaemic index diets on urinary chromium in healthy individuals (a cross-over study). By M. HAJI FARAJI[1] and A.R. LEEDS[2], [1]*National Nutrition and Food Technology Research Institute of I. R. Iran and* [2]*Department of Nutrition and Dietetics, King's College London, Franklin-Wilkins Building, 150 Stamford Street, London, UK, SE1 9NH*

Cr is an essential nutrient required for glucose and fat metabolism. Insufficient dietary intake of Cr leads to signs and symptoms that are similar to those observed for diabetes and CVD (Anderson, 1998). A low-glycaemic index (GI) diet can reduce postprandial glucose levels and help keep blood glucose levels within a near-normal range, and may increase insulin sensitivity (Frost *et al.* 1996). It is possible that persistent consumption of high-GI meals causes a gradual depletion of body-tissue Cr concentrations.

We postulate that in healthy individuals, urine Cr excretion following a high-GI diet is higher than after a low-GI diet. The effect of consuming low- and high-GI diets on urine Cr excretion over 6 d in a cross-over study in sixteen healthy subjects (aged 18–60 years old; nine males and seven females) was investigated. Every second day within each treatment fasting blood glucose and insulin (by Stat Analyser and Radioimmunoassay) were used to determine insulin resistance index (Matthews *et al.* 1985), and 24 h urine samples were collected to measure Cr excretion. Dynamic Reaction Cell Inductively Coupled Plasma-MS was used to measure Cr.

The GI of low- and high-GI meals was estimated to be 44.8 and 78.9 respectively. During the 6 d diet study there were no significant differences of 24 h urine Cr losses between the two groups following the low-GI (0.58 (SE 0.08) μg/24 h) and high-GI diets (0.48 (SE 0.06) μg/24 h). However, during day six there was a non-significant trend towards greater loss (Fig. 1) of Cr after the high-GI diet (0.73 (SE 0.1) μg/24 h) in comparison with the low-GI diet (0.54 (SE 0.07) μg/24 h). Evidence that urine Cr losses after an oral glucose ingestion relates to postprandial blood glucose and insulin has not been clearly demonstrated in normal subjects, nor was there evidence for an effect of dietary GI on urinary Cr excretion. However a significant correlation ($r=0.67$) was found between Cr excretion and insulin resistance only in high-GI diet group ($P<0.05$).

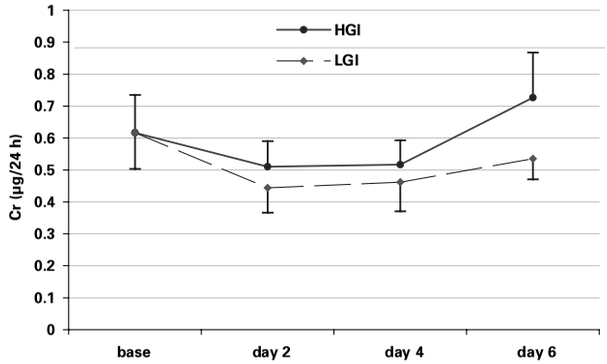

Fig. 1. 24 h Urine Cr excretion during 6 d low- and high-GI diets in sixteen normal subjects. Values are expressed as means and standard errors.

These results suggest that Cr excretion may need to be observed for longer than 6 d to address this question.

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Analysis of the relationship between frequency of consumption of non-milk extrinsic sugar meals and percentage total energy from non-milk extrinsic sugars in a representative sample of Irish adults. By T. JOYCE, A.P. HEARTY, S.N. McCARTHY and M.J. GIBNEY, *Department of Clinical Medicine, Trinity Centre for Health Science, St. James's Hospital, Dublin 8, Republic of Ireland*

The Committee on Medical Aspects of Food Policy (Department of Health, 1989) defines non-milk extrinsic sugars (NMES) as sugars not located within the cellular structure of a food and includes fruit juices and honey and 'added sugars' which comprise recipe and table sugars. Numerous studies have shown an association between the amount and frequency of free sugars intake and dental caries. Moynihan & Petersen (2004) reported that when free sugar consumption is <15–20 kg/year (40–55 g/d), which relates to 6–10% of energy intake, the incidence of dental caries is low. In addition, it was highlighted that the frequency of consumption of foods containing free sugars should be limited to a maximum of four times per d. In the UK, the recommendation is to keep NMES intake <60 g/d (<11% food energy; <10% total energy) (Department of Health, 1991). Extensive literature searches failed to provide any data to support the quantitative association between 10% energy from NMES intake and frequency (four times per day) of NMES intake. The present study therefore set out to explore this quantitative relationship in Irish adults. Analysis was based on the North/South Ireland Food Consumption Survey which established a database of habitual food and drink consumption in Irish adults aged 18–64 years (*n* 1379). The breakdown of the different sugars was calculated based on the total amount of sugar in the food and the type of food. The analysis included subjects who had up to nine eating occasions per d and excluded under-reporters with an EI:BMR of <1.05.

	Quartiles of NMES eating occasions per d															
	Quartile 1 (*n* 220)				Quartile 2 (*n* 238)				Quartile 3 (*n* 270)				Quartile 4 (*n* 222)			
	Mn	SD	Min	Max	Mn	SD	Min	Max	Mn	SD	Min	Max	Mn	SD	Min	Max
NMES eating occasions	2.9[a]	0.4	1.1	3.3	3.7[b]	0.2	0.2	3.9	4.3[c]	0.2	4.0	4.6	5.3[d]	0.5	4.7	7.7
Total energy (MJ/d)	9.0[a]	2.4	5.7	21.0	9.5[a]	2.5	2.5	18.2	10.2[b]	2.7	5.7	23.5	11.2[c]	3.1	6.3	24.2
NMES (g/d)	40.6[a]	22.6	1.7	121.8	53.2[b]	29.7	9.8	165.0	64.3[c]	34.0	13.6	278.9	84.0[d]	40.4	16.5	286.7
% TE NMES	7.1[a]	3.6	0.5	18.3	8.6[b]	3.6	1.4	20.5	9.8[c]	4.0	2.1	27.0	11.7[d]	3.9	3.6	24.7
% TE carbohydrate	42.9[a]	6.5	25.3	70.9	44.2[ab]	6.0	28.7	58.3	44.8[b]	5.9	26.6	65.0	45.7[b]	5.7	26.2	59.0
% TE total sugar	14.8[a]	4.1	6.0	26.6	16.3[b]	4.6	5.1	29.4	17.5[c]	4.6	6.7	31.0	18.6[c]	4.3	7.5	29.8
% TE intrinsic sugar	5.1[a]	3.5	0.6	22.4	4.8[a]	3.1	0.1	16.5	4.8[a]	2.9	0.5	15.3	4.1[b]	2.3	0.5	13.4
% TE milk sugar	2.5[a]	1.5	0.0	7.9	2.9	1.5	0.1	9.4	3.0[b]	1.5	0.0	7.3	2.8	1.3	0.1	6.7

Mn, mean; TE, total energy.
[a,b,c,d] Mean values within a row with unlike superscript letters were significantly different ($P<0.05$).

Results of the analysis showed that as the frequency of NMES eating occasions increased, there was a significant increase in percentage total energy from total and NMES and a significant decrease in percentage total energy from intrinsic sugar. Percentage total energy from NMES went above the UK recommendation of 10% total energy from NMES at approximately 4.5 extrinsic sugar meals per d. There was a significant positive correlation between NMES eating occasions and percentage energy from NMES (r 0.410, n 950; $P<0.01$). When a similar analysis was carried out based on quartiles of total sugar-eating occasions as opposed to NMES, no association was found. This is the first study to have presented data on the quantitative association between the recommended level of <10% total energy from NMES intake and frequency of NMES eating occasions per d.

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Alcohol consumption in pregnant Irish women. By C.M. MURRIN[1,3], G. BURY[1], S. DALY[1], U. FALLON[1], F. HANNON[2], B.G. LOFTUS[2], J. MORRISON[2], A. MURPHY[2], G. NOLAN[3], D. O'MAHONY[1] and C.C. KELLEHER[1,3], [1]*Health Research Board Unit for Health Status and Health Gain, University College Dublin, Republic of Ireland,* [2]*National University of Ireland, Galway, Republic of Ireland and* [3]*National Nutrition Surveillance Centre, University College Dublin, Republic of Ireland*

Consumption of alcohol during pregnancy adversely affects fetal development and pregnancy outcomes. Heavy drinking in early pregnancy in particular leads to fetal alcohol syndrome. The influence of moderate drinking is more controversial. The Health Promotion Unit advises, to 'drink as little as possible or avoid alcohol altogether during pregnancy' (Health Promotion Unit, 2003). The Royal College of Obstetricians and Gynaecologists recommends that women should limit alcohol consumption to 'no more than one standard drink per day' (Royal College of Obstetricians and Gynaecologists, 1999).

The aim of this analysis was to examine the socio-demographic and lifestyle factors which may influence the change in drinking patterns of women who become pregnant. The Lifeways study is a longitudinal population-based cross-generational cohort study comprising of 1120 mothers recruited during their first antenatal visit between October 2001 and January 2003, in Galway and Dublin. Mothers were asked to complete a questionnaire on a variety of socio-demographic, health and lifestyle factors including alcohol consumption.

While 20.5% of women did not normally drink alcohol, 77.7% changed their drinking patterns since becoming pregnant and 1.8% made no change. With increasing age, women were more likely to drink less rather than stop; 30.4 (SD 5.8) *v.* 28.6 (SD 5.8) years; difference in mean age 1.8 (95% CI 0.99, 2.6). As indicated in the table a strong trend across age categories was demonstrated ($P=0.001$).

	Age group			
Change in drinking pattern while pregnant	≤25 (%)	26 to 30 (%)	31 to 34 (%)	≥35 (%)
Drank less	39.1	40.9	49.5	62.4
Stopped drinking	60.9	59.1	50.5	37.6

Those who reduced alcohol consumption had a higher prevalence of 3rd level education relative to those who stopped drinking; 56.7 *v.* 50.4% ($P=0.03$). There was no significant difference in drinking patterns between women who lived in the east of the country compared with the west. Marital status, medical card status, smoking, change in smoking habit and folic acid consumption were not associated with stopping or reducing. There was significant difference in baseline alcohol consumption (number of drinks consumed per drinking session) between reducers and stoppers in the 12-month period before pregnancy; median number of drinks 4.0 in stoppers and in reducers (Stoppers $Z=-2.448$; $P=0.014$). Women who decided to drink less during their pregnancy consumed wine more frequently than other types of alcohol. Twenty percent consumed wine twice or more per week while 11.1% consumed beer or lager, or cider; 1.6% consumed spirits; 0.8% consumed either port, sherry or liqueurs twice or more per week. Women who breast-fed their last child were more likely to reduce alcohol consumption (62.7%) than women who did not breast-feed (50.6%).

Despite similar pre-pregnancy baseline consumption of alcohol in the present study, some women stopped drinking alcohol when pregnant whereas others women reduced the amount they consumed. Reducers were slightly older and better educated, but other major socio-demographic and lifestyle factors did not differ between the two groups. The variation in drinking patterns among women during pregnancy may be as a result of vague guidelines on alcohol consumption during pregnancy. Further study is required to profile these women and enhance our understanding of why and how they make these decisions.

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