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A minute on the lips, forever on the waist! Does food intake affect our waistlines? By S.N. McCARTHY[1], P.J. ROBSON[2], M.B.E. LIVINGSTONE[2], M. KIELY[3], A. FLYNN[3] G.W. CRAN[4] and M.J GIBNEY[1], *Irish Universities Nutrition Alliance at* [1]*Department of Clinical Medicine, Trinity College Dublin, Ireland,* [2]*Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, BT52 1SA,* [3]*Department of Food and Nutritional Sciences, University College Cork, Ireland and Department of Epidemiology & Public Health, Mulhouse Building, Grosvenor Road Belfast BT12 6BA.*

In the North/South Ireland Food Consumption Survey, 24% of the adult Irish population were found to have a waist circumference at or above action level 2 (men ≥102 cm, women ≥88 cm) (McCarthy et *al.* 2001). These adults have a high risk for many cardiovascular disease (CVD) risk factors, such as high blood pressure, high LDL cholesterol and low HDL cholesterol (Han *et al.* 1995).

There has been very little research examining the association between food intake *per se* and waist circumference. The aim of this study was to examine the predictive influence of food intake on waist action levels. Food intake was measured using a 7 d food diary. Each food consumed was assigned to one of twenty-eight food groups. A mean daily intake database was generated for the twenty-eight food groups. Subjects who reported themselves to be dieting or unwell were excluded from the analyses, leaving a final sample of n=1150 (588 men, 562 women). Multivariate binary logistic regression was used to determine the predictive influence of the mean daily intake of twenty-eight different food groups on waist action level, controlling for age, sex, education level and energy misreporting. The following table illustrates the odds ratios for waist action level 2 compared to below action level for a median intake (g/d) of each food group.

		Below action level *v.* action level 2			
	Median	*P*	Odds ratio	95% Confidence intervals	
	(g/d)			Lower	Upper
All breads & rolls	127.0	0.000	2.84	1.71	4.70
Fresh meat	71.1	0.000	2.20	1.52	3.18
Meat dishes	72.6	0.000	1.90	1.46	2.48
Chips & processed potatoes	62.9	0.000	1.84	1.39	2.44
Low/high-calorie beverages*		0.042	1.74	1.02	2.95
Creams, ice creams & desserts	32.9	0.000	1.70	1.36	2.12
Sugars & confectionery	29.0	0.000	1.69	1.31	2.20
Butter & spreads	17.4	0.001	1.69	1.22	2.32
Meat products	37.1	0.000	1.65	1.30	2.09
Whole milk	182.3	0.005	1.45	1.12	1.88
Alcoholic beverages	296.9	0.000	1.45	1.26	1.67
Potatoes: boiled, mashed etc.	125.7	0.001	1.39	1.14	1.70
Savoury snacks	8.6	0.025	1.31	1.03	1.67
Egg & egg dishes	17.9	0.026	1.24	1.03	1.49

Below waist action level (men <94 cm, women <80 cm), Action level 2 (men ≥102 cm, women ≥88 cm).

Model is controlled for EI:BMR, age, gender and education.

*Reference category for low/high-calorie beverages = low-calorie beverages.

Of the twenty-eight food groups examined, increased consumption of the fourteen listed above were found to be significantly associated with an increased likelihood of being designated as waist action level 2 compared with a waist circumference below action level (all P<0.05). Consumption of high-calorie (≥209kJ) compared with low-calorie (<209kJ) beverages also has an increased likelihood for action level 2. In other words, higher consumption of each of these food groups is associated with an increased waist circumference, and hence a higher risk of having one or more CVD risk factors.

Waist circumference in adults is strongly influenced by the amount of food consumed. Public health policies for a reduction in centrally distributed body fat may be more effective and informative if the emphasis is placed on a reduction in food and beverages consumed rather than on macronutrients.

This project was funded by the Food Safety Promotion Board.

Han TS, van Leer EM, Seidell JC & Lean MJE (1995) *British Medical Journal* **311**, 1401–1405.
McCarthy S, Harrington KE, Kiely M, *et al*. (2001) *Public Health Nutrition* **4**, 1099–1106.

Food and nutrient intakes across distributions of meat consumption in Irish adults. By M. COSGROVE, A. FLYNN and M. KIELY, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Dietary inadequacies in the Irish population have recently been highlighted. A high proportion of adults exceed recommended fat intakes and there is a high prevalence of inadequate intakes of vitamins A and D, riboflavin, iron, copper and zinc, especially in women. Meat and meat products are the primary dietary sources of protein, fat, vitamin D, and zinc and are an important source of iron, riboflavin, copper and vitamin A (Hannon *et al*. 2001; Harrington *et al*. 2001; O'Brien *et al*. 2001). The aim of the current analysis was to examine intakes of the meat and meat products subgroups in relation to food and nutrient intakes in Irish adults.

We classified men and women into tertiles of low, medium and high consumers of *red meat* (beef (excluding mince), lamb and pork), *poultry* (chicken and turkey, primarily chicken) and *processed meat* (burgers, sausages and minced beef) using disaggregated meat intake data from the North/South Ireland Food Consumption Survey (IUNA, 2001). Differences in nutrient and food intakes were compared using analysis of variance, and associations between meat intakes, compliance with dietary recommendations and adequacy of micronutrient intakes were examined using Pearson's χ^2.

A higher total meat intake was associated with higher intakes (P<0.05) of protein, fat, zinc and niacin and lower intakes (P<0.05) of carbohydrate, dietary fibre, calcium, magnesium, vitamin D (women) and folate (women). There were similar associations (P<0.001) between red meat and nutrient intakes, except for fat, dietary fibre (women), magnesium, vitamin D and folate. A higher poultry consumption was associated (P<0.01) with higher intakes of protein, magnesium (women) and niacin and lower intakes (P<0.05) of dietary fibre (men) and vitamin B_{12} (men). A higher processed meat consumption was consistently associated (P<0.05) with a lower nutrient density, particularly of protein, carbohydrate, dietary fibre, iron (men), vitamin D, thiamin, riboflavin (women) and folate. The exception was fat, which increased (P<0.001) with increasing processed meat intake.

Meat intakes were also examined in relation to compliance with dietary recommendations and adequacy of micronutrient intakes. Higher total meat consumption was associated with less compliance with fat and carbohydrate recommendations and increased compliance with fibre recommendations. A higher processed meat consumption was associated with a lower compliance with dietary recommendations for fat, carbohydrate and fibre. Higher total meat consumption was associated with better adequacy of micronutrient intakes. In women, non-consumers of processed meat were more likely than consumers to have adequate iron intakes.

With greater total meat intakes there was a higher (P<0.05) in the intakes of white bread, butter, potatoes, chips, vegetables (men), alcoholic beverages (men) and carbonated beverages (women) and a lower intake of fish. There were similar associations (P<0.001) between red meat intakes and potatoes, chips (men), vegetables (men), alcoholic beverages (men) and carbonated beverages (women). With an increase in poultry there was a significant decrease (P<0.001) in potato intakes in men and a significant increase (P<0.05) in vegetable intakes in women. Individuals with high intakes of processed meats had significantly higher (P<0.05) intakes of white bread and rolls, carbonated drinks, chips (men) and significantly lower intake of vegetables (men), fruit and fish (men).

High meat consumption is associated with reduced compliance with dietary recommendations for fat and carbohydrate, and better adequacy of micronutrient intakes. However, the current results emphasise that not all meats make a similar contribution to the diet. Processed meat consumption at high levels is related to poor compliance with dietary recommendations and a lower nutrient density, which reflects the dietary choices observed.

Hannon E, Kiely M, Harrington KE, Robson PJ, Strain JJ & Flynn A (2001) *Public Health Nutrition* **4**(5A), 1081–1088.
Harrington KE, McGowan MJ, Kiely M, Robson PJ, Livingstone MBE, Morrissey PA & Gibney MJ (2001) *Public Health Nutrition* **4**(5A), 1051–1060.
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O'Brien MM, Kiely M, Harrington KE, Robson PJ, Strain JJ & Flynn A (2001) *Public Health Nutrition* **4**(5A), 1069–1079.

Food and healthy eating during working hours: attitudes and behaviours of Western Health Board Staff. By J. HARRINGTON[1], A.M. LYDON[2] and D. EVANS[1], [1]*Department of Public Health, Western Health Board, Merlin Park Hospital, Galway, Ireland and* [2]*Community Nutrition and Dietetic Service, Western Health Board, West City Centre, Seamus Quirke Road, Galway, Ireland*

A healthy diet is a key factor in helping to reduce chronic disease (Nutrition Advisory Group, 1995). In 1998 the Department of Health and Children recommended that measures be put in place to ensure that workers both become aware of the benefits arising from healthy lifestyle choices and are helped to incorporate these into their working lives (National Consultative Committee on Health Promotion, 1998). The workplace has been shown by many investigators to be an effective setting for delivering nutrition-based interventions (Hope & Kelleher, 1999). However, to be effective, attitudes and current behaviours need to be assessed to identify the issues that policy-makers need to address. This study aimed to assess the attitudes and behaviours of Western Health Board staff regarding food and eating habits during working hours. The results will inform the development of a Food and Healthy Eating Policy for staff of the Western Health Board. Its key goal will be to provide a supportive and sustainable working environment, which will encourage staff to choose the healthy eating option.

A self-administered postal questionnaire was sent to 2130 staff across all disciplines that included staff from management/administration; medical/dental; nursing; allied health professionals; support services; maintenance/technical. The sample was selected randomly and represented 20% of each discipline. Overall there was a 53% response rate. The questionnaire was based upon the nutrition components of two existing surveys (National Consultative Committee on Health Promotion, 1998; Friel & Kelleher, 1999). χ^2 tests, *t* tests, and Mann-Whitney tests were conducted to analyse the data.

Fifty two percent of respondents were satisfied with their current eating habits. However, 71% recognised that what they ate could be healthier. Many respondents reported being on some sort of diet, with the most commonly reported diet being weight reducing/management. Fifteen percent of respondents reported that what they ate at work was unhealthy, of which, significantly more worked in nursing compared to other occupations. Those with unhealthy eating habits at work also tended to work more unsociable hours. Significantly more males reported eating a full lunch at work ($P<0.05$), whilst significantly more females reported to eating a light lunch ($P<0.05$). In relation to fried food consumption at work, significantly more males compared to females consumed fried food on a regular basis ($P<0.05$). In addition, significantly more respondents who regularly consumed fried food, recognised what they ate could be healthier ($P<0.01$). Time pressure, lack of facilities and lack of healthy eating choices were the main factors, which influenced staff eating behaviours during working hours.

Results indicate that policy-makers need to develop a number of initiatives to address staff eating habits and to effect a behaviour change. Initiatives identified by staff included access to healthy eating information, healthy eating choices in the canteen, and healthier food products in vending machines.

Friel S, Nic Gabhainn S & Kelleher C (1999) *Survey of Lifestyles, Attitudes and Nutrition*. Galway: Ireland: Centre for Health Promotion Studies, National University of Ireland.

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Attitudes towards perceived diets and actual dietary behaviour among Irish adults. By A.P. HEARTY, S.N. McCARTHY, J.M. KEARNEY and M.J. GIBNEY, *Department of Clinical Medicine, Trinity College Dublin, Ireland*

Research indicates that a discrepancy still exists between dietary recommendations and actual food consumption (Harrington *et al*. 2001). This lag in accomplishment of dietary guidelines, in the face of degenerative diseases, gives impetus to research on eating behaviour. Examination of consumer attitudes to nutrition and diet is a relatively recent area of research. However, it is felt that for effective healthy-eating promotion, it is necessary to understand how people perceive their diets (Lennernas *et al*. 1997).

Dietary attitudes were assessed using data obtained from a self-administered attitudinal questionnaire from subjects participating in the North/South Ireland Food Consumption Survey (IUNA, 2001). Subjects selected responses using a 4-point Likert scale ranging from 'most of the time' to 'hardly ever'. This abstract aims to describe the relationship between attitudes and actual dietary behaviour by examining responses to the statements 'I make conscious efforts to try and eat a healthy diet' and 'I try to keep the amount of fat I eat to a healthy amount', as illustrated by the table.

		Food energy (%)			Total energy (%)			NSP fibre (g)	
		CHO	Total fat	Sat. fat	CHO	Total fat	Sat. fat	Mean	(SD)
Target:		(≥50%)	(≤35%)	(≤11%)	(≥47%)	(≤33%)	(≤10%)	(≥18 g/d)	
Population mean		46.5	37.1	14.3	44.4	35.4	13.7	15.2	(6.4)
I make conscious efforts to eat a healthy diet									
Most of time	(*n* 505)	47.5[a]	35.2[a]	13.5[a]	45.8[a]	33.9[a]	13.0[a]	16.4[a]	(6.4)
Quite often	(*n* 279)	45.8[b]	37.6[b]	14.5[b]	43.5[b]	35.6[b]	13.7[b]	14.7[b]	(6.2)
Now & again	(*n* 317)	45.7[b]	38.8[c]	15.0[bc]	43.4[b]	36.9[c]	14.2[bc]	13.6[b]	(5.8)
Hardly ever	(*n* 148)	45.6[b]	39.0[c]	15.7[c]	43.0[b]	36.7[bc]	14.8[c]	13.9[b]	(5.6)
I try to keep fat to a healthy amount									
Most of time	(*n* 481)	47.4[a]	35.3[a]	13.5[a]	45.7[a]	33.9[a]	13.0[a]	16.2[a]	(6.5)
Quite often	(*n* 320)	46.4[a]	37.1[b]	14.3[b]	44.3[b]	35.3[b]	13.7[b]	14.8[b]	(6.4)
Now & again	(*n* 237)	45.7[b]	38.7[c]	15.0[c]	43.2[bc]	36.6[c]	14.2[bc]	13.7[b]	(5.5)
Hardly ever	(*n* 196)	45.2[b]	39.5[c]	15.6[c]	42.6[c]	37.3[c]	14.7[c]	14.6[b]	(6.4)

[a,b,c] Different superscripts within columns denote significant differences for intakes across attitudes ($P<0.05$).

The percentage contribution of carbohydrate (CHO) to food and total energy and the intake of non-starch polysaccharide (NSP) (g/day) was significantly greater in subjects who chose 'most of the time' than 'hardly ever' in response to both statements ($P<0.05$). Conversely, the percentage contribution of total fat and saturated fat (Sat. fat) to food and total energy was significantly lower in subjects who chose 'most of the time' than 'hardly ever' ($P<0.05$). For all macronutrients, mean daily intakes did not comply with dietary guidelines (Department of Health, 1991). Within the four attitudes, comparable macronutrient intakes were recorded across each statement. In general, subjects who chose 'most of the time' had dietary intakes closest to, and those who chose 'hardly ever' had intakes furthest from recommendations.

This study reveals that in the Irish population, some accordance exists between dietary attitudes and behaviour, suggesting that people can reasonably evaluate their own diets. Results from the Pan-EU survey (Lennernas *et al*. 1997) emphasize the need for targeted nutrition messages. Therefore, with the aim of translating dietary guidelines into practice, further research should focus on these attitudes towards perceived diet. This study indicates that particular focus should be paid to those participants who selected either 'most of the time' or 'hardly ever' with regard to further examining the different dietary and socio-demographic characteristics of these groups.

This project was funded by the Department of Agriculture, Food and Rural Development, Dublin.

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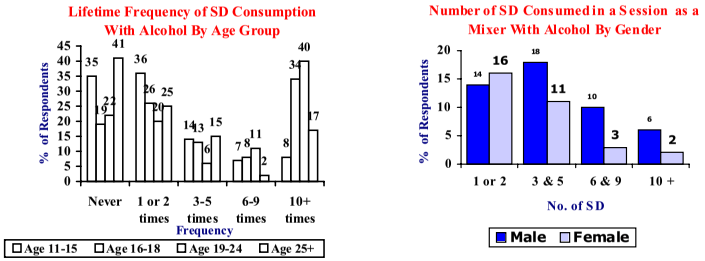
Stimulant drinks: consumption, context and risk factors across the island of Ireland. By D. FINNEGAN[1], G. QUINN[1] and S. NIC GABHAINN[2], [1]*safe*food, *7 Eastgate Avenue, Little Island, Cork, Ireland and* [2]*Centre for Health Promotion Studies, Department of Health Promotion, NUI Galway, Ireland*

There is anecdotal evidence to suggest that stimulant drinks may have detrimental health effects. The Stimulant Drinks Committee set up by *safe*food (the Food Safety Promotion Board) defined stimulant drinks (SD) as: beverages which typically contain caffeine, taurine and vitamin(s) and may contain an energy source (e.g. carbohydrate) and/or other substance(s) marketed for the specific purpose of providing real or perceived enhanced physiological and/or performance effects. Tea, coffee and caffeinated softdrinks were not included in the definition, as they are not marketed as drinks with the latter specific properties. In 2002, the committee published its report "*A Review of the Health Effects of Stimulant Drinks*". Subsequently, the Centre for Health Promotion Studies, NUI Galway was commissioned by *safe*food to carry out further research, in order to investigate the patterns and correlates of stimulant drinks consumption across socio-demographic groups on the island of Ireland. The methodology of this study was qualitative and analysis of results came from two all-island surveys.

	Stimulant Drinks Survey	HBSC Study
Population	Participants recruited from third level institutes, youth clubs and training centres aged (11-35 years)	School going children aged (10-17 years)
Jurisdiction	NI/ ROI	ROI
Survey Instrument	Self completion questionnaire	Self completion questionnaire
Obtained sample	608 completed questionnaires	93 schools/ 5,712 pupils
Aim	Explore consumption of SD in a qualitative fashion	Frequency and consumption of SD

Stimulant drink consumption patterns were analysed alone and as a mixer with alcohol, by age, sex and location. The study also identified self-attributed responses to stimulant drink consumption and identified reasons why the respondents first consumed stimulant drinks.



Results show that 65% of those aged of 11–15 years consumed stimulant drinks with alcohol while 40% of those aged 19–24 years had consumed stimulant drinks on a number of occasions (>10). High consumption levels of stimulant drinks were associated with its use as a mixer with alcohol and the context for this high consumption is by males in licensed premises, especially nightclubs. Curiosity was the most regularly cited reason for first consumption of stimulant drinks. Physiological effects were frequently cited by (>50% of respondents) and the possibility of addiction to stimulant drinks also emerged as an important perception by (42% of respondents). Marketing statistics indicate that Northern Ireland has the highest per capita consumption figure of stimulant drinks in the world, followed by Republic of Ireland, Britain and Germany (*safe*food, 2002). If stimulant drink consumption is considered to be a public health issue and if attempts are to be introduced to reduce consumption, there are now data available which suggest that young, urban males should be the target group.

*Safe*food (The Food Safety Promotion Board) (2002) A Review of the health effects of stimulant drinks. http://www.safefoodonline.com/pdf/health_effects_of_stimulant_drinks.pdf

Percentage body fat in Irish adults measured by bioelectrical impedance analysis compared with published prediction equations. By C.M. MURPHY, M.A. GALVIN, A. FLYNN and M. KIELY, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Percentage body fat (%BF) was measured by bioelectrical impedance analysis (BIA) using the Bodystat 1500 analyser (Bodystat Ltd, UK) during the North/South Ireland Food Consumption Survey (NSIFCS) in 1098 adults (495 men, 603 women) aged 18–64 years. This analyser uses built-in equations to calculate % BF, the origins of which are unknown. Percentage BF data from BIA were compared with %BF data from published prediction equations using various statistical techniques.

Anthropometric data were applied to five age- and sex-specific prediction equations using the following variables: BMI (Deurenberg *et al.* 1991*b*), weight and height (Womersley & Durnin, 1977), waist circumference (Lean *et al.* 1996), impedance, weight and height (van Loan & Mayclin, 1987; Deurenberg *et al.* 1991*a*).

Mean %BF and mean differences (Bland & Altman) between %BF from BIA and %BF from each prediction equation

	Mean %BF				Mean differences			
	Men		Women		Men		Women	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
BIA	21.2	5.8	33.2	7.1	N/A	N/A	N/A	N/A
Deurenberg *et al.* 1991a	28.3	5.7	38.2	5.8	-7.1	1.7	-5.0	2.2
van Loan *et al.* 1987	24.8	5.6	33.4	5.3	-3.6	1.9	-0.2	2.6
Deurenberg *et al.* 1991b	25.0	6.1	34.1	6.6	-3.9	2.7	-0.9	3.1
Womersley *et al.* 1977	25.9	6.1	33.1	6.0	-4.7	3.7	0.6	3.3
Lean *et al.* 1996	25.5	6.9	34.6	6.0	-4.3	3.5	-1.6	4.1

Mean %BF from BIA was significantly lower ($P<0.005$) than mean %BF values estimated by the five equations, with two exceptions: van Loan & Mayclin (1987) in women of all ages and Womersley & Durnin (1977) in women aged 18–50 years. The mean difference (measured using Bland and Altman plots (1986)) between %BF from BIA and %BF from the equations was considerably lower (−1.5%) in women than in men (−4.7%). Mean %BF calculated using BIA was highly correlated ($P<0.001$) with mean %BF from the equations ($r=0.70–0.97$).

%BF results from the van Loan & Mayclin (1987) equation were in most agreement with %BF from BIA, with a mean difference of −3.6% in men and −0.2% in women. In addition, the two methods were highly correlated ($r=0.91–0.96$). %BF results from the Deurenberg *et al.* (1991*a*) (impedance) equation were least similar to %BF from BIA, with a mean difference of −7.1% in men and −5.0% in women. These two methods were also highly correlated ($r=0.94–0.97$).

The study highlights the variability observed between %BF data obtained using the BIA method and the selected prediction equations, and between the %BF data from the prediction equations. This demonstrates inconsistencies between methods for determining %BF in epidemiological studies and highlights the need for independently validated methods.

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Deurenberg P, van der Kooy K, Leenen R, Westrate JA & Seidell (1991*a*) *International Journal of Obesity* **15**, 17–25.
Deurenberg P, Weststrate JA & Seidell JC (1991*b*) *British Journal of Nutrition* **65**, 105–114.
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van Loan M & Mayclin P (1987) *Human Biology* **59**, 299–309.
Womersley J & Durnin JVGA (1977) *British Journal of Nutrition* **38**, 271–284.

Dietary restraint and the diets of Irish adults. By J.L. O'NEILL, S.N. McCARTHY and M.J. GIBNEY, *Department of Clinical Medicine, Trinity College Dublin, Ireland*

Restrained eating refers to restrictions on food intake as a method of controlling body weight through dieting. The theory of restrained eating explains that extreme dieting leaves the individual susceptible to disinhibition, which is subsequently followed by overeating (Van Strien, 1986). This study examines the implications of dietary restraint on anthropometric measurements and dietary intakes of men and women.

This study was based on analysis of data from the North/South Ireland Food Consumption Survey (n=1379) (IUNA, 2001). Dietary restraint was assessed using the Dutch Eating Behaviour Questionnaire (Van Strien *et al.* 1986). Men and women were classified by tertiles of restrained eating as low, medium and high restraint.

	Men						Women					
	low		medium		high		low		medium		high	
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Total population	*n=191*		*n=206*		*n=192*		*n=231*		*n=220*		*n=215*	
Weight(kg)	79.0^{a}	(12.1)	82.9^{b}	(11.9)	87.8^{c}	(14.2)	62.5^{a}	(10.9)	70.2^{b}	(13.3)	70.5^{b}	(12.2)
Height(m)	1.75	(0.1)	1.76	(0.1)	1.75^{ns}	(0.1)	1.61^{a}	(0.1)	1.63^{b}	(0.1)	1.62^{ab}	(0.1)
Waist(cm)	90.6^{a}	(10.6)	93.6^{b}	(10.1)	98.4^{c}	(11.5)	77.6^{a}	(9.9)	83.2^{b}	(12.5)	83.3^{b}	(12.3)
Hip(cm)	101.9^{a}	(6.3)	103.9^{a}	(7.1)	106.4^{b}	(8.0)	98.4^{a}	(8.2)	103.8^{b}	(10.5)	103.6^{b}	(10.1)
Mean BMI	25.5^{a}	(3.6)	26.8^{b}	(3.6)	28.6^{c}	(4.0)	24.1^{a}	(4.2)	26.6^{b}	(5.1)	27.0^{b}	(4.8)
EI:BMR	1.6^{a}	(0.4)	1.4^{b}	(0.4)	1.3^{c}	(0.4)	1.4^{a}	(0.4)	1.3^{b}	(0.3)	1.1^{c}	(0.3)
Food energy (MJ/d) & macronutrient intakes for acceptable energy reporters (EI:BMR ≥ 1.05)*												
	n=178		*n=178*		*n=138*		*n=195*		*n=182*		*n=124*	
Energy	11.3^{a}	(3.1)	10.4^{b}	(2.6)	9.3^{b}	(2.7)	7.8^{a}	(1.9)	7.7^{a}	(1.8)	6.5^{b}	(1.8)
Total Fat$^{\#}$	38.0	(4.7)	37.2	(5.0)	37.0^{ns}	(5.4)	38.9^{a}	(5.3)	38.4^{a}	(5.3)	36.1^{b}	(5.3)
Saturated fat$^{\#}$	15.2^{a}	(2.9)	14.4^{b}	(2.9)	14.0^{b}	(3.1)	15.3^{a}	(2.9)	14.9^{a}	(3.0)	13.8^{b}	(3.0)
Protein$^{\#}$	15.8^{a}	(2.6)	16.5^{b}	(2.9)	16.3^{ab}	(2.4)	15.0^{a}	(2.6)	15.7^{b}	(2.7)	16.7^{c}	(2.9)
Carbohydrate$^{\#}$	46.1	(5.4)	46.1	(5.0)	46.5^{ns}	(5.3)	46.2	(5.7)	45.8	(4.9)	47.0^{ns}	(5.5)

^{a,b,c} Denote significant differences at P<0.05 across tertiles of restrained eating, ^{ns} not significant at P≥0.05.

$^{\#}$ Macronutrient intake as a percentage of food energy (excluding energy from ethanol).

* n=494 (men), n=501(women) (excluding under-reporters EI:BMR<1.05, as defined by Goldberg *et al.* (1991)).

There was a significant increase for both men and women in mean weight, waist and hip circumferences and BMI (body mass index) with increasing restraint (P<0.05).

EI:BMR significantly decreased with increasing restraint for both men and women (P<0.05), indicating that under-reporting is prevalent with restrained eating. Therefore, in order to correctly examine the macronutrient intakes, it was necessary to exclude those respondents with an EI:BMR<1.05 (Goldberg *et al.* 1991). The percentage food energy from total fat significantly decreased with increasing restraint in women (P<0.05). The percentage food energy from saturated fat also significantly decreased with increasing restraint (P<0.05) for both men and women.

When examining the macronutrient intakes, it would appear that the high-restrained eaters have a generally better profile than that of their low-restrained counterparts, although dietary recommendations for total fat, saturated fat and carbohydrate intakes (≤35, ≤11 and ≥50% of food energy, respectively) are still not being achieved (Department of Health, 1991). In contrast, the anthropometric measurements tend to show that low-restrained eaters have a better profile than high-restrained eaters. The relationship between lower macronutrient intakes and higher anthropometric measurements in subjects with high restraint requires further in-depth investigation to see if the lower macronutrient intakes are a result of obese/overweight subjects reporting less than the acceptable energy reporters for food and energy intakes.

This project was funded by the Department of Agriculture, Food and Rural Development, Dublin, Ireland.

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Percentage contribution of macronutrients to energy from foods eaten at home and outside the home: findings from the North/South Ireland Food Consumption Survey. By N.A. O' DWYER, S.N. McCARTHY, S.J. BURKE, A.J. WALLACE and M.J. GIBNEY, *Department of Clinical Medicine, Trinity College Dublin, Ireland*

Research from a number of studies on the Food Service Sector (FSS) have indicated that the contribution of fat to energy intake is higher, and the contribution of carbohydrate to energy intake is lower from foods eaten outside the home than from foods eaten at home (Lin *et al.* 1999; Le Francois *et al.* 1996; Department of Environment, Food and Rural Affairs, 2000). This abstract aims to examine the contribution of the macronutrients to total energy and food energy (excluding ethanol) at home and outside the home. This study is based on analysis of the data from the North/South Ireland Food Consumption Survey for the Republic of Ireland (n 958). The following table shows the contribution of macronutrients to total energy and food energy.

	Male					Female				
	Home		Out			Home		Out		
	(n = 475)		(n =411)			(n = 483)		(n = 410)		
	Mean	(SD)	Mean	(SD)		Mean	(SD)	Mean	(SD)	
% contribution to total energy										
Protein	16.4	(3.3)	11.6	(5.6)	***	16.2	(3.2)	11.7	(6.3)	***
Fat	35.3	(6.1)	28.5	(15.3)	***	35.4	(6.6)	34.6	(14.5)	ns
Carbohydrate	45.7	(6.7)	32.9	(13.1)	***	46.1	(6.8)	36.1	(14.3)	***
Alcohol	2.0	(4.0)	26.9	(27.1)	***	1.7	(3.5)	17.1	(23.4)	***
% contribution to food energy										
Protein	16.7	(3.3)	17.8	(13.0)	ns	16.5	(3.2)	15.4	(11.9)	***
Fat	36.1	(6.0)	35.2	(14.9)	***	36.0	(6.8)	40.0	(13.9)	ns
Carbohydrate	46.6	(6.4)	47.4	(16.2)	ns	46.9	(6.4)	44.2	(16.1)	***

*** P<0.001, ns Non Significant (P>0.05) are for statistical comparisons between locations for men and women

The contributions of protein and carbohydrate to total energy were significantly (P<0.001) lower when eating out than when eating at home for both men and women, while the contribution of alcohol was significantly (P<0.001) higher when eating out. The contribution of fat to food energy was significantly higher at home for men only (P<0.001).

The contribution of fat to total energy and food energy was further examined across tertiles of eating occasions out. There was a decrease in the contribution of fat to total energy from 31 to 27% for men and a significant decrease in the contribution for women from 37 to 31% (P<0.01). However, the percentage contribution of fat to food energy only increased for men from 34 to 36%, with the contributions for women remaining relatively constant across the tertiles of eating occasions out.

From this study, it is evident that ethanol is a confounding factor in examining the contribution of fat to energy in the FSS and this needs to be taken into consideration. Examination of the contribution to food energy illustrates that fat is close to or above the recommendations for men, and always above the recommendations for women, and these must be targeted in developing public health strategies for fat reduction.

This project is funded by the Department of Agriculture, Food and Rural Development.

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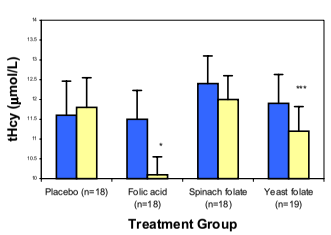
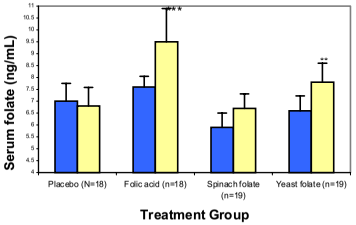
Bioavailability of food folates in a controlled intervention study. By M.P.A. HANNON-FLETCHER[1], N.C. ARMSTRONG[1], J.M. SCOTT[3], M. WARD[1], J.J. STRAIN[1], K. PENTIEVA[1], A.A. DUNN[2], A.M. MOLLOY[3], M. SCULLION[1] and H. McNULTY[1], [1]*Northern Ireland Centre for Food and Health (NICHE)* [2]*School of Hotel, Leisure & Tourism, University of Ulster, Coleraine, BT52 1SA and* [3]*Department of Biochemistry, Trinity College, Dublin, Ireland*

With the introduction of the concept of dietary folate equivalents (DFE) (Institute of Medicine, 1998), current US dietary recommendations for folate are for the first time based on the differences in bioavailability between natural food folates and the synthetic vitamin, folic acid. However, the DFE relies heavily on one study which estimated food folate bioavailability from a mixed diet to be approximately 50% that of folic acid (Sauberlich *et al.* 1987); the interpretation of many bioavailability studies published since then may be problematic as a result of a number of confounding factors. The aim of this study was to examine food folate bioavailability under strictly controlled conditions, which eliminated potential confounding effects including poor subject compliance, displacement of usual dietary folate intake, and folate losses during cooking.

In a placebo-controlled 30-d intervention, eighty male subjects (median age 31 years) were randomised on the basis of their screening plasma homocysteine (tHcy) concentration to receive one of the following treatments administered daily under supervision: food folate (fed either as a folate-enriched meal or drink) or folic acid, each providing 200 µg of total folate, or placebo. To represent the wide range in extent of folate conjugation found in foods, we used two food folate sources, spinach or yeast, in which folates are present as 50% and 100% polyglutamyl folate, respectively. Double fasting blood samples (2–4 d apart) were taken at baseline and on completion of the intervention period. The figure shows mean values for plasma homocysteine and serum folate pre- and post-treatment with 200 µg folate/folic acid.





Plasma homocysteine (µmol/l) **Serum folate (ng/ml)**

Values are mean ± SEM. Differences between pre and post-treatment were measured using a paired *t*-test. Significantly different from pre-treatment: * $P<0.05$, ** $P<0.005$, *** $P<0.001$. Pre-treatment (shaded), post-treatment (open).

The results show significant responses (lowering of tHcy, increase in serum folate) in the folic acid group and in the yeast folate group; response to spinach folate followed this expected pattern, but failed to reach significance. The overall estimations of relative bioavailability for food folate compared with folic acid were 54% and 39% for yeast and spinach folate, respectively. Our estimations of relative bioavailability are consistent with those estimated by Sauberlich *et al*. (1987) from a metabolic study of non-pregnant women which was the cornerstone of the recently derived US DFE value, and furthermore, indicate that the extent of glutamation is not a limiting factor in the bioavailability of folates from natural food sources. These data support the validity of the novel approach to setting dietary folate recommendations recently adopted in the USA.

Supported by a grant from the UK Food Standards Agency, Project Number: NO5013.

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The effects of different processing methods on the *in vitro* accessibility of α- and β-carotenes from carrots. By J.J. MOYLAN, S.M. O'SULLIVAN and N.M. O'BRIEN, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Bioaccessibility is defined as the fraction of a nutrient that is released from its food matrix during digestion and made available for absorption into mucosa. Factors that affect bioaccessibility of carotenoids include the food matrix, the content of dietary fat and fibre, particle size and the food processing method applied. The objective of this study was to investigate the *in vitro* accessibility of α- and β-carotene from carrots that were subjected to various processing methods.

In the present study, raw and cooked carrots, which had previously been washed, peeled and crowned, were freeze-dried to ensure that the same batch of carrots could be used throughout the study. The cooked and raw carrots were subjected to one of three mechanical processing methods: homogenisation, pulping or chopping. The carrots were homogenised by blending in a Moulinex Optiblend 2000. The carrots were finely pulped by passing through a 250 µm aperture sieve. Finally, the chopped carrots were fractionated between two sieves of 4.0 and 2.8 mm aperture. All carrot samples were subjected to an *in vitro* digestion procedure, as described by Garrett *et al.* (1999). The resultant digestate was ultracentrifuged and the supernatant was filtered to remove microcrystalline non-micellularised carotenoids that were not pelleted during ultracentrifugation. This provided a supernatant containing micellularised carotenoids that represented the bioaccessible carotenoids. Carotenoids were quantified by reverse-phase HPLC.

		% Carotenoid bioaccessible			
		α-Carotene		β-Carotene	
		Mean	SD	Mean	SD
Raw	Homogenised	0.99	0.76	0.63	0.43
	Pulped	0.96	0.95	0.66	0.47
	Chopped	0.29	0.15	0.17	0.06
Cooked	Homogenised	1.41	0.69	0.94	0.28
	Pulped	1.39	0.33	1.05	0.12
	Chopped	1.45	0.72	0.98	0.37

n=2

The initial carotenoid content of the carrots prior to processing was 25.0 and 55.7 µg/g carrot for α-carotene and β-carotene, respectively (305.7 and 753.5 µg/g dry matter, respectively). Both carotenoids were transferred to the micelles. The percentage of α- and β-carotene transferred to the micelles was greater for the cooked carrots than for the raw carrots for all treatments. Cooking had an especially marked effect on enhancing the percentage accessible carotenoid from the chopped carrot samples. Homogenisation and pulping resulted in similar percentage of α- and β-carotene accessibility from both raw and cooked carrot. However, chopped raw samples resulted in lower percentage carotenoid accessibility. In conclusion, cooking of carrot resulted in a greater percentage accessibility of carotenoids. The particle size of the carrot had no influence on percentage accessible carotenoid following cooking. However in the raw form, homogenisation and pulping enhanced the percentage accessibility of both carotenoids. In conclusion, in terms of carotenoid bioaccessibility, cooking seems to override other mechanical processing methods.

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Evaluation of the *in vivo* antioxidant activity of wheat bran in human subjects. By R.K. BEATTIE[1], A.M. LEE[1], J.J. STRAIN[1], R.J. FLETCHER[2] and R.W. WELCH[1], [1]*School of Biomedical Sciences, Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, BT52 1SA and* [2]*The Kellogg Company, Manchester M16 0PU*

Observational studies have shown that including whole-grain and bran-rich cereal products in the diet can reduce the risk of coronary heart disease and certain cancers (Jacobs *et al.* 1998, 2000). The basis for this protection is unknown. However one potential mechanism involves the non-nutrient antioxidant phenolic phytochemicals found in cereals such as wheat. *In vitro* studies show that this antioxidant activity in wheat is concentrated in the outer bran layers of the kernel (Beattie *et al*. 2002). Similar non-nutrient antioxidants in wine, tea and fruit, have been shown to be active *in vivo* (Serafini *et al*. 2000; Cao *et al.* 1998) but there are no reports of any such evaluations with wheat. Therefore the aim of this investigation was to evaluate the *in vivo* antioxidant effects of wheat bran in humans.

The study was a cross-over design, randomised within subjects. Seventeen healthy subjects (9F: 8M, 19–39 years, BMI 19–32) participated on two occasions, 5–14 d apart, consuming either 100 g of wheat bran or a ground rice control after an overnight fast. Blood and total urine samples were collected at baseline, and at 30, 60, 120 and 180 min; with a further urine sample at 240 min. Samples were analysed for total antioxidant potential (ferric reducing antioxidant potential, FRAP; Benzie & Strain, 1996) and for total phenolics (TP; Singleton & Rossi, 1965). *In vitro* data indicated TP equivalents (185 mg, 22 mg) and FRAP activity (870 µmol, 130 µmol) for wheat bran and rice control, respectively.

Fig 1: Change in plasma FRAP after the consumption of 100g wheat bran or ground rice (control)

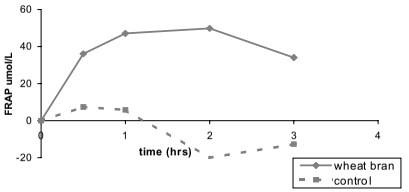

Fig 2: Change in urine FRAP after the consumption of 100g wheat bran or gound rice (control)

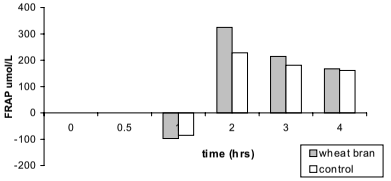

Figure 1 shows a significant increase in plasma FRAP after the consumption of wheat bran, as compared to the control. The rise began soon after consumption and peaked at about 2 h, indicating that absorption is rapid. There was, however, no significant increase in plasma TP over time and results did not positively correlate with the increase in FRAP, which was unexpected (R=0.15, P>0.1). There was also a significant rise in urine FRAP and TP excreted as compared with the control. FRAP excretion began later (Figure 2), again peaking 2 h after consumption of the bran; indicating that the antioxidants absorbed do not stay in the body for a long period of time. This excretion positively correlates with TP excretion (R=0.97, P<0.001). This study indicates that wheat bran can increase plasma total antioxidant status, supporting the hypothesis that bran-rich cereals have the potential to be protective against disease.

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Effects of the inclusion of carrots in different forms in a mixed meal on post-prandial satiety and subsequent intakes. By S.A. MOORHEAD[1], M. McCOURT[1], R.W. WELCH[1], M.B.E. LIVINGSTONE[1], A.A. BURNS[2] and A. DUNNE[3], [1]*Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, BT52 1SA,* [2]*School of Hotel, Leisure and Tourism, University of Ulster, Portrush BT56 8JL and* [3]*Statistics Department, University College Dublin, Dublin 4, Ireland*

Current dietary recommendations are to increase the consumption of fruit and vegetables as they are a good source of fibre and are low in energy density (Department of Health, 1997). Dietary fibre in vegetables has been shown to increase satiety (Gustafsson *et al.* 1994). Previous research has shown that the both the fibre and the structure of fruit were important factors in determining ingestion rate and subsequent satiety (Haber *et al.* 1977). The aim of this study was to extend this work to a realistic eating situation by assessing the effects of carrots in different forms in a mixed meal on post-prandial satiety and subsequent intakes.

The study was a repeated-measures, randomised, within-subject cross-over design. Premenopausal women (n 36, age range 20–40 years, BMI 20–30) were recruited to participate on three occasions, 4 weeks apart. On each occasion the subjects consumed a standard breakfast (25% estimated energy requirement). A lunch meal that included carrots was served 210 min later, followed after a further 210 min by an *ad lib*itum buffet style afternoon meal at which intakes were assessed. Postprandial satiety was measured by subjective assessment using visual analogue scales completed immediately before the lunch meal and every 45 min up to the afternoon meal. Subjects completed food diaries for the remainder of the day. The lunch meal consisted of sweet and sour sauce with chicken and rice (600 g; 2959 kJ) served with carrots in three conditions, whole carrots (fibre and structure), blended carrots (fibre and no structure) and carrot nutrients (no extra fibre and no structure). The carrots or the carrot nutrients (200 g; 289 kJ) were added into the sauce; all meals had the same energy, macronutrients, sodium, potassium and calcium contents and the same volumes and weights. All meals were served with bottled water.

Of the thirty-six subjects recruited, thirty-four completed the whole carrot and the blended carrot conditions while thirty-two completed the carrot nutrient condition. The subjects took significantly less time (mean; SD) to consume the meals with the carrot nutrients (10.2; 1.37 min) followed by the meals with blended carrots (12.9; 1.54 min) and then the whole carrots (16.9; 3.08 min) (P<0.0001). The subjects were significantly (P<0.05) less hungry, fuller and their desire to eat and prospective consumption were less after consuming the meals with the whole and blended carrots compared to the meal with the carrot nutrients. At the *ad libitum* afternoon meal, intakes of food and drink, energy and macronutrients were significantly different (P<0.05) between the conditions and increased in the order whole carrots<blended carrots<carrot nutrients. Similar effects on food intake were observed for the remainder of the day.

Intakes at the *ad libitum* afternoon meal 210 min after consumption of the carrot lunch meals

	Condition					
	Whole Carrots (n 34)		Blended Carrots (n 34)		Carrot Nutrients (n 32)	
	Mean	SD	Mean	SD	Mean	SD
Weight of food & drink (g)	491.1[a]	96.4	536.1[a]	151.5	634.6[b]	103.5
Energy (kJ)	1707[a]	489	2293[b]	904	2935[c]	778
Protein (g)	12.5[a]	4.6	16.3[b]	6.6	18.5[c]	7.0
Fat (g)	17.3[a]	9.9	26.2[b]	16.1	38.0[b]	11.4
Carbohydrate (g)	51.0[a]	17.3	62.5[b]	22.7	72.5[c]	23.7

Means in the same rows with different superscript letters are significantly different (P<0.05).

This study has shown that both the fibre content and the structure of carrots were important determinants of satiety and subsequent food intake when carrots were included in a mixed lunch meal.

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Phytosterol, tocopherol and squalene content of five edible nuts. By L.S. MAGUIRE, K. GALVIN, T.P. O'CONNOR and N.M. O'BRIEN, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Nuts are typically high in fat content but have a fatty acid profile that may be beneficial in relation to risk of coronary heart disease (Kris-Etherton *et al*. 1999). In addition, nuts also contain other potentially cardioprotective constituents, including phytosterols, tocopherols and squalene. Phytosterols are similar in structure to cholesterol and have been shown to reduce absorption of dietary cholesterol (Neil & Huxley, 2002). Tocopherols are powerful antioxidants, protecting cells and lipid carriers from free radical attack. The objective of the present study was to determine and compare the total oil content, the levels of phytosterols, tocopherols, squalene and fatty acid profiles in five edible nuts. Oil was extracted from freshly ground macadamia nuts, hazelnuts, almonds, groundnuts and walnuts based on a procedure previously outlined by Savage *et al.* (1997). The extracted oil was evaluated by HPLC for levels of phytosterols, tocopherols and squalene and by GC for fatty acid profile.

	% yield		Campesterol (μg/g oil)		Stigmasterol (μg/g oil)		β-Sitosterol (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
Macadamia	59	2	73	9	38	3	1506	141	122	25	trace	0	185	27
Hazelnut	45	3	50	9	22	6	828	185	251	60	64	11	173	28
Almond	41	3	55	11	52	4	2072	26	440	5	13	2	95	9
Groundnut	38	2	198	21	163	23	1363	180	88	7	60	7	98	13
Walnut	48	3	30	5	40	14	1003	21	15	2	6	0	5	0

n 3 independent experiments.

	PUFA (g/100g)		MUFA (g/100g)		Total SFA (g/100g)		Total USFA (g/100g)		Unsaturated/ saturated	
	Mean	se	Mean	se	Mean	se	Mean	se	Mean	se
Macadamia	2.4	0.4	82.4	0.6	15.1	0.3	84.8	0.3	5.6	0.5
Hazelnut	34.6	20.0	56.3	20.4	9.2	0.4	90.8	0.4	9.9	0.1
Almond	21.7	0.5	69.9	0.4	8.5	0.5	91.6	0.5	10.8	0.2
Groundnut	46.0	1.1	38.6	0.9	15.5	0.3	84.5	0.3	5.5	0.7
Walnut	75.9	15.3	14.5	14.5	9.6	1.2	90.4	1.3	9.4	0.8

n 3 independent experiments.

The total oil content of the nuts ranged from 38 to 59% (w/w), with the macadamia nut yielding the greatest percentage of oil. The levels of squalene detected ranged from 5 to 185 μg/g oil. β-Sitosterol was the most prevalent phytosterol and was present in much higher concentrations than campesterol and stigmasterol in all five nuts. α-Tocopherol was the most prevalent tocopherol and was present in much higher concentrations in hazelnuts and almonds than in groundnuts, walnuts and macadamia nuts. γ-Tocopherol was measured in all five nut samples (see Table) while δ-tocopherol was present in trace amounts (data not shown). All five nuts were high in unsaturated fatty acids. Our data indicate that nuts are a good dietary source of phytosterols, tocopherols, squalene and unsaturated fatty acids.

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Lycopene supplementation has no effect on IGF-1 status in healthy male subjects. By R. GRAYDON, J.V. WOODSIDE and I.S. YOUNG, *School of Clinical Medicine, Queen's University Belfast, Belfast BT12 6BJ*

Insulin-like growth factor-1 (IGF-1) is a potent mitogen for cancer cells. A high circulating level of IGF-1 has been shown to be a risk factor for cancer development at a variety of sites including breast and prostate (Moschos & Mantzoros, 2002). Lycopene, a carotenoid found predominantly in tomatoes and tomato products, has been suggested to have anti-cancer activity in *in vitro*, *in vivo* and epidemiological studies (Heber *et al.* 2001). Lycopene has been shown to reduce breast cancer cell line growth through interference in IGF-1 receptor signalling and cell cycle progression (Levy *et al.* 1995). Consumption of cooked tomatoes has been inversely associated with IGF-1 concentration (Mucci *et al.* 2001), but it is not known whether lycopene supplementation can alter circulating IGF-1 concentrations *in vivo*.

This study was a 1-month randomised, double-blind, placebo-controlled study in healthy male volunteers aged 20–60 years (n=20). A supplement containing 15 mg lycopene daily was used. Fasting blood samples were collected at baseline and after 1-month's supplementation. Samples were analysed for lycopene by high-performance liquid chromatography (Craft *et al.* 1992) and insulin-like growth factor-1 (ELISA kit, ImmunoDiagnostics Systems Ltd.) and changes in these from baseline were compared in those who received placebo *versus* those who received the lycopene supplement. Mean change in lycopene from baseline (post-supplement − baseline) was higher in subjects on lycopene intervention than on placebo (lycopene group 0.28 (0.21); placebo group −0.003 (0.15) μmol/l; Mean (SD), P<0.01), but there was no difference in mean change in IGF-1 concentrations between intervention groups (lycopene group −0.33 (2.30); placebo group −0.94 (2.12) nmol/l; Mean (SD), P=NS).

In a second study, a group of clinically healthy male factory employees aged 30–49 years (n=509) were screened for serum lycopene concentrations. The top and bottom 10% of the lycopene distribution were analysed for IGF-1 concentrations. There was no difference in IGF-concentration between the top and bottom 10% of the lycopene distribution (top 10% lycopene 8.21 (2.73); bottom 10% lycopene 7.65 (2.14) nmol/l; Mean (SD), P=NS).

These studies show that lycopene supplementation in healthy male subjects has no effect on IGF-1 concentration, while lycopene status does not appear to be associated with IGF-1 status in a healthy male population.

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Use of nutritional supplements in Irish adolescents. By S. Byrne and C. Corish, *St. Catherine's College of Education for Home Economics, Sion Hill, Blackrock, Co. Dublin, Republic of Ireland*

Recent studies reveal that 33% (Stang *et al*. 2000) and 20% (Dwyer *et al*. 2001) of American adolescents report using nutritional supplements. In the UK, 20% of adolescents have been reported to use nutritional supplements (Gregory *et al*. 2000). The North/South Ireland Food Consumption Survey (Kiely *et al*. 2001) reported that 23% of Irish adults use nutritional supplements. However, no information is available on the use of nutritional supplements among Irish adolescents.

The purpose of the present study was to characterise nutritional supplement use in Irish adolescents aged 15 to 18 years and the relationship between use and a number of lifestyle factors.

A cross-sectional study recruited 406 adolescents from nine (three from Dublin and six from Wexford) Irish secondary schools, of whom 390 (207 boys and 183 girls) were eligible for inclusion into the study. The students completed a self-administered questionnaire that investigated their knowledge and use of nutritional supplements, their motivation for using these supplements, their levels of physical activity, their smoking habits and socio-economic status. Nutritional intake was estimated in a random sample (*n* 93) of these students using a 24-h dietary recall and photographic food atlas, reflecting weekday intake only. Basal metabolic rate (BMR) for each subject was estimated and reported energy intakes (EI) were expressed as a multiple of BMR (EI:BMR) with only subjects having an EI:BMR greater than 1.35 (*n* 52) being included in the dietary analysis. Subjects were weighed and measured to obtain anthropometric measurements of height, weight and waist circumference. BMI was calculated from the weight and height measurements.

Eighty nine percent (*n* 345) of subjects studied had heard of the term nutritional supplement, a 'nutrient booster' being the most frequently selected description. Nearly half (44%, *n* 171) reported using nutritional supplements, 25% (*n* 98) using them on a daily basis and 6% (*n* 24) using them more frequently. Multivitamin or multivitamin plus minerals were the most frequently used supplement, taken by 36% of the supplement users. The most popular single vitamin was vitamin C and the most popular mineral was iron. Only six students used non-nutritional supplements. Supplement use was greater in those of younger age (P=0.07), those with lower abdominal adiposity (P<0.05) and those with higher levels of physical activity (P<0.05). No associations were observed between supplement use and gender, socio-economic status, place of residence, BMI, smoking status or any of the nutrients (energy, fat, saturated fat, calcium, iron, folate, vitamin B_{12}, vitamin B_6, vitamin C, vitamin D and vitamin E) assessed. The majority of users (58%, *n* 120) indicated that parental influence was the motivation for supplement use, the reported rationale for supplement use given by most (61%, *n* 106) was to remain healthy.

With the exception of iron and folate, average nutrient intakes (excluding nutritional supplements) were above the recommended dietary allowances (Food Safety Authority of Ireland, 1999). Further investigation is required to determine actual nutrient intakes when the contribution from supplementation is assessed. As parents exerted greatest influence on supplement use, it may be necessary to increase parental understanding of the appropriateness of nutritional supplements.

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The impact of fortified foods on the level of inadequacy of micronutrient intake and non-compliance with dietary recommendations in Irish adults. By T. JOYCE, E.M. HANNON, M. KIELY and A. FLYNN, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

This study was carried out to examine the association of fortified food consumption with overall dietary quality. The analysis was based on the North/South Ireland Food Consumption Survey database, which was compiled from a 7 d food diary collected in a representative sample of 1379 Irish adults aged 18–64 years (662 men, 717 women) (http://www.IUNA.net/survey2000.htm). The survey showed that 69% of the population consumed fortified foods. Almost 3% of the 3060 foods consumed in total were fortified, of which 62% were breakfast cereals (Hannon *et al*. 2001). Men and women were classified by tertile of fortified food consumption (kJ/d) into low, medium and high consumers. Compliance with dietary recommendations and adequacy of micronutrient intakes were examined.

		Men				Women			
Fortified food consumers†		Non *n*=213	Low *n*=149	Medium *n*=150	High *n*=150	Non *n*=209	Low *n*=169	Medium *n*=170	High *n*=169
Guidelines		**% Not achieving dietary guidelines**							
Fat: ≤ 35% food energy*		72[a]	68[a]	68[b]	57[b]	81[a]	70[b]	57[c]	48[c]
Carbohydrates: ≥ 50% food energy*		84[a]	84[a]	73[b]	65[b]	88[a]	76[b]	70[c]	56[d]
NSP: ≥ 18g/d*		71[a]	74[a]	73[a]	49[b]	94[a]	88[b]	88[b]	75[c]
Fruit and vegetables: ≥ 400g/d**		83[a]	87[b]	77[ac]	66[c]	84	80	81	79
Alcohol: men <21 units/wk, women <14 units/wk		43[a]	38	30[b]	29[b]	20	23	16	16
Nutrient	**AR†† men (women)**	**% With intakes below the average requirement (AR)**							
Calcium	550 mg	16[a]	10[a]	11[a]	3[b]	35[a]	25[b]	18[b]	12[c]
Iron	7 mg (10 mg 18–50 years, 6 mg 51–64 years)	4[a]	3[a]	3[a]	0[b]	57[a]	53a	34[b]	15[c]
Riboflavin	1.3 mg (1.1 mg)	23[a]	14[b]	9[b]	1[c]	36[a]	28[a]	12[b]	1[c]
Folate	140 µg	5[a]	2	1	0[b]	22[a]	5[b]	4[b]	2[b]

† Daily energy from fortified foods: low 12–217 kJ (men), 5–190 kJ (women); medium 222–482 kJ (men), 188–443 kJ (women); high 480–2954 kJ (men), 448–1992 kJ (women).
[a,b,c,d] Different superscripts denote significant differences between tertiles at P<0.05 for men and women separately.
* Department of Health, 1991.
** WHO/FAO, 2003.
†† Scientific Committee for Food, 1993.

A higher consumption of fortified foods was associated with a greater percentage of men and women meeting the recommendations for fat, carbohydrate and non-starch polysaccharide (NSP) and a greater percentage of men meeting the guidelines for alcohol and fruit and vegetables. Higher consumption of fortified foods was also associated with a lower level of dietary inadequacy for calcium, iron, riboflavin and folate, particularly in women. This study shows that fortified food consumption is associated with better overall dietary quality.

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The contribution of dairy products to the Irish diet. By S.J. BURKE, S.N. McCARTHY, N.A. O'DWYER, A.J. WALLACE and M.J. GIBNEY, *Department of Clinical Medicine, Trinity College Dublin, Ireland*

Dairy products are an important source of calcium and are therefore essential in the diet for bone development and osteoporosis prevention. This abstract firstly examines the contribution of dairy products to nutrient intakes in the Irish diet and secondly examines nutrient intakes across tertiles of dairy product consumption with a view to formulating food-based dietary guidelines.

The North/South Ireland Food Consumption Survey database was used for this analysis. It was shown that almost 100% of men and women from the Republic of Ireland (n = 958) consumed dairy products, with 92% consuming full-fat milk, 77% consuming cheese, 33% consuming reduced-fat milk and 33% consuming yoghurt. Dairy products also made important contributions to the intakes of energy (11%), protein (15%), fat (17%), calcium (48%), phosphorus (24%), vitamins B_2 and B_{12} (31%) and vitamin A (27%) in the population.

The mean intake of macronutrients as a percentage of food energy and micronutrients per 10MJ of food energy was analysed by tertile of dairy product consumption, as shown in the following table

		Fat		Protein		CHO		Calcium		Iron		Folate	
		Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Full fat milk	Low (n 294)	36.1 ^{a}	(5.8)	17.0 ^{a}	(3.1)	46.5	(5.6)	899.8 ^{a}	(270.8)	14.8 ^{a}	(4.6)	332.1 ^{a}	(118.1)
	Med (n 294)	37.5 ^{b}	(5.2)	16.3 ^{b}	(2.7)	45.8	(5.1)	874.7 ^{a}	(195.1)	13.5 ^{b}	(3.2)	308.0 ^{b}	(93.2)
	High (n 294)	38.4 ^{b}	(5.2)	15.4 ^{c}	(2.4)	45.8 ^{NS}	(5.3)	1030.5 ^{b}	(223.1)	12.9 ^{b}	(3.8)	299.6 ^{b}	(88.4)
Cheese	Low (n 247)	36.3 ^{a}	(5.9)	16.4 ^{a}	(2.7)	47.0 ^{a}	(5.8)	897.2 ^{a}	(232.9)	13.9	(3.6)	321.3	(115.5)
	Med (n 243)	37.3 ^{a}	(5.1)	16.2 ^{ab}	(2.8)	46.1 ^{a}	(5.0)	930.3 ^{a}	(223.0)	13.8	(3.8)	312.9	(92.3)
	High (n 247)	39.3 ^{b}	(5.2)	15.8 ^{b}	(2.5)	44.5 ^{b}	(5.4)	1039.7 ^{b}	(243.3)	13.3 ^{NS}	(3.8)	303.8 ^{NS}	(94.5)
Reduced fat milk	Low (n 106)	37.1 ^{a}	(5.9)	16.2 ^{a}	(2.8)	46.2 ^{a}	(5.3)	914.0 ^{a}	(211.5)	14.0 ^{a}	(3.5)	316.3 ^{a}	(99.2)
	Med (n 107)	33.5 ^{b}	(5.9)	17.8 ^{b}	(3.3)	48.4 ^{b}	(6.2)	998.8 ^{b}	(206.4)	16.3 ^{b}	(6.0)	357.1 ^{b}	(133.4)
	High (n 107)	34.1 ^{b}	(5.9)	17.4 ^{b}	(2.9)	48.2 ^{b}	(5.7)	1200.4 ^{c}	(271.8)	15.2 ^{ab}	(4.2)	361.2 ^{b}	(105.2)
Yoghurt	Low (n 104)	37.5 ^{a}	(5.3)	15.9 ^{a}	(2.6)	46.4 ^{a}	(5.2)	939.0 ^{a}	(244.1)	14.0 ^{a}	(4.1)	310.8 ^{a}	(89.6)
	Med (n 105)	36.9 ^{a}	(6.3)	15.5 ^{a}	(2.7)	47.0 ^{ab}	(6.0)	1032.1 ^{b}	(244.8)	13.7 ^{a}	(4.0)	303.0 ^{a}	(86.3)
	High (n 105)	33.4 ^{b}	(6.0)	17.4 ^{b}	(3.2)	48.8 ^{b}	(5.8)	1118.3 ^{c}	(252.1)	16.2 ^{b}	(5.6)	376.7 ^{b}	(144.8)

^{abc} Denotes significant differences at $P<0.05$ for nutrient intakes across tertiles of dairy product consumption; ^{NS}, not significant ($P> 0.05$).

In general, fat and calcium intakes increased, while intakes of protein, carbohydrate, iron and folate decreased with increasing consumption of full-fat milk and cheese. Intakes of fat decreased, while intakes of all other nutrients increased, with increasing intakes of reduced-fat milk and yoghurt. In addition, high consumers of reduced-fat milk and yoghurt appeared to have diets with better nutrient quality than high consumers of full-fat milk and cheese.

Calcium intakes are below the average requirement in 11% of Irish men and 23% of Irish women (Hannon *et al.* 2001). Increased consumption of dairy products should be advised in order to increase calcium intakes. However, this could lead to undesirable increases in fat intakes. Increased consumption of reduced-fat milk and yoghurt could be advised specifically, as these products are high in calcium and low in fat. Also high consumption of reduced-fat milk and yoghurt appears to be associated with better nutrient quality diets. Increased consumption of these foods could be achieved by increasing the number of consumers (as only 33% of the population consumed them) and/or increasing the frequency of consumption or the serving size for people who already consume the foods. An additional method to increase calcium intakes without subsequently increasing fat intakes would be to replace full fat milk with reduced fat milk, or to replace full fat cheese and yoghurt with lower fat varieties.

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Hannon EM, Kiely M, Harrington KE, Robson PJ, Strain JJ & Flynn A (2001) *Public Health Nutrition* **4**, 1081–1088.

Phosphate and calcium intakes in forty-two haemodialysis patients. By F.N. BYRNE[1,2,3], M. KIELY[2], A. FLYNN[2], W.D. PLANT[3] and D.J. MURNAGHAN[3], [1]*Department of Nutrition and Dietetics, Cork University Hospital, Cork, Ireland,* [2]*Department of Food and Nutritional Sciences, University College Cork, Ireland and* [3]*Department of Renal Medicine, Cork University Hospital, Cork, Ireland*

Hyperphosphataemia is a cause of renal osteodystrophy (Malluche & Monier-Faugère, 2000), as well as significant morbidity and mortality in haemodialysis (HD) patients (Block *et al.* 1998). As kidney function declines, kidneys lose their ability to excrete phosphorus, produce calcitriol and maintain calcium homeostasis. An important means of controlling serum phosphate (PO_4) levels is to restrict dietary phosphorus (P) intake and to prescribe PO_4 binders with meals. Orally administered calcium-containing salts, namely calcium carbonate and calcium acetate, are the most commonly used binders. The aim of the current study was to examine the temporal distribution of P intake to evaluate the effectiveness of PO_4 binder administration in relation to P intake.

Intakes of foods and nutrients, PO_4 binders and biochemical control were assessed in forty-two HD patients (twenty-five men and seventeen women). Patients kept a detailed diary of all food, drink and PO_4 binders consumed at each eating occasion over 7 d. Blood samples were taken before and after dialysis on the first and last day of the study, and were analysed for serum PO_4. Dietary analysis was carried out using WISP (Tinuviel Software, Warrington, UK), which uses McCance and Widdowson's *The Composition of Foods* (5th edn.) and supplements to calculate nutrient intakes. Bound P was assessed by assigning a binding capacity to each binder consumed. This was assessed from two P balance studies by Sheikh *et al.* (1998) and Burke *et al.* (1997).

The average P intake from dietary sources was 995 mg/d, with 74% (n=31) of patients meeting the Irish guideline of 1200 mg/d (Irish Nutrition and Dietetic Institute, 1998), but 37% of patients had poor PO_4 control as defined by a serum PO_4 of >2.1 mmol/l (Block *et al.* 1998).

	*Frequency of consumption of meals & snacks	Mean P intake (mg)		Mean P bound (mg)		
		Mean	SD	Mean	SD	
Meals						% bound
Breakfast (n=42)	286	207	105	61	40	30%
Light meal (n=40)	238	258	105	58	36	23%
Main meal (n=42)	281	434	144	72	50	17%
Snacks						
Morning snack (n=33)	157	85	66	9	18	11%
Afternoon snack (n=34)	111	90	68	7	22	8%
Evening snack (n=41)	274	99	65	10	17	9%

*Frequency of consumption describes the number of each meal type that was consumed by all subjects over the 7 d study.

The main meal was the highest source of P in the day, and on average only 17% of this was bound. More P was bound at breakfast (30%) compared with other meals. Overall, calcium intakes were dictated by the quantity of calcium-containing PO_4 binders consumed. As dairy products are restricted in an effort to limit P intakes, mean calcium intake for the group from dietary sources only was 586 (SD 212) mg/d. When PO_4 binders were included in the analysis, the mean calcium intake increased to 1996 (SD 1020) mg/d (range 334–5155 mg/d).

In conclusion, PO_4 binder administration should be increased during main meals to maximize efficacy. A coordinated multidisciplinary approach to prescription of binders is necessary to ensure optimal tailoring of binders to phosphate intake. Efforts to minimise dietary intake must be renewed. More research is needed to define the optimum calcium intake for optimum bone health in renal disease.

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Comparison of vitamin D intake and status and serum parathyroid hormone in patients with Crohn's disease and healthy sex- and age-matched control subjects. By D. MCCARTHY[1], M. O'BRIEN[1], M. KIELY[1], F. SHANAHAN[2] and K.D. CASHMAN[1,2], [1]*Department of Food and Nutritional Sciences and* [2]*Department of Medicine, University College, Cork, Ireland*

Osteopenia and osteoporosis are common conditions among patients with Crohn's disease (Driscoll *et al*. 1982). Hypovitaminosis D is also prevalent in patients with Crohn's disease (Driscoll *et al*. 1982). Deficiency of vitamin D and the consequential elevation in parathyroid hormone (PTH) levels may have a role in bone loss in these patients. Therefore, the aims of this study were firstly to compare the intake and status of vitamin D in patients with Crohn's disease with that of healthy, age-matched control subjects, and secondly, to determine the effect of vitamin D status on serum PTH levels (an important mediator of bone metabolism) in these groups. Forty-four free-living patients with Crohn's disease (mean age 37 years; range 19–65 years) were recruited from the inflammatory bowel disease clinic at Cork University Hospital. Forty-four healthy, age- and sex-matched control subjects were recruited from among staff at University College Cork and from among friends and acquaintances. Dietary intake of vitamin D and Ca was estimated by a food frequency questionnaire. Fasting blood samples were collected during September and October 2002. Bloods were processed to serums and analyzed for 25 hydroxyvitamin D and PTH by enzyme-linked immunosorbent assays (ELISA). The quality of serum 25(OH) D_3 analysis in our laboratory is assured on an ongoing basis by participation in the DEQAS external quality assurance scheme (London, UK). Serum 25(OH) D_3 cut-off values for defining vitamin D status as adequate, marginally deficient or severely deficient were: >40 nmol/l, 25–40 nmol/l and <25 nmol/l, respectively (Vieth, 1999). The adult normal range for PTH is 0.8-3.9 pmol/l, and PTH levels >4.1 pmol/l were indicative of a state of hyperparathyroidism (as indicated by IDS Ltd).

There were no significant differences in weight, height or BMI between the Crohn's disease patients and age- and sex-matched healthy controls. Daily vitamin D intakes (ie from food and supplements) were similar in Crohn's disease patients and healthy controls (mean (SD), 6.7 (5.1) and 6.9 (4.8) μg, respectively; P=0.883). Similarly, daily Ca intakes (ie from food and supplements) were similar in both groups (mean (SD), 1491 (750) and 1295 (525) mg, for Crohn's patients and matched-controls, respectively; P=0.147). The percentage of the subjects that took a vitamin D- and/or Ca-containing supplement was similar in both groups (33 and 30 %, for the Crohn's patients and matched-controls, respectively). Mean serum 25(OH) D_3 was significantly lower in the Crohn's disease patients than in the healthy controls (mean (SD), 75 (29) and 106 (56) nmol/l, respectively; P=0.001). Serum PTH levels were similar in both groups (mean (SD), 2.04 (1.22) and 1.85 (0.89) pmol/l, for Crohn's patients and matched-controls, respectively; P=0.319). Of the total group of Crohn's disease patients (n 44), four patients were marginally deficient in vitamin D, whereas two of the healthy controls (n 44) were marginally vitamin D deficient.

In conclusion, hypovitaminosis D in late summer to early winter, a time of year when vitamin D status would be expected to be at its highest, was twice as prevalent in Crohn's disease patients than healthy control subjects. Therefore, it would seem prudent to determine the prevalence of hypovitaminosis D in Crohn's disease patients during late winter to early spring, a time of year when vitamin D status would be expected to be at its lowest.

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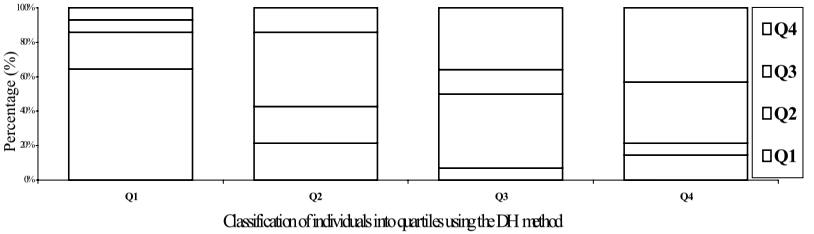
Vieth R (1999) *American Journal of Clinical Nutrition* **69**, 842-856.

Comparison of a 69-item food frequency questionnaire (FFQ) with a 14-day dietary history (DH) to estimate vitamin D intakes in 50–75-year-old Irish women. By A. COLLINS, M.M. O'BRIEN, A. FLYNN, K.D. CASHMAN and M. KIELY, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Vitamin D is the major regulator of calcium metabolism and hence is an important determinant of bone health (Van Leeuwen *et al*. 2001). O'Brien *et al*. (2001) showed that vitamin D intakes were low in Irish adults. As sunshine levels in Ireland are low and sun exposure is the main determinant of vitamin D status, it may be prudent to investigate dietary strategies to increase vitamin D intakes, particularly in vulnerable population groups. Due to the infrequent consumption of vitamin D-rich foods, the measurement of habitual vitamin D intakes is onerous and places a heavy burden on respondents. The aim of the current study was to develop a food frequency questionnaire (FFQ) to estimate vitamin D intakes. In the absence of an independent biomarker of vitamin D intakes, data from the FFQ are compared with data concerning food intake over a typical 14d period (diet history; DH).

A 69-item FFQ was constructed including the foods that contributed 95% of vitamin D intakes in the Irish population, using the North/South Ireland Food Consumption Survey database (IUNA, 2001). Fifty-six apparently healthy, free-living Irish women from the Cork city region (50–75 years; mean age 67 years) completed the FFQ and DH on two separate occasions within 1 month of each other. The same researcher carried out all assessments and estimated food portion sizes using a photographic food atlas (MAFF, 1997). Vitamin D values were assigned to the DH and FFQ primarily using McCance and Widdowson's *The Composition of Foods* (5th edn.), supplemented by recently compiled vitamin D values in selected foods (Hill *et al.* in press). Paired t-tests were used to compare methods.

Vitamin D intakes (excluding supplements) estimated by the DH (mean 2.56 μg, SD 1.65 μg) were significantly lower (P<0.001) than the FFQ (mean 4.58 μg, SD 2.90 μg) (r 0.34, P<0.05). The mean difference between vitamin D estimates from the FFQ and DH methods (Bland & Altman 1986) was −2.03 (SD 2.8). Agreement between the two methods in classifying individuals into the same or adjacent quartile levels (Q1=lowest consumers, Q2=Quartile 2, Q3=Quartile 3, Q4=highest consumers of vitamin D intake was determined by each method separately was between 79 and 93%.



The FFQ designed for this study is probably not acceptable for the assessment of individual vitamin D intakes. However, these data show that it could be a useful tool in assigning individuals into categories of vitamin D intake. Further investigations are ongoing in other population groups.

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Seasonal variation in vitamin D status in a group of 50–70-year-old Irish women. By T.R. HILL[1], M.M. O'BRIEN[1], M. KIELY[1], A. FLYNN[1] and K.D. CASHMAN[1,2], [1]*Department of Food and Nutritional Sciences and* [2]*Department of Medicine, University College Cork, Ireland*

The most widely used biochemical marker of vitamin D status is the level of plasma or serum 25-hydroxyvitamin D_3 (25(OH) D_3). Hill *et al*. (2002) previously reported that the prevalence of low vitamin D status in a group of Irish women, aged 50–70 years, was about 30% during late winter to early spring, a time when vitamin status is expected to be at its lowest. However, season is the most important determinant of vitamin D status (Stamp & Round, 1974). Therefore, in the present study, the vitamin D status of the same group of 50–70 year-old women was re-assessed during the late summer to early autumn months, when vitamin D status would be expected to be at its highest.

Fasting blood samples were collected (between 08.00 and 10.00 hours) during summer to early autumn (August and September 2002) from fifty-three apparently healthy, free-living Irish women, aged between 50 and 70 years (mean age 60 years), who had previously provided blood samples (during February/March 2002; Hill *et al*. 2002). Bloods were processed to serum and analysed for 25(OH) D_3 and PTH by enzyme-immunoassays (IDS Ltd, UK). The quality of serum 25(OH) D_3 analysis in our laboratory is assured on an ongoing basis by participation in the DEQAS external quality assurance scheme (London, UK). Serum 25(OH) D_3 cut-off values for defining vitamin D status as adequate, marginally deficient or severely deficient were: >40 nmol/l, 25–40 nmol/l and <25 nmol/l, respectively (Vieth, 1999). The normal adult range for PTH is 0.8–3.9 pmol/l, and PTH levels >4.1 pmol/l were indicative of a state of hyperparathyroidism (as indicated by IDS Ltd).

	Winter 2002			Summer 2002		
	Mean	SD	Range	Mean	SD	Range
25 (OH) D_3 (nmol/l)	56.3	26.9	17.1–140.0	77.5*	29.1	31.2–154.7
PTH (pmol/L)	2.48	1.16	0.42–6.14	2.46	0.77	1.05–5.31

Significantly different from values for winter: *P<0.001.

Mean serum 25 (OH) D_3 concentration was significantly higher (P<0.001) in summer 2002 than winter 2002. While the prevalence of vitamin D deficiency (serum 25 (OH) D_3 ≤ 40 nmol/l) was estimated at about 30% in winter, it was only about 4% (*n* 2 out of total of 53) in summer 2002. Neither of these two women affected, used vitamin D-containing supplements, the use of which appears to protect against vitamin D deficiency during winter (Hill *et al*. 2002). There was no significant difference (P=0.878) in serum PTH levels among the women in winter compared with summer. However, while 12% of women (*n* 4) had hyperparathyroidism during the winter, only 2% (*n* 1) of the group had hyperparathyroidism during the summer.

In conclusion, although a significant number of 50–70-year-old women (about 30%) may be at risk of hypovitaminosis D during winter to early spring, this risk is significantly reduced in summer. Increased dermal synthesis of vitamin D upon exposure to sunlight during the summer months appears to protect against vitamin D deficiency in this group of women.

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Comparison of vitamin K intake and status and bone metabolism in patients with Crohn's disease and healthy sex- and age-matched control subjects. By P. DUGGAN[1], M. O'BRIEN[1], M. KIELY[1], F. SHANAHAN[2] and K.D. CASHMAN[1,2], [1]*Department of Food and Nutritional Sciences and* [2]*Department of Medicine, University College, Cork, Ireland*

Osteopenia and osteoporosis are common conditions among patients with Crohn's disease. While anti-inflammatory steroid treatments prescribed for these patients can adversely affect skeletal integrity, a deficiency of certain bone-active nutrients may also have a role in the bone loss. For example, a reduced vitamin K status has been reported in Crohn's disease patients compared with healthy controls (Schoon *et al*. 2001). Vitamin K is a cofactor for γ-carboxylase and, as such, is required for the carboxylation of at least three bone-related proteins, including osteocalcin. Serum undercarboxylated osteocalcin (Glu; a marker of vitamin K status) is a risk factor for bone loss and hip fracture (Szulc *et al*. 1994, 1996). Therefore, the aims of this study were firstly to compare the intake and status of vitamin K of patients with Crohn's disease with that of healthy, sex- and age-matched control subjects, and secondly, to determine the effect of vitamin K status on bone metabolism in these groups. Forty-four free-living patients with Crohn's disease (mean age 36.9 years; range 19–65 years), currently in remission, were recruited from the inflammatory bowel disease clinic at Cork University Hospital. Forty-four healthy, sex- and age-matched control subjects were recruited from among staff at University College Cork and from among friends and acquaintances. Fasting blood and urine samples were collected during September and October 2002. Bloods were processed to serums and analysed for Glu by enzyme-linked immunosorbent assay (ELISA). Urine was analysed for creatinine (Cr) and cross-linked N-telopeptides of Type 1 collagen (NTx; a marker of bone resorption) by a colorimetric and ELISA technique, respectively. Dietary intake of vitamin K_1 was estimated by a food frequency questionnaire, using food compositional data provided by Bolton-Smith *et al*. (2000).

Mean daily vitamin K_1 intakes in Crohn's disease patients tended to be lower than that of matched healthy controls (mean (SD), 117 (82) *v*. 148 (80) μg, respectively; P=0.059). Mean serum Glu and urinary NTx levels in Crohn's disease patients were higher than that of healthy controls (mean (SD), 5.1 (3.1) *v*. 3.9 (2.1) ng/ml, respectively; P=0.03 for Glu; and 49.0 (41) *v*. 25.8 (19.5) nM bone collagen equivalents/mM Cr, respectively; P=0.001 for NTx). In the Crohn's disease patients serum Glu was significantly correlated with urinary NTx (r=0.488; P<0.001). Multiple regression analysis showed that this relationship was maintained even after controlling for vitamin D status in the patients.

In conclusion, vitamin K status of patients with Crohn's disease was lower, and vitamin K_1 intake tended to be lower than that of sex- and age-matched healthy control subjects. Furthermore, the rate of bone resorption in the Crohn's patients was double that of the controls. In the Crohn's disease patients, the rate of bone resorption was inversely correlated with vitamin K status. An increased rate of bone resorption is associated with an increased risk of bone loss in these patients. Therefore, it would seem timely to investigate the effect of vitamin K supplementation on bone health of Crohn's disease patients.

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Fruit and vegetable consumption and bone mineral density in adolescence: the Northern Ireland Young Hearts Project. By C. McGARTLAND[1], P.J. ROBSON[1], L. MURRAY[2], G. CRAN[2], D. WATKINS[3], M. ROONEY[4], J. SAVAGE[5] and C. BOREHAM[6], [1]*Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, BT52 1SA,* [2]*Department of Epidemiology, The Queen's University of Belfast, BT12 6BJ,* [3]*Department of Neonatal Medicine, St Mary's Hospital for Women and Children, Manchester M13 OJH,* [4]*Queen's Orthopaedic Building, Musgrave Park Hospital, Belfast BT9 7JB,* [5]*Department of Child Health, The Queen's University of Belfast, BT12 6BJ and* [6]*School of Applied Medical Sciences and Sports Studies, University of Ulster, Jordanstown, BT37 0QB*

High intakes of fruits and vegetables may be protective of bone health in elderly people (Tucker *et al.* 1999), and childhood consumption of fruit has been positively linked with bone mineral density (BMD) in postmenopausal women (New *et al.* 1997). The aim of the present study was to examine the association between fruit and vegetable consumption and BMD measured at the non-dominant forearm and dominant heel in boys (*n* 506) and girls (*n* 620) aged either 12 or 15 years. Habitual intakes of fruits and vegetables were assessed using the diet history method, and BMD was measured using dual energy x-ray absorptiometry. Height and weight were measured in all subjects, and questionnaires were used to assess smoking habits, usual alcohol consumption, physical activity patterns, parental socio-economic status, and length of time exposed to oestrogen (females only). A paediatrician assessed pubertal stage by visual assessment of secondary sexual characteristics. Users of nutritional supplements were excluded from all analyses.

	Boys (*n* 506)				Girls (*n* 620)			
	12-year-olds		15-year-olds		12-year-olds		15-year-olds	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Height (cm)	152	8	172	8	154	7	163	6
Weight (kg)	46	11	62	13	48	11	58	10
BMD forearm (g/cm^2)	0.338	0.05	0.386	0.06	0.328	0.05	0.408	0.06
BMD heel (g/cm^2)	0.470	0.08	0.557	0.09	0.453	0.08	0.491	0.08
Physical activity[a]	32	16	28	14	22	14	18	13
Calcium intake(mg/d)	1026	380	1136	447	862	305	860	337
Fruit intake (g/d)	138	147	138	152	168	151	161	155
Vegetable intake (g/d)	61	42	69	51	54	35	60	45
Vitamin C (mg/d)	85	53	89	57	99	54	86	52
Carotene (mg/d)	1760	1212	1890	1418	1749	1006	1665	1064
Vitamin D (μg/d)	2	2	3	3	2	1	2	2
Zinc (mg/d)	8	2	9	3	7	2	7	2
Potassium (mg/d)	3618	995	4144	1175	3168	822	3233	936
Magnesium (mg/d)	299	83	338	97	255	70	259	76
Fibre(g/d)	23	7	27	8	20	6	21	7

[a] Highest possible activity score = 100.

Using multiple linear regression analyses, high fruit intake was significantly positively associated with heel BMD in all girls (β 0.10; 95% CI 0.01–0.18). After adjusting for height, weight, age, pubertal stage, physical activity, alcohol intake and smoking, low fruit intake was significantly inversely related to heel BMD in all girls (β −0.09; 95% CI −0.18 to 0.00). The significant positive relationship between high fruit intake and heel BMD in girls remained after further adjustment for intakes of potassium, magnesium, calcium, vitamin C, vitamin D, zinc, fibre and carotene, as did the significant inverse association between low fruit intake and heel BMD in girls. No associations were observed between vegetable intake and BMD in either sex, or between fruit intake and BMD in boys. In conclusion, high intakes of fruit may be important to bone health in girls. It is possible that the nutrients contained in fruit, or the alkaline-forming properties of fruit mediate the body's acid–base balance. However, the findings of this observational study remain to be corroborated by intervention studies.

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Treatment of low bone mineral density in adult coeliac patients. By C. O'NEILL, L. O'SHAUGHNESSY and N.P. KENNEDY, *Unit of Nutrition and Dietetic Studies, Trinity Centre for Health Sciences, St. James's Hospital, Dublin 8, Ireland*

Undiagnosed coeliac disease is a common condition in Northern Europeans, with prevalence estimates varying between 1:300 and 1:110 from serological screening. The incidence of osteoporosis in diagnosed coeliac disease is almost 50% and bone mineral density (BMD) is decreased in up to 70% of patients (Bai *et al.* 1997; McFarlane *et al.* 1995). Approximately 20% of coeliacs also have a five-fold greater risk of bone fracture (McFarlane *et al.* 1995). The British Society of Gastroenterology has suggested guidelines for the management of osteoporosis in coeliac disease (Scott *et al.* 2000). However, a lack of published evidence concerning treatment of this condition in coeliac patients renders these somewhat arbitrary. The goals of treatment of osteoporosis in patients with coeliac disease are the same as those for the general population; that is, to stimulate bone formation, prevent bone loss and prevent fracture. Gluten exclusion, dietary supplementation with calcium and vitamin D, and other standard pharmaceutical measures are all accepted to have a role in prevention or treatment of low bone mineral density in coeliac disease. However, the effectiveness of supplements and medications has not been established in the context of variable compliance with a gluten-free diet. Further investigation of potential management approaches and an assessment of their efficacy is needed in order to allow more efficient intervention in this patient group.

This is a preliminary study of patients with low bone mineral density attending this coeliac clinic who had follow-up dual energy X-ray absorptiometry (DXA) scans in order to monitor change in BMD with intervention. We aimed to document which treatment interventions were employed and to observe subsequent changes in BMD, with a view to optimising future patient management. Ninety-two patients (aged 21–77 years) considered to be at increased risk of osteoporosis were assessed using DXA. If osteopenia or osteoporosis was found, advice or treatment was formulated accordingly. Twenty-seven patients underwent a second DXA scan approximately 2 years later. Of these, twenty-five (21 women and 4 men) attended an interview and comprised the follow-up group. A further forty-five coeliac patients (35 female and 10 male) were interviewed as a baseline group. Each patient completed an interviewer-assisted questionnaire concerning factors influencing BMD, which included questions on medical history, lifestyle factors, adherence to the gluten-free diet (GFD) and a food frequency questionnaire designed to evaluate calcium and vitamin D intake.

Interventions employed for osteopenia included hormone replacement therapy (HRT), serum oestrogen receptor modulators (SERMs), bisphosphonates, supplemental vitamin D and calcium and modification of lifestyle factors. Two of the least well followed interventions were adherence to GFD and increasing physical activity. BMD at both the hip and the spine increased or remained within expected limits for age in 60% of the follow-up group. Those with the lowest initial BMD had the greatest increase in BMD at both the spine and the hip at 18.9 and 9.8%, respectively. An increase in BMI correlated with a decrease in BMD at the lumbar spine (r=−0.695, P=0.002). Sunlight exposure was positively related to an increase in BMD (P=0.021). Use of calcium and vitamin D supplements was associated with a significant increase in BMD at the hip (P=0.008).

Low BMD in adult coeliacs is amenable to treatment with pharmacological and lifestyle interventions. This study highlights the need for promotion of inexpensive and easily employed interventions such as physical activity, adherence to a GFD and adequate calcium and vitamin D (endogenous and exogenous) for effective management of these patients. Osteoporosis and osteomalacia are both complications of coeliac disease that are at least partially preventable. With appropriate investigation and the development of an evidence-based strategy for management, a significant burden on the health of these patients may be lifted.

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Prevalence of osteoporosis and osteopenia among attenders at an eating disorder programme. By A Freyne[1], M Doyle[1], A Clarke[2] and M Darby[1] [1]*Department of Psychiatry, St Vincents University Hospital, Elm Park, Dublin 4,* [2]*Department of Community and Preventive Medicine, University College Dublin, Ireland*

Anorexia nervosa (AN) affects 0.5–1 % of adolescents and young women in the US (Pope *et al*. 1972). It has a chronic course in 50 % of cases. The onset of AN during a period of growth and before the attainment of peak bone mass is a factor in the development of osteoporosis, which is now recognised as a major complication of the disease. Reduced bone mineral density (BMD) in young anorectics may cause irreversible developmental and growth retardation, and in chronic patients there is a higher risk of pathological fractures (Lucas *et al*. 1999). With the greater availability of assessment methods such as dual energy x-ray absorption (DEXA) scanning to measure bone density, it was felt opportune to review patients attending the eating disorders programme in St Vincent's University Hospital to ascertain their levels of bone mineral density.

Subjects attending the eating disorder programme in St Vincents University Hospital with a primary diagnosis of anorexia nervosa or bulimia nervosa with a previous history of anorexia nervosa were identified over a 6-month period.

Inclusion criteria included a history of amenorrhoea for at least 3 months or other risk factors for osteoporosis such as steroid therapy. Both inpatients and outpatients were included. Once these criteria were met there were no exclusion criteria. Participants completed a questionnaire in which sociodemographic data and medical history was obtained. A dietary history was taken and daily dietary intake of calcium and other nutrients and the use of calcium and oestrogen supplements was calculated. Subjects were also asked about their awareness of osteoporosis. Bone density was measured using DEXA scans of the lumbar spine and femur. Osteoporosis and osteopenia were defined according to World Health Organisation guidelines (WHO 1994). Differences between groups were compared using ANOVA for parametric and chi squared tests for non-parametric variables.

Twenty-nine subjects were recruited, of whom twenty-seven were female. They ranged in age from 17 to 54 with a mean of 25 years. BMI for the group ranged from 12 to 22.2 with a mean of 17.5. Only three subjects (10.3%) had normal bone scans of both lumbar spine and femur. The distribution of osteoporosis and osteopenia at lumbar spine and femur is seen in the table.

	Lumbar spine		**Femur**		**Both sites**	
	N	**(%)**	**N**	**(%)**	**N**	**(%)**
Osteoporosis	7	(24.1)	7	(24.1)	3	(10.3)
Osteopenia	18	(62.1)	12	(41.4)	10	(34.4)

Clinical factors were examined to see if they correlated with diminished bone density. There was no correlation between duration of amenorrhoea, use of oestrogen or calcium supplements and bone density. The only factor differentiating those with low bone density and normal bone density was weight – mean BMI for those with normal bone density at both sites was 20.3, compared with 17.3 for all others – this is a statistically significant difference.

The implication of this for clinical management is to emphasise the importance of weight gain. There is no evidence that prescribing oestrogen is effective treatment against osteoporosis. However we now routinely prescribe calcium and Vitamin D supplementation for eating disorder patients with diminished bone density in addition to supporting weight gain. As only a minority of subjects with eating disorders is referred to specialist services, it is vital that assessing clinicians in primary care are aware of the high possibility of osteopenia or osteoporosis and investigate appropriately. Objective evidence of deficient bone density may act as an incentive to patients with eating disorders, and enable them to accept treatment which they may otherwise deem unnecessary.

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Independent and combined effects of 17 β-oestradiol and 1,25 dihydroxycholeciferol on transepithelial calcium transport in Caco-2 cells maintained in an oestrogen-depleted environment. By A.A. COTTER[1] and K.D. CASHMAN[1,2], [1]*Department of Food and Nutritional Sciences and* [2]*Department of Medicine, University College Cork, Ireland*

Oestrogen therapy remains the mainstay for prevention of bone loss in postmenopausal women (Gallagher, 2001). Oestrogen therapy also corrects the diminished efficiency of intestinal Ca absorption associated with ovarian hormone deficiency (Heaney *et al*. 1978). The mechanism by which 17 β-oestradiol stimulates Ca absorption is unclear. 1,25 dihydroxycholeciferol (1,25 D_3), the biologically active, steroid hormone-like metabolite of vitamin D, has long been established as a critical regulatory factor for intestinal Ca absorption (Giuliano & Wood, 1991). It is not clear whether intestinal Ca absorption is influenced by an interaction between these two steroid hormones. Therefore, the aim of this study was to investigate the independent and combined effects of 1,25 D_3 and 17 β-oestradiol (17 β-E_2) on transepithelial Ca transport in the human Caco-2 intestinal-like cell model. Caco-2 cells were seeded onto permeable filter supports and allowed to differentiate into a highly organized structure similar to that of the intestinal wall. On day 21, the Caco-2 monolayers (*n* 9 per treatment), grown in oestrogen-depleted media, were exposed to media containing dimethyl sulphoxide (DMSO) alone (control) or with 10 nM 1,25 D_3, 10 nM 17 β-E_2, or 10 nM 1,25 D_3 plus 10 nM 17 β-E_2, for 48 h. After exposure, transepithelial transport of ^{45}Ca and fluorescein transport (a marker of paracellular diffusion), transcellular Ca transport and transepithelial electrical resistance (TEER; an index of monolayer permeability) were measured. mRNA levels for the oestrogen receptor α (ORα) and ORβ were assessed using a PCR technique.

Treatment…	−1,25 D_3		+1,25 D_3			Statistical significance		
	−17 β-E_2	+17 β-E_2	−17 β-E_2	+17 β-E_2	Pooled	of variance ratio (*P*), effects of:		
	(*n* 9)	(*n* 9)	(*n* 9)	(*n* 9)	SEM	1,25 D_3	17 β-E_2	1,25 D_3 × 17 β-E_2
Ca transport:								
Total transepithelial (%/h)	0.70	0.69	1.19	1.28	0.08	<0.0001	0.673	0.534
Transcellular (nmol/well/min)	0.12	0.11	0.23	0.25	0.07	<0.0001	0.903	0.212
Paracellular (%/h)	0.13	0.15	0.13	0.12	0.01	0.315	0.878	0.286
TEER (Ω.cm^2)	2531	2758	2368	2243	108	0.004	0.638	0.115

Caco-2 cells expressed mRNA for the ORβ, but not for the ORα. As expected, 1,25 D_3 stimulated total (P<0.001) transepithelial Ca transport in Caco-2 cells, by upregulating transcellular (P<0.001) transport. On the other hand, 17 β-E_2 had no effect on Ca transport, nor did it augment the stimulatory effect of 1,25 D_3. Paracellular transport was unaffected by any treatment. In conclusion, while 1,25 D_3 stimulated total transepithelial Ca transport in Caco-2 cells grown in an oestrogen-depleted environment, 17 β-E_2-treatment had no regulatory effect on Ca transport. The lack of stimulatory effect of 17 β-E_2 on Ca transport in these cells may be due to the lack of ORα, which is present in normal human mucosal epithelium. Therefore, further research is needed to clarify how oestrogen therapy enhances intestinal Ca absorption.

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Conjugated linoleic acid, a molecular determinant of cholesterol homeostasis in THP-1 derived macrophages? By S.M. WELDON, M.J. GIBNEY and H.M. ROCHE, *Unit of Nutrition, Department of Clinical Medicine, Trinity Centre for Health Sciences, St. James's Hospital, Dublin 8, Ireland*

In animal models, conjugated linoleic acid (CLA) can prevent diet-induced atherosclerosis and initiate the regression of established atherosclerosis (Kritchevsky *et al.* 2000). Peroxisome proliferator-activated receptors (PPARs) control the expression of a number of genes involved in cholesterol homeostasis in macrophages. The net effect of these agonists on macrophage lipid accumulation appears to be negative, with repression of the class A scavenger receptor SR-A balancing the induction of the class B scavenger receptor CD36 (Moore *et al.* 2001) and stimulation of cholesterol efflux pathways of ATP-binding cassette (ABC) transporters e.g. ABCA1, via induction of the related nuclear transcription factor LXR-α (Chinetti *et al.* 2001). Given that CLA is a PPAR ligand, this study investigated whether the anti-atherogenic effects of CLA are as a result of altered cholesterol transport in the macrophage.

THP-1 derived macrophages were treated with 100 μM of linoleic acid, *cis*-9,*trans*-11 CLA (*c*9,*t*11 CLA), *trans*-10,*cis*-12 CLA (*t*10,*c*12 CLA), stearic acid, 1 μM of the pharmacological PPAR-γ ligand rosiglitazone and 50 μM of the pharmacological PPAR-α ligand Wy14643 for 48 h. Total RNA was extracted and the mRNA levels of CD36, SR-AI, ABCA1, LXR-α, PPAR-α and PPAR-γ were quantified using TaqMan real-time PCR analysis. Protein levels of CD36 were analysed by flow cytometry and fluorescent microscopy. To assess the effects of CLA on macrophage lipid accumulation and efflux, THP-1 macrophages were loaded with cholesterol by incubating with AcLDL for 48hr. ApoA-I mediated efflux studies were performed by incubating lipid-loaded cells with or without apoA-I for 24hr. Intracellular total and free cholesterol concentrations were assayed using enzymatic assays. Esterified cholesterol was measured as the difference between total and free cholesterol. Statistical analysis was completed using one-way ANOVA.

In the macrophages *c*9,*t*11 and *t*10,*c*12 CLA had no significant effect on the expression of important genes involved in cholesterol homeostasis with the exception of CD36 (182% and 117% relative to vehicle control, respectively. $P<0.01$). The pharmacological ligands increased the mRNA expression of CD36 ($P<0.001$) and LXR-α ($P<0.05$), but only Wy14643 significantly increased ABCA1 mRNA ($P<0.001$). In foam cells, CD36 mRNA expression was significantly increased by both *c*9,*t*11 and *t*10,*c*12 CLA (40% and 87% relative to vehicle control, respectively. $P<0.05$) and pharmacological PPAR ligands ($P<0.001$). In addition, Wy14643 significantly increased ABCA1 mRNA expression ($P<0.001$), while a decrease in SR-AI mRNA levels was observed ($P<0.05$). No treatment exerted any significant effect on PPAR mRNA. Analysis of CD36 protein expression showed that the pharmacological agents and CLA increased CD36 expression. Cholesterol accumulation, measured by intracellular total, free, or esterified cholesterol concentrations, was not significantly affected by the CLA isomers. Although apoA-I stimulated cholesterol export, this efflux was not further enhanced by either CLA isomer.

Overall these findings indicate that the anti-atherogenic effects of CLA seen *in vivo* cannot be explained by altered cholesterol homeostasis in this cell model system.

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The involvement of caspase activation and PARP degradation in 7β-hydroxycholesterol-induced apoptosis. By L. RYAN, Y.C. O'CALLAGHAN and N.M. O'BRIEN, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Oxysterols are products of cholesterol oxidation that may be produced endogenously or absorbed from the diet and are commonly found in highly processed foods of animal origin. The oxysterol 7β-hydroxycholesterol (7β-OH) has been shown to induce apoptosis in a number of cell lines. The mechanism through which this oxysterol induces cell death has yet to be fully elucidated. However, many apoptotic pathways centre on the activation of caspase-3 which is the key executioner protease of apoptosis. The objective of the present study was to investigate the pathway in 7β-OH-induced apoptosis, over 48 h. U937 cells, a human monocytic blood cell line known to undergo apoptosis upon treatment with 7β-OH, were used in this study. We monitored alterations in caspase-3 activity, the expression of caspases 3, 8 and 9 and cleavage of the caspase-3 substrate poly ADP-ribose polymerase (PARP). Cell viability and apoptosis were also assessed at each of the time-points.

U937 cells were adjusted to a density of 2×10^5 cells/ml in RPMI 1640 medium supplemented with 25 ml/l fetal bovine serum. Cells were treated with 30 μM 7β-OH and incubated at 37°C, air:CO_2 (95:5). Control cells were treated with an equal volume of ethanol. Caspase-3 activity was determined, using a method previously described by Huigsloot *et al.* (2001), at 3, 6, 12, 24 and 48 h and expressed as fold increase relative to control. Analysis of caspase-3, -8 and -9 activation and of PARP degradation was performed by Western blot. Viability was assessed by the fluorescein diacetate-ethidium bromide assay and apoptotic nuclei were quantified following staining with Hoechst 33342.

	7β-Hydroxycholesterol									
	3 h		6 h		12 h		24 h		48 h	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Viable cells (% control)	97.3	1.3	98.4	0.8	85.8	5.0	75.7*	1.4	11.5*	2.3
Apoptotic nuclei (fold increase)	1.2	0.3	1.5	0.4	1.5	0.5	3.5*	1.1	8.0*	3.0
Caspase-3 activity (fold increase)	1.0	0.3	1.0	0.2	1.4	0.2	4.0*	0.3	5.4*	1.7

n=3 independent experiments, *$P<0.05$ = significantly different from control cells.

There was no significant ($P<0.05$) decrease in viability of U937 cells treated with 7β-OH until the 24 h time-point and this was coupled with a significant increase in apoptotic nuclei. Caspase-3 activity increased significantly after 24 h incubation with 7β-OH. The cleaved, active form of caspase-9, an initiator caspase, was visualized by Western blot after 9 h of treatment, caspase-3 expression occurred after 12 h and degradation of PARP was evident after 24 h. Caspase-8 did not appear to play an active role in this particular apoptotic pathway. In conclusion these results suggest that 7β-OH-induced apoptosis proceeds via activation of caspase-9 with subsequent activation of the effector caspase, caspase-3, ultimately leading to cleavage of the caspase-3 substrate PARP.

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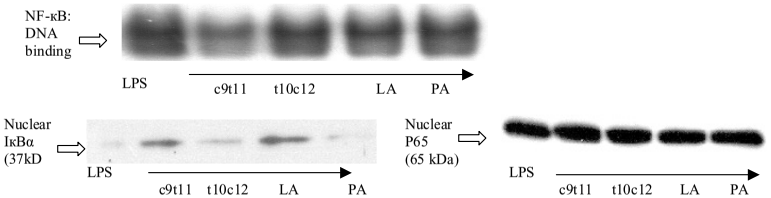
Cis-9, _trans_-11 conjugated linoleic acid and linoleic acid modulate the nuclear factor-κB family of transcription factors. By A.P. NUGENT, M.J. GIBNEY and H.M. ROCHE, *Unit of Nutrition, Department of Clinical Medicine, Trinity Centre for Health Sciences, St. James's Hospital, Dublin 8, Ireland*

The influence of various fatty acids on immune function and the inflammatory response has been extensively researched. However, less is known about the molecular mechanisms underlying these immuno-modulatory effects. This study investigates how the dietary fatty acids, *cis*-9, *trans*-11 (*c*9,*t*11) CLA, *trans*-10, *cis*-12 (*t*10,*c*12) CLA, linoleic acid (LA) and palmitic acid (PA), modulate the nuclear factor-κB (NF-κB) family of transcriptional activator proteins (Karin, 2000).

Investigations were conducted in peripheral blood mononuclear cells (PBMCs) isolated from healthy donors and in the transformed monocytic cell line, THP-1. Both cell types were cultured for 48 h in the presence of 10% heat inactivated fetal calf serum and fatty acid dissolved in DMSO. The final cell density for both cell types was 8×10^6/well. Experiments were set up in duplicate to ascertain the effects of fatty acids on NF-κB activity, IκBα and p65 expression in resting and activated cells. At the end of this 48 h period, PHA (10 μg/ml) was added to the one set of the PBMCs and culture continued for a further 2 h. For THP-1s, LPS (1 μg/ml) was added and the culture continued for 30–60 min. Controls included cells alone or cells and PHA or LPS. Final concentrations of DMSO were <0.1% in all conditions and did not have a significant effect on any of the experiments. Fatty acids used included the CLA isomers *cis*-9, *trans*-11 CLA (*c*9,*t*11), *trans*-10, *cis*-12 CLA (*t*10,*c*12), linoleic acid (LA) and palmitic acid (PA). Fatty acids were added at 100 μM. The binding of NF-κB to κB enhancer elements on DNA was measured by electrophoretic mobility shift assays (EMSAs) in resting and activated PBMCs and THP-1s. The antibodies IκBα (C-21), *sc*-371 and p65 (C-20), *sc*-372 (Santa Cruz biotechnology, USA) were used to detect IκBα and p65 protein expression in the nuclear and cytoplasmic fractions of THP-1s and in the cytoplasmic extracts of PBMCs. Quantitation of both EMSA and Western blotting autoradiographic bands was performed with the aid of the Kodak Image Station 440cf and analysed with the Kodak 1D analysis software. Four to six individual experiments were conducted and statistical analysis performed by ANOVA with LSD. Shown below are representative blots from typical EMSA and nuclear IκBα and p65 western blots in the nuclear extracts of LPS-activated THP-1s.



Fatty acids downregulated NF-κB:DNA binding in THP-1 monocytes and human PBMCs. Results were more apparent in resting cells. A significant ($P<0.05$) inhibition of NF-κB activity in the order of *c*9,*t*11 CLA > LA > *t*10,*c*12 CLA was observed in THP-1s. In resting PBMCs all fatty acids significantly ($P<0.05$) decreased NF-κB:DNA binding, with the *c*9,*t*11 CLA isomer being most effective. The degree of downregulation was less pronounced in activated cells; however, the *c*9,*t*11 CLA isomer significantly ($P<0.05$) reduced NF-κB activity in LPS-activated THP-1s. Both *c*9,*t*11 CLA and LA significantly ($P<0.03$) increased nuclear IκBα expression in resting and LPS- activated THP-1s. None of the fatty acids affected cytoplasmic IκBα expression in THP-1 monocytes. Cytoplasmic IκBα expression decreased in resting PBMCs co-cultured with fatty acids, however this effect was lost upon PHA-induced activation. Fatty acids did not affect cytoplasmic or nuclear p65 expression in PBMCs.

These results show that *c*9,*t*11 CLA and LA decrease NF-κB transcriptional activity. This reduction in NF-κB:DNA binding was accompanied by an increase in nuclear IκBα expression. Further research is required to elucidate exactly how these fatty acids are mediating their effects on NF-κB signalling (e.g. by affecting phosphorylation status or kinase activity).

Karin M (2001) Annual Review of Immunology **18**, 621–663.

The influence of composite foods on estimates of habitual meat intakes. By M. COSGROVE, A. FLYNN and M. KIELY, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Individual quantitative data on the consumption of commonly consumed foods are central to the development of food-based dietary guidelines and for food safety assessments. This is particularly true of meat, which has received particular attention due to the established risk of variant Creutzfeldt-Jakob Disease (vCJD) in humans from bovine spongiform encephalitis (BSE) outbreaks in cattle (Morabia *et al*. 1999), and other microbial pathogens. In addition, high consumption of meat and meat components have been negatively associated with an increased risk for cardiovascular disease and some cancers (Willett *et al*. 1990; Hu *et al*. 1999). Most food intake assessments are based on aggregated data, where individual components of composite foods are not quantified on an individual basis. The aim of the current study was to determine the size of the bias in meat intake estimates from aggregated food consumption data.

Meat intake was estimated using data from the North/South Ireland Food Consumption Survey (IUNA, 2001), which estimated habitual food intake using a 7 d food diary in a representative sample of Irish adults aged 18–64 years. From 3060 foods recorded, 670 foods were meat and meat product foods, and seventy-two were typically non-meat foods that contained meat (e.g. meat on pizzas, in soups and sauces). Meat was consumed either as a cut of meat, as part of a composite food or as a meat product. Cuts of meat accounted for 320/742 (43%) meat codes, and calculations were required to exclude the weight of bone for forty cuts. The remaining 422 composite foods were disaggregated to quantify the weight of the meat component.

Using the aggregated database, the intake of meat and meat products (including non-meat components) for consumers (98.3%) was 192 g/d. Following the disaggregation of meat from composite foods, the intake of meat for consumers (98.5%) was 134 g/d, representing a decrease in the estimate of total meat intake of 57 g/d (30%).

	Aggregated†			Disaggregated*			Difference	
Type of Meat	% consumers	Mean	SD	% consumers	Mean	SD	(g/d)	(%)
Bacon, ham, lamb & pork	92.2	57.6	46	95.4	52.9	39	4.7	8.2
Beef	77.8	68.1	55	79.9	39.4	32	28.7	**42.2**
Chicken	79.7	53.8	45	89.1	36.7	27	17.1	31.8
Offal	4.4	20.0	15	8.1	13.6	11	6.4	32.0
Burger	27.2	24.0	20	27.2	12.7	9	11.3	**47.0**
Sausage	62.0	15.8	13	63.7	16.1	13	-0.3	-1.9
Meat products	72.1	30.8	29	29.6	10.0	9	20.8	**67.4**
Total meat	**98.3**	**191.5**	**95**	**98.5**	**134.3**	**65**	**57.2**	**29.9**

† Includes non-meat components (e.g. vegetables and sauces) coded with the meat. * Meat only data.

Overall, a quarter of total meat was consumed as part of a composite dish. The principal types of meat consumed as part of a dish were burgers (63%), beef (41%), chicken (39%) and offal (38%). The relative proportion of meat consumed as part of a dish was higher in women (29%) than in men (23%) with the exception for burgers (men consumed 67% and women 55%). Younger adults (18–35 years) were more likely to consume meat as part of a dish (31% of total meat) than older adults (36–50 years), particularly beef (46%) and chicken (45%).

In conclusion, meat is a key food component of the Irish diet that >98% choose to consume. Intake estimates of meat are substantially overestimated when composite foods are not disaggregated to quantify the meat component separately, particularly for meat products, burgers and beef. This could have serious implications in epidemiological studies of food intakes and disease patterns and is an important factor in the design of food consumption databases.

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Irish Universities Nutrition Alliance (2001) *North/South Ireland Food Consumption Survey Database.*www.iuna.net
Morabia A, Bernstein MS, Heritier S & Beer-Borst S (1999) *European Journal of Clinical Nutrition* **53**(2), 158–163.
Willett WC, Stampfer MJ, Colditz GA, Rosner BA & Speizer FE (1990) *New England Journal of Medicine* **323**, 1664–1672.

An exploratory investigation into the diets of student smokers and non-smokers. By M. SMITH and J.R.A. REEKIE, *Division of Food and Biological Sciences, School of Applied Sciences, Ellison Terrace, Northumbria University, Newcastle upon Tyne NE1 8ST*

One-third of the 152 480 deaths from cancer in the UK in 1999 were the result of smoking (Hastings, 2001). Diet is also a contributory factor for both cancer (Frankish, 2003) and coronary heart disease (Hu & Willett, 2002). A small scale exploratory survey was designed to determine whether students who smoked had a different dietary profile than non-smoking students. A lifestyle questionnaire and a 24 hour dietary recall were completed for 17 female non-smokers (FNS), 20 female smokers (FS), 20 male non-smokers (MNS) and 20 male smokers (MS). Height, weight and blood pressure were measured and a self reported physical activity level was also obtained.

No significant differences (P<0.05) were found between the two female groups or the two male groups for mean weight, body mass index, age and dietary energy intake from alcohol, and between the male and female groups for number of cigarettes smoked per day. Physical activity levels, obtained from self reported rating scales, were not significantly different for MNS and MS, but were so for FMS and FS, (P=0.0275), where smokers reported the lower physical activity. All students in the sample were aged between 18 and 23 years and had blood pressures within the normal range for their age group.

Both female and male smokers consumed more carbohydrate, fat, saturated fatty acid (SFA) and energy than non-smokers, but this was mainly only significant (P) for males.

Nutrient quantity	FNS Mean	% SE	FS Mean	% SE	*P*	MNS Mean	% SE	MS Mean	% SE	*P*
Protein (g)	61.3	11.0	64.9	9.8	0.350	68.3	7.3	77.9	8.1	0881
Carbohydrate (g)	194.8	10.3	224.8	5.9	0.112	244.7	5.7	307.1	7.7	0.015*
Fat (g)	50.6	12.1	66.3	11.4	0.058	67.9	9.8	115.4	9.5	0.000Ψ
SFA (g)	16.3	13.8	24.1	13.3	0.027 *	25.5	12.6	34.1	10.8	0.043*
Energy (kJ)	6451	9.8	7637	7.4	0.085	8340	6.3	10900	7.4	0.006Ψ
Energy %fat	30.1	6.3	31.4	7.3	0.335	30.4	5.9	39.3	5.1	0.001Ψ
Energy %SFA	10.0	12.6	11.4	9.5	0.201	11.5	8.4	12.1	9.2	0.354

Non-smokers significantly greater than smokers: * P<0.05, Ψ P<0.01.

Fruit and vegetable consumption was measured by a 6-point itemised rating scale in the lifestyle questionnaire, although this included potatoes as a vegetable. For both females (P=0.006) and males (P=0.032) non-smokers reported consuming significantly more portions of 'fruit and vegetables' than smokers (FNS, 3–6 portions per week; FS, 1–2; MNS, 3–6; MS, 1–2). There was no significant difference (P=0.809) between female (FNS and FS) (3-6 portions per week) and male (MNS and MS) (3–6) 'fruit and vegetable' self reported consumption ratings. Fruit and vegetable consumption, excluding potatoes, from 24 hr dietary recalls, did not support the significant difference between smokers and non-smokers (FNS, 1.9 portions per week; FS, 1.8; MNS, 2.0; MS, 1.7).

Results of this survey suggest that student smokers consume more energy, carbohydrate and fat than non-smokers and that smokers, especially male smokers, tend to have a less healthy diet than non-smokers. The greater intake of total fat and saturated fatty acids (SFA) is of particular concern.

Frankish H (2003) 15 million new cancer cases per year by 2020 says WHO *The Lancet* **361**, 1278.
Hastings G (2001) *Briefing Sheet Smoking: The Cancer Research Campaign.* The Cancer Research Campaign.
Hu FB and Willett (2002) Optimal diets for the prevention of coronary heart disease *The Journal of the American Medical Association* **288**, 2569–2578.

Vitamin C and E: main and interactive acute effects on DNA damage in human lymphocytes measured by the Comet Assay. By S.W. CHOI[1,3], J.J. STRAIN[2], B. HANNIGAN[3] and I.F.F. BENZIE[1], [1]*Ageing and Health Section, The Hong Kong Polytechnic University, Kowloon, Hong Kong SAR,* [2]*Northern Ireland Centre for Food and Health (NICHE), and* [3]*Faculty of Life and Health Sciences, University of Ulster, Coleraine, BT52 1SA*

Dietary antioxidants such as vitamins C and E have been shown to scavenge reactive oxygen species (ROS) *in vitro*, and may protect DNA from oxidant challenge (Brennan *et al*. 2000) However, while epidemiological evidence of benefit is strong, intervention studies have not shown the expected health benefits to date, and results using biomarkers for surrogate endpoints have been conflicting, resulting in doubt as to the role of dietary antioxidants in health promotion (Fang *et al*. 2002). This double-blind acute supplementation study of multiple cross-over design aimed to investigate the main and interactive acute effects of vitamins C and E on nuclear DNA damage, and was conducted on twelve healthy non-smoking volunteers (three men, nine women, aged 24–38 years). Subjects were allocated, on a non-selective basis (stratified by number), to one of four treatments on each of four visits to our laboratory in Hong Kong. Treatments were: (1) 500 mg vitamin C (ascorbic acid) and 400 IU vitamin E (as α-tocopheryl succinate), (2) 500 mg vitamin C and placebo vitamin E; (3) 400 IU vitamin E and placebo vitamin C; (4) double placebo. There were at least 10 d between each treatment; all subjects took all four treatments. For each treatment, venous blood was taken at time 0 (fasting), 90 and 180 min post-ingestion. Subjects remained fasting during this time except for the supplement and sips of water. Subjects returned the following morning, and a fasting 24 h post-supplement blood sample was taken. Within 2 h of blood collection, lymphocytes were harvested using gradient centrifugation, and DNA baseline damage and resistance to a standardised oxidant challenge (induced with hydrogen peroxide) was assessed using the Comet Assay (Collins, 2002). Results pre- and post- each treatment were investigated using ANOVA, and a 5% significance level sought. Results, as %DNA comet tail (mean (SEM); n=12) in challenged and unchallenged cells at different times after supplement ingestion, are presented in the Table.

Treatment	0 min; no challenge	0 min; 15 μM H_2O_2	+90 min; no challenge	+90 min 15 μM H2O2	+180 min no challenge	+180 min 15 μM H_2O_2	+24 h no challenge	+24 h 15 μm H_2O_2
Vit C 500 mg +	8.1	28.6	9.9	34.5	8.1	32.7	8.1	33.7
Vit E 400 IU	(1.0)	(1.5)	(0.98)	(2.1)	(0.95)	(1.1)	(0.85)	(1.1)
Vit C 500 mg +	6.0	28.4	9.9	31.7	7.0	35.3	7.4	33.92
Placebo E	(0.82)	(1.9)	(1.0)	(1.7)	(0.70)	(1.6)	(0.69)	(1.1)
Placebo C +	7.0	28.2	11.0	29.6	6.91	33.7	9.5	35.2
Vit E 400 IU	(1.1)	(1.7)	(0.9)	(2.2)	(0.94)	(1.2)	(1.0)	(1.8)
Placebo C +	6.6	28.5	11.3	36.3	8.42	33.6	8.4	36.3
Placebo E	(0.85)	(1.5)	(0.93)	(2.13)	(0.82)	(1.68)	(1.1)	(1.7)

None of the four treatments was associated with a noticeable effect in terms of either a decrease in baseline DNA damage or an increase in resistance to oxidant challenge. Neither was there any evidence of a damaging pro-oxidant effect. The results, therefore, showed no significant main or interactive effects in the short term after ingestion of a single dose of vitamin C and/or E. These data support those of Huang *et al.* (2000) who found no main or interactive effects on a different biomarker of DNA damage (urinary 8-OHdG) after 2 months' supplementation with vitamin C and/or E in the same dosage as used in our study. Lack of response may be owing to the fact that all subjects were of adequate vitamin C status (mean 63 μmol/l) at baseline. In addition, a single dose only was ingested. Acute effects may be seen in individuals with poor antioxidant status, or in those suffering from oxidative stress, and a considerable duration or higher dose of supplementation may be required for healthy, vitamin C-replete subjects (Griffiths & Lunec, 2001). Moreover, it is possible that vitamin C and E exert separate or interactive protective effects unrelated to DNA strand breaks. However, on the basis of the data presented here, there is no evidence of either beneficial or detrimental effects of single doses of vitamin C, vitamin E or both on DNA damage as measured by strand breaks in normal, healthy adults.

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Collins AR (2002) *Methods in Molecular Biology* **203**, 163–177.
Fang YZ, Yang S & Wu G (2002) *Nutrition* **18**, 872–879.
Griffiths HR & Lunec J (2001) *Environmental Toxicology and Pharmacology* **10**, 173–182.
Huang HY, Helzlsouer KJ & Appel LJ (2000) *Cancer Epidemiology Biomarkers and Prevention* **9**, 647–652.

Bilirubin and cardiovascular risk in the PRIME study. By J.A. TROUGHTON[1], J.V. WOODSIDE[1], I.S. YOUNG[1], C.C. PATTERSON[1], D. ARVEILER[2], P. AMOUYEL[3], J. FERRIERES[4], P. DUCIMETRIERE[5], J.W.G. YARNELL[1] and A. EVANS[1], on behalf of the prime investigators, [1]*School of Clinical Medicine, Queen's University Belfast, Belfast BT12 6BJ,* [2]*The Strasbourg MONICA Project, Department of Epidemiology and Public Health, Faculty of Medicine, Strasbourg, France,* [3]*The Lille MONICA Project, INSERM U508, Pasteur Institute of Lille, Lille, France,* [4]*The Toulouse MONICA Project, INSERM U588, Department of Epidemiology, Paul Sabatier-Toulouse Purpan University, Toulouse, France and* [5]*The Coordinating Centre, INSERM U258, Hôpital Paul Brousse, Villejuif, France*

Classical risk factors for coronary heart disease (CHD) fail to explain the large incidence gradient in CHD between Northern Ireland and France. The PRIME study (prospective epidemiological study of myocardial infarction) is a multi-centre prospective study that aims to investigate novel risk factors in these populations. One potential risk factor is bilirubin, a bile pigment formed during haem catabolism, which is known to prevent oxidation of low-density lipoprotein but only in the presence of α-tocopherol.

In total, 10 592 men aged 50–59 years were recruited between 1991 and 1993 and examined for evidence of CHD at baseline. Subjects were followed annually by questionnaire and a 5-year follow-up was completed. There were 138 cases identified in Belfast and 197 in France. Each case was matched to 2 controls who were study participants of the same age (±3 years), recruited in the same centre on the same day (±2 days) as the corresponding case and were free of CHD on the date of the ischaemic event of the case. The present analysis included 216 cases and 434 controls. Total serum bilirubin was measured using an automated colorimetric assay.

There was a significant positive association between bilirubin and HDL-cholesterol and a significant negative association between bilirubin and triacylglycerols in both cases and controls. Cases had significantly lower bilirubin concentration than controls (geometric mean (IQ range); cases 7.95 (5.32, 12.33); controls 9.07 (6.16, 12.76) μmol/l; P=0.005). There were no significant differences in bilirubin concentrations between the three French centres, or when the three French centres were pooled and compared with the Belfast centre. This was true for both cases and controls. After adjustment for other cardiovascular risk factors, there appeared to be a U-shaped pattern between CHD risk and bilirubin concentration, with CHD risk significantly lower for bilirubin concentrations in the 3rd (P=0.011) and 4th quintiles (0.060), compared with the 1st (lowest) quintile, but there was no significant difference in CHD risk for bilirubin concentrations in the 2nd (P=0.30) and 5th quintiles (P=0.29) compared with the 1st (see table). Bilirubin concentration did not differ according to weekly alcohol intake category (cases P=0.76; controls P=0.33) or smoking status (cases P=0.13; controls P=0.49). However in pooled cases and controls, current smokers had significantly lower bilirubin concentrations compared with combined never and ex-smokers (P=0.029).

This study suggests that a U-shaped relationship exists between bilirubin and CHD incidence in middle-aged men. Additional studies are required to determine whether antioxidant and cholesterol clearance mechanisms have a role in determining bilirubin concentration.

Conditional logistic regression analysis showing the unadjusted and adjusted odds ratio of CHD risk for fifths of bilirubin based on control values.

Bilirubin (μmol/l)	Unadjusted OR (95% CI)	P-value	Adjusted Model 1* OR (95% CI)	P-value
< 5.66	1.00		1.00	
5.67-8.22	0.68 (0.40, 1.17)	0.16	0.70 (0.36, 1.37)	0.30
8.23-10.15	0.36 (0.20, 0.64)	0.001	0.37 (0.17, 0.80)	0.01
10.16-13.83	0.57 (0.34, 0.96)	0.04	0.52 (0.27, 1.03)	0.06
13.84-53.62	0.55 (0.32, 0.94)	0.03	0.68, (0.34, 1.39)	0.29

Unadjusted model, test for quadratic trend, χ^2=11.3, df=2; P=0.004.
Model 1: adjusted for model 1 variables (case-control triplet, WHR, DBP, smoking, total cholesterol, HDL cholesterol and triglycerides) and glucose, alcohol, physical activity, apoB, fibrinogen, GPx and cystatin C. Test for quadratic trend, χ^2 =6.80, df=2; P =0.035.

The protective effects of antioxidants against UVA-induced DNA damage. By Y.C. O'CALLAGHAN, A.E. O'DWYER, M.M. HODNETT and N.M. O'BRIEN, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Exposure to UVA and UVB radiation from sunlight is the primary cause of skin cancer. Both UVA and UVB have been shown to induce DNA damage *in vitro*. However, nucleotides do not absorb light in the UVA range (320–400 nm); therefore, the DNA-damaging effects of UVA-irradiation are indirect. UVA is absorbed intracellularly by riboflavin and NADH and causes the generation of reactive oxygen species which attack DNA, resulting in the formation of lesions and oxidised bases. Antioxidants have been shown to protect DNA from UV-induced damage (Lehmann *et al.* 1998).

The objective of the present study was to assess the ability of the antioxidants α-tocopherol, γ-tocopherol, β-carotene and astaxanthin to protect against UVA-induced DNA single-strand breaks, as measured by the Comet Assay. The cell line employed was the human foreskin fibroblast cell line (HFFF2). Cells were seeded at a density of 2×10^4 cells/cm^2 and allowed to adhere overnight. Cells were treated with 10 μM of either α- or γ-tocopherol, 5 μM β-carotene or 5 μM astaxanthin and incubated at 37°C, air:CO_2 (95:5) for 24 h. Cells were then harvested, suspended in agarose and placed on a microscope slide. The slides were exposed to light in the UVA range (366 nm) for 1 h, while on ice and shielded with glass plates to prevent contamination with UVB. Samples were processed for the Comet Assay, viability was assessed by fluorescein diacetate–ethidium bromide staining.

	Viability (%)		Olive tail moment (arbitrary units)	
	Mean	SD	Mean	SE
Untreated control	61.6	5.8	1.0	0.2
UV-treated control	66.5	2.8	9.8	1.5
α-tocopherol	63.7	4.7	4.9*	0.9
α-tocopherol and β-carotene	65.3	0.2	7.1*	0.9
γ-tocopherol	66.9	0.6	7.2	1.2
γ-tocopherol and β-carotene	64.1	0.2	7.9	0.6
β-carotene	69.8	1.1	7.9	1.2
Astaxanthin	66.4	1.5	6.6*	1.0

$n\geq3$ independent experiments,*$P<0.05$ = significantly different from UV-treated control cells.

Cell viability did not differ significantly ($P<0.05$) from the untreated control under any of the treatments. DNA damage was quantified by image analysis and results were expressed as the olive tail moment (tail length × tail intensity). There was an approximately tenfold increase in DNA damage following exposure to UVA. α-Tocopherol was the most protective of the antioxidants investigated in the present study. Astaxanthin also significantly ($P<0.05$) protected HFFF2 cells from UVA-induced DNA damage. However β-carotene and γ-tocopherol did not significantly ($P<0.05$) alter DNA damage in UVA-treated cells. When cells were pre-treated with both β-carotene and α-tocopherol simultaneously, the protective effect was less than that observed for α-tocopherol alone. Therefore, while β-carotene or γ-tocopherol did not appear to protect, α-tocopherol significantly decreased UVA-induced DNA damage, at concentrations which may easily be attained in the diet.

Lehmann J, Pollet D, Peker S, Steinkraus V & Hoppe U (1998) *Mutation Research* **407**, 97–108.

Effects of oat bran intake on postprandial antioxidant capacity and total phenolic content of plasma and urine. By A.M. LEE, R.K. BEATTIE, J.J. STRAIN and R.W. WELCH, *Northern Ireland Centre for Food and Health (NICHE), University of Ulster, Coleraine, BT52 1SA*

Reactive oxygen species (ROS) are produced in the body in response to oxidative stress. These ROS can lead to oxidative damage to cells and tissues and may ultimately result in the development of chronic diseases such as atherosclerosis and cancer (Halliwell, 1996). Recent evidence suggests that non-nutrient antioxidants present in fruit, vegetables and whole-grain cereals may play an important role in decreasing oxidative stress in the body. Oat is a whole-grain cereal which contains non-nutrient phenolic antioxidants including ferulic acid, caffeic acid and avenanthramides, which are unique to oat, and which have been shown to have antioxidant activity *in vitro* (Peterson *et al*. 2002; Nardini *et al*. 1995). Phenolics have been found to be concentrated in the bran layer of the whole-grain and are therefore abundant in oat bran cereal products. The aim of the present investigation was to evaluate the postprandial effects of oat bran on antioxidant indices in the body in comparison with a rice control.

The study was a randomised within-subject cross-over design and eighteen healthy subjects (eight males, ten females) participated. The mean age was 23.6 + 4.9 (18–39) years and the mean BMI was 23.6 + 3.3 (19.7–30.4). Subjects arrived at the metabolic suite following an overnight fast on three occasions, at least 5 d apart. On arrival, subjects gave a baseline blood and urine sample and ingested one of two test products, including either oat bran (100 g, supplied by Cerealia, Sweden) or a ground rice control (100 g). Postprandial blood and urine samples were collected 30 min after ingestion and every following hour for 4 hours. Subjects consumed only water between sampling times. The ferric reducing ability (FRAP) (Benzie & Strain, 1996) and total phenolic content (TPC) (Singleton & Rossi, 1965) of postprandial blood and urine samples were measured. The intakes of total phenolics and antioxidant activity from the oat bran were 140 mg ferulic acid equivalents (FAE), and 1224 µmol FRAP, respectively. For the control the intakes of total phenolics and antioxidant activity were 29 mg FAE and 323 µmol FRAP, respectively.

Figures 1 and 2 show variations in the antioxidant indices following ingestion of the oat bran compared with the control. Results show that plasma total phenolics peaked 120 min after ingestion of the oat bran in comparison with the rice control. The FRAP activity of the plasma reflected changes in the total phenolics and also peaked at 120 min when compared to the control. Substantially more total phenolics were found in the urine of the subjects 180 min after ingestion of the oat bran compared with the control. The FRAP activity of the urine also peaked within 180 min of ingestion of the oat bran.

Figure 1: Changes in plasma total antioxidant status and total phenolics following consumption of 100g of oat bran compared to 100g of ground rice control product.

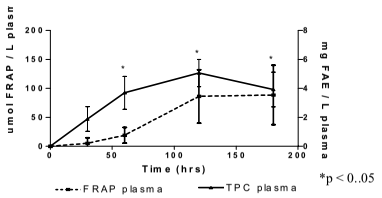
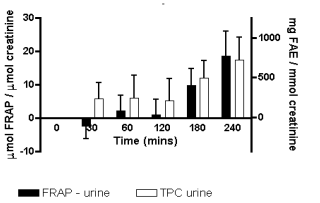


Figure 2: Changes in urine total antioxidant status and total phenolics following consumption of 100g of oat bran compared to 100g of ground rice control product.



These results suggest that increased plasma antioxidant potential after ingestion may be due to antioxidant activities of the absorbed phenolics. These phenolics, therefore, may enhance plasma antioxidant status of the body, and impact on disease processes. Furthermore, results show that the total amount of phenolics absorbed and hence excreted was small compared with the amount ingested. Thus, unabsorbed phenolics may exert antioxidant effects further down the gastrointestinal tract. This is a consideration for further study.

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Peterson DM, Hahn MJ & Emmons CL (2002) *Food Chemistry* **79**, 473–478.
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Halliwell B (1996) *Annual Review of Nutrition* **16**, 33–50.

Flavour release from gelatin, starch and pectin gels. By A.B. BOLAND and S.M. VAN RUTH, *Department of Food Science, Food Technology and Nutrition, University College Cork, Western Road, Cork, Ireland*

In the development of new food products, hydrocolloids have widely been extended into the area of reduced fat products. Therefore, there is an increased demand for knowledge of the effects of hydrocolloids on flavour release. The release of eleven flavour compounds from three gel systems, which varied in hydrocolloid (gelatin, starch and pectin), was investigated. The flavour compounds were diacetyl, 2-butanone, ethyl acetate, 1-butanol, 3-methyl-1-butanol, ethyl butyrate, hexanal, 2-heptanone, heptanal, 2-octanone, and 2-decanone. The texture of the gels was characterized as the Young's modulus of elasticity (E), which is a measure for the rigidity of the gel (Bourne, 1982). Flavour release profiles were obtained by model mouth analysis using proton transfer reaction–mass spectrometry (PTR-MS) over a 5-min period (Fig. 1). Static headspace analysis was conducted to determine the air/gel partition coefficients of compounds.

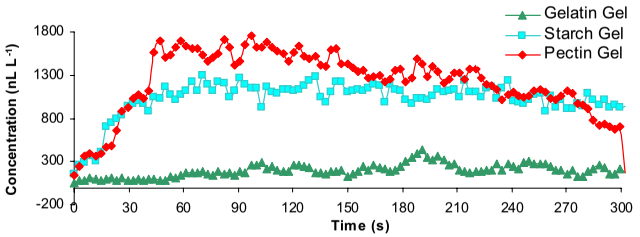


Fig. 1. The dynamic release of ethyl acetate as determined by model mouth/PTR-MS analysis

The gelatin gel was significantly more rigid than the starch and pectin gels, which were not significantly different from each other (P<0.05). For the gelatin gel, significantly lower maximum concentrations (I_{max}) were measured in model mouth analysis than for the starch or pectin gels for all flavour compounds (P<0.05). Six of the flavour compounds also had significantly lower air/gel partition coefficients in gelatin (P<0.05), indicating an effect of the gelatin on the thermodynamic component of flavour release. For the other compounds, the determining factor for the difference in aroma release between gelatin and the other two gels is the mass transfer coefficient of flavour compounds from the food to the air. Differences in I_{max} values and partition coefficients between starch and pectin gels could be attributed to matrix–volatile interactions involving gel components (e.g. starch, pectin and sucrose).

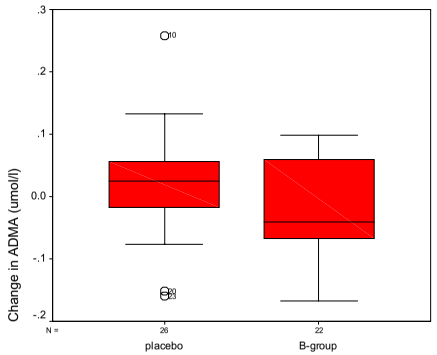
The most rigid gel, gelatin gel, showed significant increases in I_{max} values in the presence of saliva (P<0.05). Saliva enhanced the water content of the system, thereby increasing the surface area available for the diffusion of flavour compounds. The starch and pectin gels displayed significant decreases in I_{max} values (P<0.05). Dilution, salivary proteins and the increased hydrophilic nature of the system contributed to the reduced release of flavour compounds. For both the gelatin and pectin gels, the partition coefficients were significantly reduced for hydrophilic compounds and increased for hydrophobic compounds in the presence of saliva (P<0.05), which can be attributed to the increased hydrophilic nature of the system. Therefore, flavour release is significantly affected by the texture of the gels.

Bourne M (1982) *Food Texture and Viscosity: Concept and Measurement,* pp. 44–117. London: Academic Press.

B-vitamins and asymmetric dimethylarginine: a placebo-controlled study. By S.E.C.M. GILCHRIST[1], J.V. WOODSIDE[1], I.S. YOUNG[1], J.W.G. YARNELL[1], K.F. GEY[2] and A. EVANS[1], [1]*School of Clinical Medicine, Queen's University Belfast, Belfast BT12 6BJ, and* [2]*Department of Biochemistry, University of Berne, Switzerland.*

Mild hyperhomocysteinemia is accepted as a risk factor for premature cardiovascular disease. One mechanism which may contribute to homocysteine-induced endothelial dysfunction is increased synthesis of asymmetric dimethylarginine (ADMA), an endogenous nitric oxide synthase inhibitor (Boger *et al.* 2000). B-group vitamins can lower homocysteine (Brattstrom *et al.* 1998), but it is not known what effect they have on ADMA concentrations in humans.

In a population with a high prevalence of cardiovascular disease, we screened a group of clinically healthy male factory employees aged 30-49 (n=509) for plasma homocysteine. Those with mildly elevated homocysteine concentrations (≥8.34 μmol/L) were selected for intervention. In a randomised, placebo-controlled trial, the effect of B-group vitamin supplementation on ADMA concentrations were assessed. Subjects were randomly assigned to one of two groups: supplementation with B-group vitamins (1 mg folic acid, 7.2 mg pyridoxine, 0.02 mg cyanocobalamin daily) or placebo. A total of 48 men completed the 8-week intervention. ADMA was assessed by HPLC with fluorescence detection according to Teerlink *et al*. (2002). When the change in ADMA levels over the supplementation period was analysed by group, there was no significant difference between placebo and intervention group, as shown in the Figure below (baseline – week 8 level; placebo group 0.020 (0.086); B-vitamin group –0.021 (0.075) μmol/L; Mean (SD); P=0.08).



This study shows that supplementation with the homocysteine-modulating B-group vitamins has no significant effect on ADMA, although there is a tendency for ADMA levels to decrease.

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Teerlink T, Nijveldt RJ, de Jong S, & van Leeuwen PAM (2002) *Analytical Biochemistry* **303**, 131–137.

Human milk oligosaccharides and intestinal permeability in Egyptian infants. By S.K. STRANDERS[1], C.A. NORTHROP-CLEWES[1], G.A. YAMAMAH[2], M.E. ABOU-ZEKRI[3] and D.I. THURNHAM[1], [1]*Northern Ireland Centre for Food and Health, University of Ulster, Coleraine, BT52 1SA,* [2]*National Research Center, Clinical Medical Sciences Department, El-Tahrir Street, Giza, Egypt and* [3]*Cairo University Children's Hospital, Social and Preventive Pediatric Center, Tropical Pediatric Clinic, El-Kasr El-Eini Street, Cairo, Egypt*

Human milk oligosaccharides (HMO) may exert anti-adhesive properties by acting as soluble receptor ligands for bacteria invading the intestinal mucosa (Newburg, 1999). Bacterial adherence to the enterocyte has been shown to precede deterioration in permeability of cultured cells. Abnormally increased intestinal permeability in infants of developing countries has been shown to begin during weaning when food is introduced that is likely to be contaminated (Lunn *et al.* 1991). To test the hypothesis that HMO may protect the gut mucosa, the intestinal permeability (i.e. gut integrity) of 2–12-month-old Egyptian infants was measured from the urinary recovery of the orally administered markers lactulose and mannitol. Both leakage in the paracellular pathway (increased lactulose passage) and villous atrophy (decreased mannitol recovery) will increase the lactulose:mannitol (L:M) ratio. L:M ratios of exclusively breast-fed (group B) and mixed-fed, i.e. partially breast-fed infants (group M) were compared and related to the concentration of HMO and HMO fractions of their mothers. Infants who received only formula milk instead of breast milk served as the control group. HMO were separated into acidic and seven neutral fractions by a modified gel permeation chromatographic method and monitored by refractive index detection (Thurl *et al.* 1991). The presence of infections increases gut permeability. Urinary neopterin was used as a sensitive marker for subclinical infections. Most infants were from poor socio-economic backgrounds.

Gut integrity was worse in older infants , i.e. urinary L:M ratios in groups B and M combinded (no differences between B and M for different age groups) increased with age to abnormal values (ratios >0.12) mainly due to decreased mannitol absorption. The highest L:M ratios were found in infants of the control group, which differed significantly from those in group B and M combined (P<0.01) at <4 months. When all age groups were combined the control group had significantly higher L:M ratios (P<0.05) and significantly lower mannitol:creatinine (M:C) ratios (P<0.05) than infants of group B and M combined. The neopterin values were used to remove the age associated influence of infection on the gut integrity measurements to generate residuals from the L:M and M:C ratios.

The concentrations of total HMO and most of the neutral fractions declined over the course of lactation. Regression analysis revealed significant correlations between certain HMO fractions (logged) and the permeability markers (logged). About 14% (negative, P=0.001) of the variance in the residuals from the L:M ratios was explained by the influence of a neutral fraction containing HMO of high molecular weight (>1500 Da). This result was consistent even when the variable "age" was included into the analysis. This particular fraction also predicted ~15% (positive, P=0.006) of the observed variance in the residual M:C ratio. However, when age was included, only age significantly explained the variance of residual M:C ratio (negative, P=0.000).

These findings support the hypothesis that HMO, particularly the high-molecular-weight fraction, help to prevent a deterioration of intestinal integrity. However, correcting the gut integrity data using neopterin has not fully removed the age associated deterioration. The high-molecular-weigth HMO fraction also correlates with age but this represents a physiological change in milk composition and is not infection-related. Whether there is a direct causal relationship between certain HMO fractions and gut integrity markers can only be shown on the basis of intervention studies.

Ethical consideration: The study protocol was approved by the Head of the Social and Preventive Pediatric Center, Cairo University Children's Hospital. Informed consent was obtained from the parent(s).

Acknowledgements: The authors acknowledge the assistance from an International Training Fellowship of the Nutricia Research Foundation to carry out this work.

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Thurl S, Offermanns J, Müller-Werner B & Sawatzki G (1991) *Journal of Chromatography* **568**, 291–300.

The effect of conjugated linoleic acid on viability and metabolic activity of human SaOS-2 osteoblastic cells. By S. CUSACK, C. JEWELL and K.D. CASHMAN, *Department of Food and Nutritional Sciences, University College Cork, Ireland*

Conjugated linoleic acid (CLA) has been shown to reduce both the biosynthesis of prostaglandin E_2 (PGE_2), a potent modulator of bone metabolism, and the rate of bone formation in experimental rats (Li *et al*. 1999), but the mechanism of action is unclear. The influence of CLA on human bone metabolism has not been investigated. Consequently, the objectives of this study were first to investigate the influence of CLA, at physiological and super-physiological levels, on the viability of human osteoblastic cells (which are responsible for bone formation) and, secondly, to determine the effect of CLA on the biosynthesis of PGE_2 and alkaline phosphatase (important mediators of bone formation) by these cells. A human osteoblast-like cell line, SaOS2, was grown to confluency and exposed to increasing concentrations (0–50 μM) of linoleic acid, or the pure *cis* 9:*trans* 11 and *trans* 10:*cis* 12 isomers of CLA, for 24 h. Cell viability was assessed using the MTT, Neutral Red uptake and Resazurin colorimetric assays. PGE_2 levels were assessed using an enzyme-linked immunosorbent assay, while alkaline phosphatase activity was measured using a colorimetric assay. In all studies, at least three wells were examined per treatment. Experiments were repeated three times.

Treatment	PGE_2 (pg/mg protein)		Alkaline phosphatase (U/l)	
	Mean	SEM	Mean	SEM
Control	1.641	0.342	0.034	0.004
6.25 μM Linoleic acid	0.673	0.145	0.032	0.003
12.5 μM Linoleic acid	0.610	0.350	0.691	0.026
25 μM Linoleic acid	0.580*	0.100	0.094	0.024
50 μM Linoleic acid	0.556*	0.230	0.134	0.035
6.25 μM *cis* 9: *trans* 11 CLA	1.093	0.370	0.099**	0.001
12.5 μM *cis* 9: *trans* 11 CLA	0.836	0.083	0.246**	0.039
25 μM *cis* 9: *trans* 11 CLA	0.770	0.020	0.352**	0.011
50 μM *cis* 9: *trans* 11 CLA	0.776	0.146	0.438**	0.037
6.25 μM *trans* 10: *cis* 12 CLA	0.716*	0.092	0.030	0.007
12.5 μM *trans* 10: *cis* 12 CLA	0.636*	0.073	0.070	0.004
25 μM *trans* 10: *cis* 12 CLA	0.593**	0.036	0.177*	0.001
50 μM *trans* 10: *cis* 12 CLA	0.613*	0.042	0.419**	0.013

Significantly different from control: *P<0.05; **P<0.01.

Human osteoblastic cell viability, as measured by all three assays, was unaffected by any fatty acid treatment. While PGE_2 biosynthesis by SaOS-2 cells was significantly reduced by (25–50 μM) linoleic acid and (6.25–50 μM) *trans* 10:*cis* 12 isomer of CLA, it was unaffected by *cis* 9:*trans* 11 isomer of CLA. On the other hand, alkaline phosphatase activity, a marker of osteoblast activity, was unaffected by linoleic acid, but significantly increased by both isomers of CLA. An increased activity of alkaline phosphatase was achieved at physiological concentrations of the *cis* 9:*trans* 11 CLA isomer, whereas *trans* 10:*cis* 12 CLA only increased alkaline phosphatase activity at pharmacological concentrations. In conclusion, these results suggest that CLA may stimulate the rate of bone formation in human osteoblast cells. The mechanism of action of this effect requires further investigation.

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Watkins B, Lippman HE, Le Bouteiller L, Li Y & Seifert MF (2001) *Progress in Lipid Research* **40**, 125–148.

Effect of medium-chain fatty acids on Caco-2 cell transepithelial calcium transport. By C. JEWELL and K. CASHMAN, *Nutritional Sciences, Department of Food Science, Food Technology and Nutrition, University College Cork, Ireland*

Capric, lauric, linoleic and conjugated linoleic acid isomers are widely found in dairy produce. An increase in bone ash and rate of bone formation has been found in experimental animals given conjugated linoleic acid (CLA) (Cook *et al*. 1997). CLA may achieve this by an indirect action of increasing intestinal permeability to calcium. The aim of this study was to measure the effect of these fatty acids on transepithelial calcium transport, using the human Caco-2 intestinal cell model. Caco-2 cells were grown on permeable transport membranes for 14 d and allowed to differentiate, resulting in a structure similar to that of the intestinal wall. During this period the Caco-2 cells (n=9 per treatment) were grown in media containing 0 or 80 μM fatty acid. 1,25 dihydroxycholeciferol ($1,25(OH)_2D_3$) (10 nM) was used as a positive control. After exposure, calcium transport across the membrane was measured using the isotope ^{45}Ca. Paracellular transport was determined simultaneously using sodium fluorecein as a marker (Lindmark *et al.* 1998). No effect on calcium transport was seen with cells treated with capric, lauric or linoleic acid. However, cells treated with *cis*9:*trans*11 CLA and *trans*10:*cis*12 CLA isomers led to an increase in total calcium transport. Interestingly, *cis*9:*trans*11 CLA and *trans*10:*cis*12 CLA isomers dramatically increased calcium transport across the epithelium by both paracellular (146 and 166%) and transcellular (2148 and 2938%) transport routes (P<0.001). However, only treatment with the *trans*10:*cis*12 CLA isomer resulted in a significant decrease in transepithelial electrical resistance (TEER), a marker of monolayer permeability. The mechanism(s) behind these effects are still not known and further investigations are needed to determine how CLA isomers modulate calcium transport.

Treatment	Calcium transport									
	Total transepithelial				Transcellular*		Paracellular		TEER	
	(nmol/well/min)		(%/h)		(nmol/well/min)		(%/h)		(Ω.cm^2)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Control	0.275^a	0.021	0.916^a	0.068	0.008^a	0.011	0.892^a	0.032	1217^a	32
$1,25(OH)_2D_3$	0.508^c	0.060	1.693^c	0.200	0.187^c	0.052	1.070^b	0.027	818^d	14
Capric acid	0.336	0.053	1.121	0.177	0.052	0.037	0.948	0.053	1189	44
Lauric acid	0.351	0.014	1.171	0.047	0.053	0.008	0.994	0.020	1119	49
Linoleic acid	0.333	0.009	1.107	0.033	0.009	0.002	1.077^b	0.027	1208	13
*c*9,*t*11 CLA	0.518^d	0.057	1.839^d	0.191	0.163^b	0.039	1.300^d	0.062	1222	41
*t*10,*c*12 CLA	0.666^d	0.058	2.222^d	0.193	0.223^b	0.044	1.480^d	0.046	823^d	90
P value	<0.0001		<0.0001		<0.0001		<0.0001		<0.0001	

TEER = Transepithelial electrical resistance (at day 14).
Mean values within a column with different superscript letters were significantly different (ANOVA followed by least significant difference test; $^{a,b}P$<0.05, $^{a,c}P$<0.01, $^{a,d}P$<0.001).
*Transcellular transport is total calcium transport corrected for paracellular (fluorescein) transport.

Cook ME, Jerome DL & Pariza MW (1997) *Poultry Science* **76**, 41.
Lindmark T, Kimura Y & Artursson P (1998) *Journal of Pharmacology and Experimental Therapeutics* **284**, 362–369.

The effect of 1,25 dihydroxycholeciferol on calcium transport in intestinal-like Caco-2 cells. By M. BUTLER[1], C. JEWELL[1] and K.D. CASHMAN[1,2], [1]*Department of Food and Nutritional Sciences and* [2]*Department of Medicine, University College Cork, Ireland*

Ca is absorbed by two routes, namely transcellular and paracellular transport. While 1,25 dihydroxycholeciferol (1,25 $(OH)_2$ D_3) is known to enhance transcellular calcium transport, its effect on paracellular transport is unclear. While some researchers have shown that 1,25 $(OH)_2$ D_3 stimulates paracellular Ca transport in Caco-2 cells (Chirayath *et al.* 1998), others have reported a lack of effect (Fleet *et al.* 1996, 2002). Caco-2 cells are regarded as a good model of intestinal Ca absorption in humans (Fleet *et al.* 1996). Therefore, the aim of this study was to examine the effect of short-term exposure of intestinal-like human Caco-2 cells to 1,25 $(OH)_2$ D_3 on the two routes of calcium transport. Caco-2 cells were seeded onto permeable filter supports and allowed to differentiate into a highly organized structure similar to that of the intestinal wall. On day 15, the Caco-2 monolayers (*n* 10–15 per treatment) were treated with either 0, 10 or 100 nM 1,25 $(OH)_2$ D_3 for 24 h (Experiment 1), or with 0 or 100 nM 1,25 $(OH)_2$ D_3 for 48 h (Experiment 2), before measuring ^{45}Ca transport over 60 min. After exposure, transepithelial transport of ^{45}Ca, and fluorescein and ^{14}C-mannitol transport (markers of paracellular diffusion), transcellular Ca transport and transepithelial electrical resistance (TEER; an index of monolayer permeability) were measured.

	Ca transport							
	Total transepithelial (%/h)		Transcellular (nmol/well/min)		Paracellular (%/h)		TEER ($\Omega.cm^2$)	
Treatment	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Expt. 1								
Control	0.337[a]	0.020	0.034[a]	0.007	0.210[a]	0.013	2030[a]	31
1 nM 1,25 $(OH)_2$ D_3	0.383[a]	0.030	0.055[a]	0.010	0.213[a]	0.010	1983[a]	23
10 nM 1,25 $(OH)_2$ D_3	0.550[b]	0.060	0.084[b]	0.015	0.230[a]	0.013	1798[b]	15
100 nM 1,25 $(OH)_2$ D_3	0.687[c]	0.047	0.136[c]	0.016	0.227[a]	0.010	1720[c]	16
P value	<0.0001		<0.0001		0.498		<0.0001	
Expt. 2								
Control	0.573	0.047	0.059	0.004	0.370	0.050	2063	25
100 nM 1,25 $(OH)_2$ D_3	0.960*	0.033	0.180*	0.004	0.363	0.033	1352*	16

[a,b,c] Mean values within a column (in Expt. 1) with different superscript letters were significantly different ($P<0.05$).
Significantly different from control (within Expt..2): *$P<0.0001$.

Treatment of Caco-2 cells with 10 and/or 100 nM 1,25 $(OH)_2$ D_3 for either 24 or 48 h significantly increased total transepithelial Ca transport and transcellular Ca transport relative to control, untreated cells. Paracellular calcium transport (as measured by two different indices of paracellular permeability, i.e. fluorescein and ^{14}C-mannitol transport across the Caco-2 cell monolayers as well as TEER, a marker of monolayer integrity) was unaffected by 1,25 $(OH)_2$ D_3. These findings suggest that short-term exposure of Caco-2 cells to 1,25 $(OH)_2$ D_3 increases total transepithelial transport of calcium via the transcellular and not the paracellular route.

Chirayath MV, Gajdzik L, Wolfgang H, Graf J, Cross HS & Peterlik M (1998) *American Journal of Physiology* **274**, G389–G396.
Fleet JC, Bradley J, Satyanarayana R, Rahul R & Wood RJ (1996) *Archives of Biochemistry and Biophysics* **329**, 228–234.
Fleet JC, Eksir F, Hance KW & Wood RJ (2002) *American Journal of Physiology* **283**, G618–G625.

Relationship between sources of vitamin D, sunlight exposure and diet, and bone mineral density in a group of Spanish elderly women. By L. QUINTANILLA[1], C. CUADRADO[2], N. LILLO[2], O. MOREIRAS[2], S. POZO[2], M. RODRÍGUEZ[2], V. RODRÍGUEZ[2], B. RUÍZ-ROSO[2] and G. VARELA-MOREIRAS[2] [1]*Sección de Nutrición, Bromatología y Dietética, Facultad de Ciencias Experimentales y de la Salud, Universidad San Pablo-CEU, 28668, Boadilla, Spain* [2]*Departamento de Nutrición, Facultad de Farmacia, Universidad Complutense de Madrid, Ciudad Universitaria, 28040, Madrid, Spain*

The aim of this study was to evaluate the sources of vitamin D (sunlight exposure and diet) in a group of Spanish elderly women, as part of Optimal Strategy for Vitamin D Fortification (OPTIFORD), an EU project involving five countries (Finland, Denmark, Poland, Ireland and Spain) which has as a final objective to determine whether the fortification of food with vitamin D is a feasible strategy in the prevention of osteoporosis.

Forty-nine volunteer elderly women aged 70–75 years were recruited and examined during June and July 2002, when sunshine (u.v. radiation) is at its maximum. Sunshine exposure was measured by an individual dosimeter (VioSpor), which subjects wore on their shoulder for 1 week. The values of sunshine exposure were obtained from BioSense (Germany). The participants filled out a diary questionnaire for the u.v.-Dosimeter with information about the number of hours spent outside, weather conditions and the clothes they were wearing during that time.

A 3-d food record and a food-frequency questionnaire were used to assess the intake of vitamin D from the diet. Bone mineral density (BMD) was determined using a Hologic Sahara Clinical Bone Sonometer based on an ultrasound measurement of the calcaneous (heel bone). A general questionnaire regarding the history of bone fractures and lifestyle (conducted in relation to sunshine during the sunny months) was filled out.

In terms of sunlight exposure the results (in J/m^2) were: mean (SD) = 776.4, (635.2), 25th percentile = 388.0, median = 582.5, 75th percentile = 933.5, range: 42–3008. From this study 57 % of the participants avoid staying in direct sunshine, these results are similar to those obtained in SENECA study (Moreiras *et al.* 1992), where 56 % of Spanish participants avoid being in the sun. Only 12.2 % the participants enjoy staying in the sunshine regularly.

The mean daily vitamin D intake from the 3-d food record was 3.9 ± 5.1 µg/d, which represents only 26 % of the Recommended Dietary Intake for Spain, 15 µg/day. The fish food group was the main dietary source of vitamin D.

Table. Values obtained based on an ultrasound measurement of the calcaneous (T-Score)

	Mean (SD)	Minimun	Maximun	T-Score >–1 (Normal)	T-Score –1 - –2.5 (Osteopenia)	T-Score <–2.5 (Osteoporosis)
Women (n=44)	–1.27 (0.87)	–2.70	1.20	25 %	72.7%	2.5%

With a linear regression analysis, a positive significant correlation (r=0.35) ($P<0.05$) was observed between dietary intake of vitamin D (3-d food record) and BMD (T-Score). No correlation was found between sunlight exposure and BMD.

In relation to sources of vitamin D in this population group: a majority of the participants avoided staying in direct sunshine and the mean daily intake of vitamin D represents only 26 % the Recommended Dietary Intake. The main dietary source of vitamin D was fish.

Moreiras O, Carbajal A, Perea I & Varela-Moreiras G (1992) *International Journal of Vitamin Nutritional Research* **62**, 303–307.

Vitamin D status in two risk groups from four European countries. By R. ANDERSEN[1], C. BROT[1], K.D. CASHMAN[4], J. CHARZEWSKA[6], A. FLYNN[4], J. JAKOBSEN[1], M. KÄRKKÄINEN[2], M. KEILY[4], C. LAMBERG-ALLARDT[2], O. MOREIRAS[5], C. MØLGAARD[3], A.M. NATRI[2], M. O'BRIEN[4] and L. OVESEN[1], [1]*Danish Veterinary and Food Administration, Denmark,* [2]*University of Helsinki, Finland,* [3]*The Royal Veterinary and Agricultural University, Denmark,* [4]*University College Cork, Ireland,* [5]*Universidad Complutense de Madrid, Spain and* [6]*National Food and Nutrition Institute, Poland*

Severe vitamin D deficiency leads to rickets in children and to osteomalacia in adults. Optimal vitamin D status is essential for proper bone mineralization, and vitamin D deficiency may be an important risk factor for hip fractures (Trivedi *et al*. 2003). The best measurement of vitamin D status is serum 25-hydroxy vitamin D (S-25OHD). There is no international consensus on cut-off levels for vitamin D deficiency and vitamin D insufficiency (McKenna & Freaney, 1998). In this study, deficiency has been defined as S-25OHD <25 nmol/l and insufficiency as S-25OHD <50 nmol/l.

The aim of this observational study was to determine the vitamin D status in adolescent girls (11.6–13.5 years) and elderly women (70–75 years) from different parts of Europe. Four countries (Denmark, Finland, Ireland and Poland) participated in the first cross-sectional part of the study, which was simultaneously conducted in all four countries during February and March 2002. All S-25OHD analyses were performed by HPLC in the same laboratory. Results are given in the table below.

	Denmark	Finland	Ireland	Poland
Girls				
n	59	57	17	61
Age[a]	12.5 (0.5)	12.8 (0.4)	12.1 (0.8)	12.6 (0.6)
Weight (kg)[a]	47.7 (12.8)	48.4 (7.9)	49.7 (12.8)	47.9 (11.5)
Height (cm)[a]	157.2 (7.9)	158.7 (6.2)	152.4 (10.3)	157.7 (6.7)
BMI (kg/m^2)[a]	19.1 (3.8)	19.1 (2.4)	21.2 (4.3)	19.2 (4.4)
S-25OHD (nmol/l)[a]	27.9 (14.3)*#	29.4 (9.9)*#	38.5 (11.4)*	32.3 (14.3)*
Women				
n	54	60	43	68
Age[a]	71.7 (1.4)	71.9 (1.4)	72.3 (1.5)	71.7 (1.4)
Weight (kg)[a]	69.8 (13.5)	71.2 (11.8)	65.1 (11.7)	71.1 (12.2)
Height (cm)[a]	162.1 (6.5)	158.4 (6.1)	158.2 (6.1)	156.7 (6.4)
BMI (kg/m^2)[a]	26.5 (4.3)	28.3 (4.1)	26.1 (4.4)	28.9 (4.2)
S-25OHD (nmol/l)[a]	47.3 (22.6)*#	46.7 (18.7)*#	46.9 (20.6)*	32.5 (12.9)*

[a] Mean (SD). *Significant difference between countries within same age group (P<0.01).
Significant difference between women and girls within same country (P<0.0001).

If the four countries were combined, 37 and 92% of the girls had S-25OHD <25 nmol/l and 50 nmol/l, respectively, and 17 and 67% of the women had S-25OHD <25 nmol/l and 50 nmol/l, respectively. The table below shows the percentile values for the combined results.

Percentile – Girls	5	10	25	50	75	90	95
nmol/l	13.8	16.1	20.3	29.4	38.3	47.4	55.0
Percentile – Women	5	10	25	50	75	90	95
nmol/l	14.9	18.7	28.0	40.7	54.2	69.4	76.7

In conclusion, a significant number of adolescent girls and elderly women in Europe may be at risk of hypovitaminosis D during winter and early spring, with possible consequences for their bone health.

Blood samples were taken again in August–September 2002 and February–March 2003. However, results for S-25OHD are not yet available (March 2003). We will evaluate the relative impact of sun exposure and dietary intake (including food fortification and supplementation), on seasonal variation of vitamin D status across Europe. The study is part of the EU project OPTIFORD 'Towards a strategy for optimal vitamin D fortification' (Andersen *et al*. 2001).

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The impact of using revised food composition data for vitamin D on estimates of vitamin D intakes in 18-64 year-old Irish adults. By T.R. HILL[1], M.M. O'BRIEN[1], K.D. CASHMAN[1,2], A. FLYNN[1] and M. KIELY[1], [1]*Department of Food and Nutritional Sciences and* [2]*Department of Medicine, University College Cork, Ireland*

Vitamin D is the major regulator of calcium homeostasis and is thus an important determinant of bone health (Van Leeuwen *et al*. 2001). Exposure of the skin to sunlight is regarded as the most important source of vitamin D, but dietary sources become increasingly important in winter when sunlight is limited. Earlier analysis of the North/South Ireland Food Consumption Survey (NSIFCS) showed that the mean vitamin D intake in adults aged 18–64 years was low, at 3.7 µg/d (O'Brien *et al*. 2001). These low values may be partly attributed to the relatively dated and incomplete vitamin D data in McCance and Widdowson's *Composition of Foods* (5th edn.). The aim of the current study was to re-assess vitamin D intakes in the NSIFCS sample using a revised vitamin D compositional database.

The NSIFCS established a database of habitual food and drink consumption using a 7 d food diary in 1379 randomly selected adults, aged 18–64 years. Of the 3060 food codes in the survey database, the vitamin D content of sixty-one were changed. Of these 61 foods, fresh samples of 13 were sent to Eclipse Laboratories (Cambridge, UK) for analysis of their vitamin D_2 and D_3 content by a reverse phase High Performance Liquid Chromatography method. These foods included cod, ham, chicken in breadcrumbs, mushrooms and six different type of vegetable fat spread. Of the 13 foods tested, a vegetable fat spread had the highest vitamin D_3 content (3.49µg/100g). Mushrooms were the only food to contain vitamin D_2 (0.7µg/100g). These analytical values were used to update the 13 food codes and the recipes (n=48) in which they were included.

Mean daily vitamin D intakes calculated from the revised vitamin D composition data in men and women (including the contribution of nutritional supplements) are shown below:

	Men				Women			
	18–35 y	36–50 y	51–64 y	All ages	18–35 y	36–50 y	51–64 y	All ages
	n 253	*n* 236	*n* 173	*n* 662	*n* 269	*n* 286	*n* 162	*n* 717
Mean	3.7*	4.7*	5.1	4.4*	3.3	4.0	5.1	4.0
Median	3.0	3.7	3.6	3.3	2.2	2.6	3.3	2.6
Standard deviation	2.5	3.3	4.5	3.5	3.1	3.4	4.9	3.8

Significantly different from women, *P <0.001.
Differences between age groups were significant for men and women, *P <0.001.

The total mean daily intake of vitamin D from this study was 4.2 µg, which was significantly (P<0.001) higher than previously reported values by O'Brien *et al*. (2001) of 3.7 µg/d. The RDA for vitamin D is 0–10 µg/d for adults, depending on sunlight exposure (FSAI, 1999). Out of a total of 1379 respondents, 1026 (74%) had a vitamin D intake less than the midpoint of the RDA (5µg/d). Meat and meat products were the main food group contributing to vitamin D intake in men (34%) and women (26%). The percentage contribution of nutritional supplements and breakfast cereals to vitamin D intake was higher in women (12 and 7%, respectively) than in men (7 and 3%, respectively).

In conclusion, higher vitamin D intakes than were previously reported were obtained from the same food consumption database, showing that a substantial bias had been introduced to the estimates by outdated food compositional data. However, it remains clear that a large number of Irish adults have low vitamin D intakes and this, along with emerging evidence which show a low vitamin D status in at least some population subgroups (Hill *et al*. 2002), suggests that strategies to increase vitamin D intakes should be investigated.

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Further details on the North/South Ireland Food Consumption Survey see: www.iuna.net/

Sources of Vitamin D -sunlight exposure and diet- in two Spanish age groups: elderly women and adolescent girls. By N. LILLO[1], C. CUADRADO[1], L. PÉREZ-OLLEROS[1], S. POZO[1], L. QUINTANILLA[2], M. RODRÍGUEZ[1], V. RODRÍGUEZ[1], G. VARELA-MOREIRAS[2], O. MOREIRAS[1] [1]*Departamento de Nutrición, Facultad de Farmacia, Universidad Complutense de Madrid, Ciudad Universitaria, 28040, Madrid, Spain,* [2]*Sección de Nutrición, Bromatología y Dietética, Facultad de Ciencias Experimentales y de la Salud, Universidad San Pablo-CEU, 28668, Boadilla, Spain,*

The aim of this study was to evaluate the sources of vitamin D in two age groups (elderly and adolescence), as part of OPTIFORD (Optimal Strategy for Vitamin D Fortification), an EU project involving five countries (Finland, Denmark, Poland, Ireland and Spain) which has as final objective to study if the fortification of food with vitamin D is a feasible strategy to remedy the insufficient vitamin D status of large population groups in Europe to ensure the bone integrity.

Ninety-six volunteer subjects, forty-nine elderly women aged (70–75 years) and forty-seven girls aged (11.6–13.5 years) were recruited and studied during June and July 2002, when the sunshine (u.v. radiation) is the highest. In relation to sunlight exposure, all participants wore an individual dosimeter (VioSpor) for a week, the measurement of the UV radiation was carried out by BioSense (Germany), also a diary questionnaire for the UV-Dosimeter was filled out by the subjects, with information about: hours spent outside, weather conditions and their clothing worn during that time.

To assess the intake of vitamin D from the diet, a 3-d food record and a food frequency questionnaire were used.

		Mean (SD)	25th percentile	Median	75th percentile	Range
Sunlight exposure	Women	776.4 (635.2)***	388	582.5	933.5	42–3008
(J/m^2)	Girls	1519.1 (832.2)	886.5	1446	1796.8	206–3248
Total outdoors hours/d	Women	3.4 (2.0)	2.0	3.1	4.4	0.1–9.6
(for a week)	Girls	4.7 (1.6)	3.5	4.5	5.8	1.7–8.0
Vitamin D intake (μg/d)	Women	3.9 (5.1)	0.7	1.5	4.7	0.03–23.2
from the 3-d food record	Girls	2.9 (2.6)	1.0	1.7	3.7	0.2–10.7

*** P<0.001.

Sunlight exposure of adolescent girls was significant higher than elderly women (P<0.001), however not found significant differences in dietary intake of vitamin D.

In relation to the results obtained from the food frequency questionnaire, the types of fish (which a significant content of vitamin D) most consumed were sardines (60.9%) and canned tuna/bonito (37%) for elderly women and canned tuna/bonito (56.5%) and anchovies (43.5%) for adolescent girls. This food group was the main source of vitamin D in both group of participants.

Skimmed cow milk was the most consumed (39.1%) for elderly women, however 53.1% of adolescent girls consumed whole fat cow milk.

The most consumed cheeses for the adolescent girls were: semi-ripe Manchego (39.1%), Dutch (56.5%), curd (36.9%) and Petit Suisse (43.5%) and for the elderly women were: semi-ripe Manchego (52.2%), Galician (50%) and cottage (47.8%).

In relation to sources of vitamin D in these groups of population: sunlight exposure of adolescent girls was significant higher than elderly women and in terms of vitamin D from the diet, although significant differences were not observed between the groups, the mean daily intake of vitamin D for elderly represents only the 26% of the recommended dietary intake (15 μg/d) whereas the mean daily intake of vitamin D for adolescent girls represents the 58% of the recommended dietary intake (5 μg/d) (Moreiras *et al*. 2003). The main dietary source of vitamin D was fish in both groups of population.

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