

Proceedings of the Nutrition Society

Abstracts of Original Communications

A Scientific Meeting was held at the Institute of Electrical Engineers, Glasgow, UK on 27–28 March 2003, when the following papers were presented.

All abstracts are prepared as camera-ready material.

The Editors of the Proceedings of the Nutrition Society accept no responsibility for the abstracts of papers read at the Society's meetings for original communications.

Differences in habitual physical activity and inactivity in young children from deprived versus wealthy families. By L.A. KELLY¹, J.J. REILLY¹, S. GRANT², and J.Y. PATON¹, ¹Division of Developmental Medicine, Yorkhill NHS Trust, Glasgow, G3 8SJ and ²IBLS, The University of Glasgow, Glasgow, G12 8QQ

In the UK an epidemic of childhood obesity occurred during the 1990s (Reilly & Dorosty, 1999). The epidemic had a disproportionately large impact on children from families of lower socio-economic status (SES), but the cause of this SES difference is unclear. The aim of the present study was to test the hypothesis that habitual physical activity and inactivity differ between young children in association with SES. We studied thirty-nine pairs ($n = 78$) of children aged 4–5 years old from upper and lower SES categories in Glasgow. SES was characterised for each family using the Carstairs Score, a geographically defined index based on postal sector of the family residence (Carstairs & Morris, 1991). The Carstairs Score is widely used in Scotland, and has been shown to denote marked and real differences in socio-economic circumstances and health outcomes (Carstairs and Morris, 1991). The Carstairs Score ranges from 1 (most affluent) – 7 (most deprived). The children were pair-matched for age, gender and season. Total physical activity and inactivity were measured for a minimum of 4 d and maximum of 7 d (mean 73.9 h, SD 12.3) using the Computer Science and Applications CSA WAM-7164 (MTI, Shalimar, Florida) accelerometer (Nilsson *et al.* 2002). This accelerometer is practical for use with young children (Jackson *et al.* 2003), and has high validity relative to the criterion method of direct observation of activity (LA Kelly, JJ Reilly, SJ Barrie, G Milroy, S Grant and JY Paton, unpublished results). Time spent in different intensities of activity was estimated using published cut-off points which allow accelerometry output to be defined as behaviour (sedentary) behaviour, defined as <1100cpm (Reilly *et al.* 2003), light intensity activity defined as ≥ 1101 cpm to 3200cpm, moderate–vigorous physical activity ≥ 3201 cpm, MVPA (Puyau *et al.* 2002)). Paired *t* tests were used to assess the significance of differences in physical activity and inactivity between the two SES categories. Differences in total activity between the two groups ($P < 0.05$) were significant in boys but not in girls. However, these differences were small (affluent group mean 733cpm, SD 150.9; deprived group mean 763cpm, SD 176.4) and suggested slightly lower levels of total physical activity in the upper SES category relative to the deprived group. The more affluent boys were also slightly but significantly more inactive than the deprived group (percentage of time spent inactive by affluent group, mean 77.9cpm, SD 5.4; percentage of time spent inactive by deprived group, mean 73.3cpm, SD 6.2). For the girls we found no significant differences between the two groups for any aspect of physical activity or inactivity. In both sexes and in both socio-economic groups engagement in MVPA was low (percentage of time spent in MVPA by affluent group, mean 3.4cpm, SD 1.6; percentage of time spent in MVPA by deprived group, mean 3.7cpm, SD 1.9). In conclusion, the present study does not support the hypothesis that young children from more deprived families are less active or more inactive than children from wealthier families. The low levels of engagement in moderate–vigorous physical activity in these young children should give serious cause for public health concern.

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Cairstairs V & Morris R (1991) *Aberdeen University Press*.
 Jackson DM, Reilly JJ, Kelly LA, Montgomery C, Grant S, Paton JY (2003) *Obesity Research* **11**, 420–425.
 Nilsson A, Eklund U, Yngave A & Sjöström M (2002) *Pediatric Exercise Science* **14**, 87–96.
 Puyau MR, Adolph AL, Finos AV & Butts N (2002) *Obesity Research* **10**, 150–157.
 Reilly JJ & Dorosty, AR. (1999) *Lancet* **99**, 1874–1875.
 Reilly JJ, Coyle J, Kelly LA, Burke G, Grant S & Paton JY (2003) *Obesity Research* (In the press).

Early life risk factors for later obesity in contemporary children. By J. ARMSTRONG¹, J.J. REILLY¹, A. SHERRIFF², A.R. DOROSTY¹, P.M. EMMETT², A. NESS², I. RODGERS², C. STEER² and ALSPAC STUDY TEAM², ¹University of Glasgow, Division of Developmental Medicine, Glasgow G3 8SJ and ²ALSPAC Study Team, Division of Child Health, University of Bristol, Bristol BS8 1TQ

Childhood obesity is associated with adverse physiological and psycho-social effects for the child and an increased risk of adult morbidity and mortality. The identification of (modifiable) risk factors is the key to childhood obesity prevention, but few studies have been able to provide strong evidence for the range of risk factors, their interrelationships and the magnitude of their effect (Reilly *et al.* 2002). The aim of this study is to identify independent risk factors for childhood obesity. The study population is from the Avon Longitudinal Study of Parents and Children (ALSPAC), a prospective cohort study of the determinants of health and disease of approximately 14 000 children whose mothers were recruited during pregnancy, based on an expected date of delivery in 1991/1992 and residence in the Bristol–Avon area of the UK (Golding, 2001). The cohort is broadly representative of the UK. The present study was based on outcome (obesity: BMI>95th centile relative to 1990 UK data) measured in children at 7 years (90 months) in 1998/99, when 8299 of the original cohort attended clinic and 7758 (56%) had accurate weight and height measured. All potential risk factors analysed (see Table) are supported by *a priori* hypotheses and were measured between conception and age 3 years. Multivariate logistic regression models were used to analyse the data. Results are mutually adjusted odds ratios and 95% confidence intervals for obesity at 7 years, which are also independent of maternal education and energy intake (MJ) of the children measured at age 3 years.

Groups of risk factors

Perinatal	Infant feeding	Parental characteristics	Lifestyle
Birth weight	Breast feeding	Parental BMI	Food groups
Gender	Age of introduction of solids	Number of siblings	Time in car
Gestational age	Season of birth	Ethnicity	TV viewing
Smoking in pregnancy		Age of mother	Hours of sleep

Potential confounders: Maternal education, Energy intake.

Obesity prevalence at age 7 was 8.6% (671/7758) and did not differ significantly between boys and girls ($P = 0.08$). Obesity in 7-year-old children was independently associated with increased birth weight (unit per 100 g) (adjusted odds ratio: 1.05 95% confidence interval: [1.02, 1.07]), maternal smoking in the first 3 months of pregnancy (non-smoker (reference): 1–9 cigarettes per day: 1.42 [0.91, 2.20]; 10–19 per day: 2.18 [1.37, 3.45]; 20+ per day: 1.75 [0.76, 4.04]), early introduction to solid food (4 months+ (reference): 1 month: 2.05 [0.85, 4.94]; 2 months: 1.79 [1.12, 2.83]; 3 months: 1.22 [0.89, 1.67]), parental obesity (neither parent obese (reference): mother obese only: 3.12 [2.11, 4.62]; partner obese only: 4.64 [3.10, 6.94]; both parents obese: 13.19 [6.27, 27.71]) and a dietary pattern of predominantly junk-type foods. Conversely, compared with only children, children with three or more siblings were less likely to be obese at 7 years: 0.35 [0.15, 0.84] as were children who slept for 12 h or more per night at 30 months (reference) (when compared with those sleeping for 8 or fewer hours) 6.13 [1.78, 21.16]. There was an indication that obesity at 7 years was associated with sedentary behaviour in children at 36 months of age: Children viewing TV for more than 11 h (per week) were at a greater risk of obesity: 1.57 [1.05, 2.37]) (compared with children viewing fewer than 4 h of TV (reference)) as were children who spent more than 2 h travelling by car per weekday 3.38 [0.96, 11.86] (compared with no car use (reference)). Although, in an earlier stage of the analysis, exclusive breast feeding was a protective factor for obesity, this effect became non-significant in the final model.

We are extremely grateful to all the parents and children who took part in the ALSPAC study and the ALSPAC study team.

We thank the Chief Scientist Office, Edinburgh, Scotland for financing this project.

Golding J, Pembrey M, Jones R & ALSPAC Study Team (2001) *Paediatric and Perinatal Epidemiology* **15**, 74–87.
 Reilly JJ, Wilson ML, Summerbell CD & Wilson DC (2002) *Archives of Disease in Childhood* **86**, 392–395.

Seasonality in total energy expenditure and physical activity in young children in Glasgow. By A. FISHER¹, J. REILLY¹, C. MONTGOMERY¹, L. KELLY¹, J. PATON¹ and S. GRANT², ¹Child Energy Balance Research Group, University of Glasgow Division of Developmental Medicine, Yorkhill Hospitals, Glasgow, G3 8SJ and ²Faculty of Biomedical and Life Sciences, The University of Glasgow, Glasgow G12 8QQ

There are few published data on the effect of seasonality or weather on total energy expenditure (TEE) or physical activity in children. Two studies suggest some seasonal variation in total energy expenditure (Goran *et al.* 1998), and physical activity (Baranowski *et al.* 1993). Any seasonal effect would have implications both for health promotion strategies, and for the design of research on TEE or physical activity. The aim of the present study was to determine whether there was any significant seasonal variation in TEE or physical activity in a socio-economically representative sample of young children in Glasgow. We measured total daily energy expenditure (TEE, expressed per kilogram body mass) by the doubly labelled water method in 141 children, and estimated resting energy expenditure was used to calculate physical activity level (PAL). We measured total physical activity in 209 children (100 boys, mean age 4.7 years, SD 1.1; 109 girls, mean age 4.7 years, SD 1.2) using the Computer Science and Applications (CSA) accelerometer (using mean counts per minute per day as an index of total physical activity). Socio-economic status was assessed using the Carstairs deprivation score (Carstairs & Morris, 1991). The study was cross-sectional, with measurements of TEE and physical activity obtained throughout the year. Meteorological data were obtained from the local Meteorological Office. Significant seasonal variation was found in mean daily temperature, (ANOVA, $P < 0.001$), daily hours of sunlight (ANOVA, $P < 0.001$) (higher in summer and autumn) and daily rainfall (Kruskal-Wallis, $P < 0.001$) (higher in winter and spring). Multiple regression analysis (correcting for age and BMI) showed that mean daily temperature had a significant but slight effect on total physical activity in both boys and girls ($P < 0.05$). There was significant seasonal variation in total physical activity, with summer and autumn being associated with significantly higher activity than spring in both boys ($P < 0.01$) and girls ($P < 0.05$). There was a weak, but statistically significant, positive correlation between mean total physical activity and temperature at time of measurement, in both sexes: boys ($r = 0.36$, $P < 0.05$); girls ($r = 0.28$, $P < 0.05$). Multiple regression analysis (correcting for gender, age and BMI) showed a significant positive relationship between mean temperature and mean hours of sunshine and TEE/kg. There was a weak, but significant, positive correlation between TEE/kg and mean temperature ($r = 0.22$, $P < 0.05$) and TEE/kg and mean hours of sunshine ($r = 0.22$, $P < 0.05$). PAL was significantly higher in summer (1.69) than spring (1.55) or winter (1.49) ($P < 0.001$). Mean TEE was significantly lower than the age and sex specific estimated average requirement for energy by around 14%. The present study suggests that there may be some seasonal variation in total energy expenditure and physical activity in young children in Glasgow. However, although statistically significant, the effects observed were weak. It is also worth noting that levels of TEE and physical activity in our sample were low throughout the year. The magnitude of the seasonal/climatic variation observed make it unlikely that seasonal factors have to be considered when designing studies of TEE or physical activity in young children in Scotland.

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Goran MI & Nagdy TR (1999) *American Journal of Clinical Nutrition* **68**, 675–682.
Baranowski T & Thompson W (1993) *Research Quarterly for Exercise and Science* **64**, 127–133.
Carstairs V & Morris R (1991) *Aberdeen University Press*.
Black AE, Coward WA, Cole TJ & Prentice A (1996) *European Journal of Clinical Nutrition* **50**, 72–92.

An integrated programme of nutrition, exercise and behavioural modification in a small group of obese 7–11-year-old children. By P.M. SACHER¹, L. HOGAN², P. CHADWICK³ and M.S. LAWSON⁴, ¹Great Ormond Street Hospital for Children, London WC1N 3JH, ²Aquaworks Ltd, Oasis Sports Centre, London WC2H 9AG, ³St. Mary's Hospital, London W2 1NY and ⁴Institute of Child Health, London WC1N 1EH

Childhood obesity has reached epidemic proportions. Since childhood obesity tracks into adult life, this epidemic has significant health and cost implications (NAO, 2001). At present there is no proven, successful health and lifestyle treatment programme available for obese 7–11-year-olds in the UK in a primary care setting. This study sets out to test the feasibility of implementing the MEND (mind, exercise, nutrition and diet) programme in inner London. Obese children aged between 7 and 11 years were recruited via school nurses, hospital paediatricians and by local newspaper advertising. The MEND programme, which took place at a leisure centre, required twice-weekly visits by parents and children for a period of 3 months. Group sessions on nutrition and behaviour management techniques were held; some of these were for parents with their children, others were designed for parents or children separately. Weight loss was not encouraged as part of the programme and the emphasis was on healthy eating and elimination of high glycaemic index (GI) foods from the diet. The other key element of the programme was regular structured physiotherapy-guided swimming-pool-based exercise, which included a combination of competitive, non-competitive and group play activity. Other additional activities included a supermarket tour and a 'learn to cook' evening. Assessments were carried out at the beginning and end of the programme and comprised anthropometry, 3 d food diaries, fitness assessment and psychology questionnaires. Follow-up sessions are planned 3 months after the study intervention.

Eleven children (six girls, five boys) were recruited into the programme; one child dropped out due to family health problems. The remaining ten children completed the programme with an average attendance record of 78%.

Changes in measures between first and last visit

Measure	Mean	Standard deviation	P value (paired t test)	Minimum	Maximum
BMI (kg/m ²)	-0.87	0.82	0.009	-2.4	0.2
Weight (kg)	-0.56	1.37	0.23	-3.2	0.8
Waist circumference (cm)	-2.19	2.57	0.02	-6.0	1.5
Resting heart rate (bpm)	-11.6	8.73	0.002	-28	0
Self-esteem (Harter, 1985)	+0.38	0.17	<0.001	0.12	0.61

Despite there not being an emphasis on weight loss, 8/10 children reduced their BMI, 5/10 reduced their weight and 6/10 had a decreased waist circumference by the end of the programme. General fitness was improved: the resting heart rate was significantly reduced in the group, with 9/10 children showing a decrease. Self-esteem, which was measured by questionnaire, improved significantly in the group; all children showed an improved score. The pool-based exercise programme also helped to overcome the aversion to exercise that initially existed in most of the children.

The compliance and results of this feasibility study are encouraging and indicate that the provision of an attractive and appropriate programme can result in changes in body composition, fitness and outlook at least in the short term. A larger controlled trial is warranted, with follow-up for at least 12 months. The ultimate aim is to develop a programme that can be disseminated in a community environment anywhere in the UK.

For further information on MEND please contact P.M. Sacher at sacherp@gosh.nhs.uk
Harter S (1985) *Manual of the Self-Perception Profile for Children*, University of Denver Co.
National Audit Office (2001) *Tackling Obesity in England*. HC 220 Session 2000-2001. London: NAO.

Effects of conjugated linoleic acid on prostaglandins produced by the cells isolated from maternal intercofendometrium, fetal allantochorion and amnion in late pregnant ewes. By Z. CHENG, M. ELMES, D.R.E. ABAYASEKARA and D.C. WATHES, *Reproduction and Development Group, Department of Veterinary Basic Sciences, Royal Veterinary College, Hawkshead Lane, North Mymms, Hatfield, Herts AL9 7TA*

Conjugated linoleic acids (CLAs) are a collection of positionally and geometrically conjugated dienoic isomers of linoleic acid (LA). CLAs are known to possess a number of beneficial effects, including prevention of atherosclerosis and carcinogenesis (Pariza *et al.* 2001). Alteration of CLA content in animal products may, therefore, benefit human health through consumption of dairy and meat products. The anti-carcinogenic properties of CLA are at least partially attributed to its ability to interrupt the *n*-6 polyunsaturated fatty acid (PUFA) metabolic pathway for the biosynthesis of eicosanoids, including prostaglandins (PGs) (Banni *et al.* 1999). Both PGE₂ and PGF_{2α} also play key roles in parturition. In the present study, we compared the effects of CLAs (an approximately 50:50 mixture of *cis*- and *trans*-9, 11 and -10, 12- octadecadienoic acids) and LA on PG production by the cells isolated from maternal intercofendometrium (IC), fetal allantochorion (FC) and amnion (AM) from late pregnant ewes (135 d). There were three ewes per treatment and four replicates per ewe. Confluent cells were cultured with PUFAs for 45 h challenged with oxytocin (OT) or lipopolysaccharide (LPS), which are both known stimulants of PG production in the uterus and samples for PG analysis were collected 24 h latter.

	CM (No challenge)			
	CM (No challenge)	OT (250 nM)	LPS (0.1 µg/ml)	
Intercofendometrium (IC)	PGF _{2α} (ng/ml)	19.86 ± 1.95	31.86 ± 3.15 [†]	43.26 ± 4.64 [†]
	LA (100 µM)	2.36 ± 0.22*	4.75 ± 0.71* [†]	7.11 ± 1.03* [†]
	CLA (100 µM)	7.09 ± 0.41*	10.91 ± 1.07* [†]	19.14 ± 2.25* [†]
PGE ₂ (ng/ml)	CONT	13.07 ± 2.16	20.70 ± 6.19 [†]	39.31 ± 5.46 [†]
	LA (100 µM)	4.26 ± 0.69*	6.19 ± 0.68* [†]	14.69 ± 2.82* [†]
	CLA (100 µM)	13.59 ± 2.76	15.93 ± 1.17	38.69 ± 7.23 [†]
Fetal allantochorion (FC)	CONT	3.16 ± 0.33	N/A	26.67 ± 2.70 [†]
	LA (100 µM)	1.12 ± 0.12*	N/A	2.99 ± 0.62* [†]
	CLA (100 µM)	2.15 ± 0.26*	N/A	6.03 ± 0.55* [†]
Amnion (AM)	CONT	1.89 ± 0.49	N/A	18.41 ± 6.18 [†]
	LA (100 µM)	1.22 ± 0.13	N/A	2.09 ± 0.17* [†]
	CLA (100 µM)	5.06 ± 0.86*	N/A	9.99 ± 1.48* [†]

CONT, PUFA free medium; Split analysis of variance with block design and Bonferroni multiple comparisons: **P* < 0.05–0.001 v. CONT in the same column; †*P* < 0.05–0.001 v. CM in the same row.

The results demonstrated that chronic supplementation of LA and CLA significantly affected both the proportion and amount of PG produced by IC, FC and AM cells isolated from late gestation ewes and the ability of the uterus and placenta to respond to OT and LPS. LA significantly inhibited PGE₂ and PGF_{2α} produced by the above cells in the absence or presence of the challenges (*P* < 0.05–0.001). In IC cells with or without OT and LPS challenges, CLA suppressed PGF_{2α} generation significantly (*P* < 0.05–0.001). In the presence of LPS, CLA inhibited PGE₂ in FC and AM cells (*P* < 0.05–0.001). In the absence of LPS, CLA produced moderate inhibition (FC) or stimulation (AM) of PGE₂ generation (*P* < 0.05). The inhibitory effect of CLAs on PG production was less potent than LA (*P* < 0.05–0.001). These results suggest that chronic supplementation of LA or CLA may affect both the initiation and process of parturition.

Banni S, Angioni E, Casu V, Melis MP, Carta G, Corongiu FP, Thompson H & Ip C (1999) *Carcinogenesis* **20**, 1019–1024.

Pariza MW, Park Y & Cook ME (2001) *Progress in Lipid Research* **40**, 283–298.

Factors influencing the early introduction of solid feeding. By A.S. ANDERSON¹, B. ALDER², J.S. FORSYTH³, P. HOWIE³, P. VAN DER VELDE⁴ and F. WILLIAMS¹, ¹Centre for Public Health Nutrition Research, Department of Medicine, Ninewells Hospital and Medical School, Dundee DD1 9S, ²Faculty of Health and Social Sciences, Napier University, 74 Cannaan Lane, Edinburgh EH9 2TB, ³Tayside Institute of Child Health and ⁴Department of Epidemiology and Public Health, Ninewells Hospital and Medical School, Dundee DD1 9SY

Nutrition in the early years of life is considered an important determinant of growth and development, which may also influence adult health. In Scotland, around 28% of infants had received solids by 3 months (Hamlyn *et al.* 2002), and it is not clear why parents reject current guidelines.

Building on earlier qualitative work (Anderson *et al.* 2001), we aimed to investigate the determinants of the timing of introduction of solid food to infants by undertaking a prospective study in a community sample of primiparous mothers giving birth at Forth Park Maternity Hospital, Fife.

A total of 448 mothers were recruited in the postnatal ward and 338 agreed to be interviewed at home at 12 weeks postnatal on their infant feeding practices, using a questionnaire based on the 'Theory of Planned Behaviour' model. At 20 weeks postnatal they were sent a postal questionnaire with questions about their decisions and feeding behaviour, and this was returned by 286 mothers. Most mothers (70%) had initiated breastfeeding but only 15% exclusively breastfed for 12 weeks and 39% had introduced solids before 12 weeks. Bivariate analysis showed that mothers who introduced solids before 12 weeks were more likely to have a male baby (odds ratio 2.01 95% CI 1.26 to 3.21); to use formula feed (odds ratio 2.10 95% CI 1.27 to 3.47); to have received free manufactured food samples (odds ratio 2.74 95% CI 1.70 to 4.43); to have smoked cigarettes during pregnancy (odds ratio 3.27 95% CI 1.90 to 5.60); to be from a lower social class background (odds ratio 2.12 95% CI 1.28 to 3.52); and to have left full time education before 18 years (odds ratio 2.74 95% CI 1.42 to 5.37). Younger women (under 20 years) were also more likely to introduce solids early (odds ratio 2.5, 95% CI 1.21 to 5.2).

Multiple logistic regression analysis demonstrated that early introduction of solids was associated with the opinions of the infant's grandmother, living in a deprived area, personal disagreement with the advice to wait until the baby was 4 months, lack of encouragement from friends to wait until 4 months and being in receipt of free samples of manufactured food.

A number of factors influencing a woman's decision to introduce solids are amenable to change. Health programmes and professionals might usefully engage with grandmothers or friends to help deliver dietary messages and practical effective routes to discourage inappropriate use of manufactured weaning foods.

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Anderson AS, Guthrie C-A, Alder B, Forsyth S, Howie PW & Williams FLR (2001) *Journal of Health Education Research* **16**, 471–479.

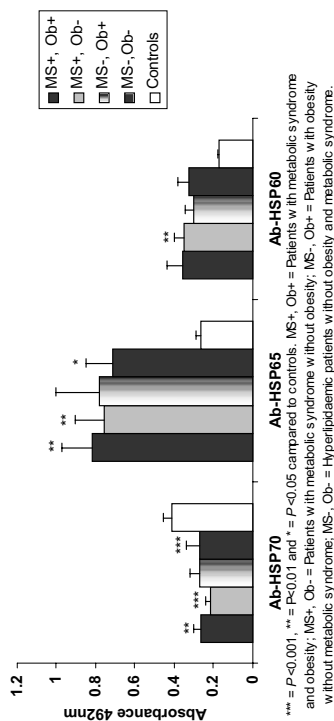
Hamlyn B, Brooker S, Oleinikova K & Wands S (2002) *Infant Feeding 2000: A survey conducted on behalf of the Department of Health, the Scottish Executive, the National Assembly for Wales and the Department of Health, Social Services and Public Safety in Northern Ireland*. London: TSO.

Antibody titres against heat shock proteins (HSPs) in metabolic syndrome and obesity. By M. GHAYOUR, D. LAMB, A. TAYLOR, C. LIVINGSTONE and G. FERNS, *Centre for Clinical Science and Measurement, School of Biomedical and Life Sciences, University of Surrey, Guildford, Surrey, GU2 7XH and The Royal Surrey County Hospital, Egerton Rd., Guildford, Surrey, GU2 7XX*

Metabolic syndrome (MS) affects up to 25% of the adult population, and hence is probably the most prevalent risk factor for cardiovascular disease (Ford *et al.* 2002). There is also good evidence that immune mechanisms play a role in atherosclerosis. HSP65 and HSP60 appear to be the most important antigens that may be responsible for this immunoreactivation during atherogenesis (Lamb *et al.* 2003). We have investigated the antibody titres against HSPs (i.e. HSP60, HSP65 and HSP70) in subjects with MS, obese patients and control subjects.

Eighty-seven patients were recruited from lipid clinics at The Royal Surrey County Hospital. Fifty-seven had MS as defined using the adult treatment panel III criteria (NCEP, 2001). Of these subjects twenty-five were obese (BMI>30, %body fat 39.2, SE 1.47), and thirty-two were not (BMI<30, %body fat 31, SE 1.38). Five subjects were obese (%body fat 37.4, SE 5.35) but did not have MS, and twenty-five were dyslipidaemic without MS and obesity (%body fat 29.8, SE 1.19). Twenty-one control subjects (%body fat 22.9, SE 1.48) were recruited from the School of Biomedical and Life Sciences of the University of Surrey. Plasma anti-HSPs titres were determined by enzyme-linked immunosorbent assay (ELISA). Analysis was carried out using the Kruskal-Wallis test for comparison of antibody titres in each group and parametric tests for normally distribution data.

Plasma anti-HSP60 titres were significantly higher in subjects with metabolic syndrome ($P<0.001$, 0.36, SE 0.04), obesity ($P<0.01$, 0.35, SE 0.06) and dyslipidaemia ($P<0.01$, 0.32, SE 0.05) compared with control subjects (0.17, SD 0.01). Also, plasma anti-HSP65 titres were significantly higher in subjects with metabolic syndrome ($P<0.001$, 0.79, SE 0.11), obesity ($P<0.001$, 0.81, SE 0.13) and dyslipidaemia ($P<0.01$, 0.72, SE 0.12) compared with control subjects (0.27, SE 0.02). Plasma anti-HSP70 titres were significantly lower in subjects with metabolic syndrome ($P<0.001$, 0.24, SE 0.02), obesity ($P<0.01$, 0.27, SE 0.03), and dyslipidaemia ($P<0.001$, 0.27, SE 0.06) compared with control subjects (0.41, SE 0.04). There were no significant differences in antibody titres for any of the HSPs between the subgroups.



Significant differences in the antibody titres against HSPs were observed in patients with metabolic syndrome, obesity and dyslipidaemia compared with control subjects. Increased antibody titres against HSP60 and HSP65 may be related to the immunoreactivation during atherogenesis in these groups.

NCEP (National Cholesterol Education Programme) (2001) *JAMA* **285**, 2846–2497.
Ford ES, Giles WH & Dietz WH (2002) *JAMA* **287**, 356–359.
Lamb DJ, El-Sankary W & Ferns GA (2003) *Atherosclerosis* (In the press).

Eating patterns and perceived healthiness of diet in the West of Scotland. By M. EBRAHIMI-MAMEGHANI¹, J.A. SCOTT¹ and G. DER², ¹Department of Human Nutrition, Glasgow University, Glasgow Royal Infirmary, Glasgow G3 7ER and ²Social and Public Health Sciences Unit, University of Glasgow, 4 Lilybank Gardens, Glasgow G12 8RZ

In order for healthy eating programmes to be effective, people must first perceive themselves to need to change their diet. Conversely, a major obstacle to nutrition promotion is the finding that many people believe that their diets are already healthy (Keamey & McElhone, 1999). The purpose of this study was to provide data on perceived healthiness of diet and to relate this to actual dietary behaviour.

Dietary data from the 35- and 55-year-old cohorts participating in the *West of Scotland 20-07 Study* were analysed (Ford *et al.* 1994). A food frequency questionnaire (FFQ) was completed by 1701 subjects and provided information on the weekly consumption of food from twenty-eight biologically different food groups. An overall food variety score (FVS) was calculated based on the consumption of these items. Perceived healthiness of diet was categorised as 'very healthy', 'healthy' and 'unhealthy' eater.

Results showed that 10% of subjects reported their diet as being 'very healthy' versus 11.2% who perceived their diet to be unhealthy. The majority of subjects perceived themselves as eating a 'healthy' diet (78.8%). Logistic regression analysis revealed that current smokers, younger subjects and those living in socially deprived areas were significantly more likely to perceive their diets as unhealthy compared with subjects who were older, living in more affluent areas and never or ex-smokers. A perceived very healthy eating pattern was significantly associated with daily consumption of brown and wholemeal bread, fruits, fresh and cooked vegetables, and frequent consumption of white and oily fish, low-fat milk and spread, and the use of liquid fat for preparing foods. These subjects also reported less frequent consumption of white bread, chips, savoury snacks, fat-enriched meat products and confectionery compared with unhealthy diet reporters. Those people who perceived their diet as being very healthy had a significantly higher FVS compared with those who thought their diet was either healthy or unhealthy (21.02 [SD 2.67] v. 19.83 [SD 3.06] and 18.71 [SD 3.60], respectively, $F=19.23$, $P<0.000$).

Results of this study revealed relatively good agreement between perceived healthiness of diet and dietary behaviour assessed by FVS. This supports data from an Irish study, which indicate that people appear to be reasonably accurate at evaluating their own diet in terms of how healthy it is (Keamey *et al.* 2001).

Ford G, Ecob R, Hunt K, Macintyre S & West P (1994) *Social Science and Medicine* **39**, 1073–1050.
Keamey JM & McElhone S (1999) *British Journal of Nutrition* **81**, Suppl. 2, S133–S137.
Keamey JM, Gibney MJ, Livingston BE, Robson PJ, Kiely M & Harrington K (2001) *Public Health Nutrition* **4**, 1117–1126.

Therapeutic Role of Probiotics: Dietitians Perspective. By D. SHAW, H. KILLALA and C. SHORT, *Yakult UK, 12-16 Telford Way, Acton, London, W3 7XS*

Currently the role which probiotics may play in the prevention and treatment of disease is receiving increasing medical interest (Marteau, 2000; Shanahan & McCarthy, 2000; Alvarez-Olmos & Oberhelman, 2001). However, few studies have evaluated their current role, or the view of dietitians regarding their use in clinical practice. The aim of the present study was to evaluate the viewpoints of UK State Registered Dietitians and their current use of probiotics within the clinical setting.

A postal questionnaire was sent to 1020 randomly selected UK State Registered Dietitians. A total of 460 (45%) of the questionnaires were returned, of which sixteen were excluded because the dietitians were no longer practicing. The results were analysed using the Minitab statistical package (version 12.0).

The respondents concluded that probiotics could play a role in clinical practice (93%). This view was reinforced by the finding that 51% stated that they currently use probiotics in the management of their clients. The top five conditions reported were antibiotic associated diarrhoea, irritable bowel syndrome, *Clostridium difficile* infection, candida infection and constipation. This suggests that the level of probiotic administration by UK State Registered Dietitians has increased since 2000 when Brice *et al.* (2000) found that 40% of a sample of UK State Registered Dietitians used probiotics in clinical practice.

The respondents that used probiotics administered them to two defined patient groups; 86% and 62% indicated administration of probiotics to adults (18-64 years of age) and older adults (65+ years of age), respectively. The level of administration to other patient groups, such as pregnant women and infants was significantly lower. This is despite research (Isolauri *et al.* 2000; Hatakka *et al.* 2001), which demonstrates positive effects of probiotic administration in infants. Thus it is clear that research findings regarding the benefits of probiotic administration in these groups are not being transmitted into practice.

Interestingly, the most common reasons given for not using probiotics in clinical practice were the apparent lack of evidence, uncertainty regarding benefits, lack of knowledge and lack of team consensus. While research on the benefits of probiotics has increased in recent years in areas such as, infantile diarrhoea, our findings highlight that the results of these clinical trials are not being transmitted effectively to interested stakeholders. The process of dissemination of research findings to health professionals is complex and it is likely that poor communication systems limit the dissemination of appropriate research findings to dietitians (Rich, 2002).

In conclusion, dietitians are actively embracing the use of probiotics in the management of their clients and are observing benefits such as amelioration of diarrhoea. However, their application is limited to a few specific areas. Further mechanistic and clinical studies are warranted to help develop the evidence base for probiotics. In addition, improvements in the communication and dissemination of research findings are necessary if health professionals are to consider probiotics as adjuncts to the clinical management of their clients.

Alvarez-Olmos MI & Oberhelman RA (2001) *Probiotic Agents and Infectious Diseases* **32**, 1567–1576.

Brice C, McKeivith B & Shortt C (2000) *Proceedings of the Nutrition Society* **60**, 79A.

Hatakka K, Savilampi E, Pönkä A, Meurman JH, Poussa T, Näse L, Saarelm M & Korpela R (2001) *BMJ* **322**, 1327–1329.

Isolauri E, Arvola T, Sitas Y, Moilanen E & Salmimen S (2000) *Clinical and Experimental Allergy* **30**, 1604–1610.

Marteau P (2000) *Lancet Perspectives* **356**, S28.

Rich MW (2002) *Journal of the American Medical Association* **287**, 1321–1323.

Shanahan F & McCarthy J (2000) *Current Gastroenterology Reports* **2**, 345–346.

Metabolism of dietary quercetin-4'-glucoside by cultured liver and intestinal cells. By B.A. GRAF¹, R. WILSON¹, S.T. CALDWELL², R.C. HARTLEY² and M.E.J. LEAN¹, ¹Department of Human Nutrition, Queen Elizabeth Building, Glasgow Royal Infirmary, Glasgow G31 2ER, U.K. ²Department of Chemistry, University of Glasgow, Glasgow G12 8QQ, U.K.

Epidemiological studies suggest that dietary intake of the flavonoid quercetin is inversely correlated with the incidence of coronary heart disease and certain cancers (Knekt *et al.* 2002). Its potential health benefits *in vivo* depend on its presence in foods and its bio-availability. In plants, quercetin is usually conjugated with sugar moieties (Haslam, 1998). Onion is a major source of dietary quercetin and mainly contains quercetin-3,4'-di-glucoside and quercetin-4'-glucoside (Moon *et al.* 2000). In rats, dietary quercetin-4'-glucoside appears almost entirely in metabolised forms in body tissues, implying that any systemic biological effects of dietary quercetin are due to its metabolites. From our results we hypothesised that the main site of metabolism was the GI-tract (Graf *et al.* 2002) and not the liver as previously suggested (Griffiths, 1982). To test this hypothesis, we investigated the metabolism of quercetin-4'-glucoside using rat intestinal epithelial cells (IEC-6) and liver epithelial cells (CC-1). After incubating the confluent cell monolayer for 72 h with 5 µM [¹⁴C]quercetin-4'-glucoside (8.3 KBq), the culture media and the cells were harvested, radiolabelled compounds were extracted and separated using HPLC with radioactivity detection.

When [¹⁴C]quercetin-4'-glucoside was incubated with the culture medium alone (without cells), it decomposed completely into unidentified, relatively polar, compounds. After cell culture, less than 5% of the administered radioactivity was within the cells, 95% was present in the culture medium in the form of metabolites as well as breakdown products. HPLC analysis revealed that liver cells formed ten and intestinal cells formed three major radiolabelled metabolites. Intestinal cells metabolised 30% of the administered [¹⁴C]quercetin-4'-glucoside, and 70% was recovered in the form of breakdown products. Liver cells metabolised 68% and only 32% was recovered in the form of breakdown products. To exclude the possibility that the metabolites were formed in the medium by enzymes released from the cells, medium was separated from cells after 72 h culture and then incubated with quercetin-4'-glucoside for 72 h. In the absence of cells no metabolites were formed, suggesting that metabolism occurs either on the cell surface or inside the cells.

In conclusion, the results confirm that intestinal epithelial cells are capable of metabolising quercetin-4'-glucoside. Although liver epithelial cells formed a larger number and quantity of metabolites, *in vivo* only intestinal cells would be exposed to the intact dietary compound, and liver metabolism is secondary.

Graf BA, Mullen W, Caldwell ST, Hartley RC, Lean MEJ and Crozier A (2002) *Free Radical Research* **36**, 94.
Griffiths LA (1982) Mammalian metabolism of flavonoids. In *The Flavonoids: Advances in Research*, pp. 68–718, [J Harborne and T Marby, editors], London: Chapman and Hall.

Haslam E (1998) *Practical Polyphenols: From Structure to Molecular Recognition and Physiological Action*. Cambridge: Cambridge University Press.

Knekt P, Kumppalainen J, Jaervinen H, Heliovaara M, Reunanen A, Hakulinen T & Aromaa A (2002) *American Journal of Clinical Nutrition* **76**, 560–568.

Moon J-H, Nakata R, Oshima S, Inakuma T & Terao J (2000) *American Journal of Regulatory, Integrative and Comparative Physiology* **279**, R461–R467.

Patient-centred outcomes in dietary research. By J.A. JACKSON¹, S. KINN² and P. DALGARNO², *School of Life Sciences, Glasgow Caledonian University, Cowcaddens Road, Glasgow G4 0BA and Nursing Research Initiative for Scotland, Glasgow Caledonian University, Cowcaddens Road, Glasgow G4 0BA*

Current NHS policy stresses the need to take patients' views into account in the provision of healthcare (Scottish Executive, 2001). As well as being involved in the process of care, patients should also have input into determining the outcome(s) of their treatment.

A literature review of dietary research was carried out to investigate whether patients had been consulted about determining the outcome measurement(s) of their treatment. The following questions were addressed: (1) What types of outcomes are commonly measured in dietary research and in what setting? (2) Were patients' views sought in determining outcomes?

In order to answer the first question, eleven databases were searched using the terms 'patient outcomes and diet' and 'dietetic and outcome'. Articles retrieved from the search were classified according to the type of outcomes measured, clinical condition concerned, and setting. Out of sixty-nine research studies which involved dietary treatment and recorded outcomes, the most commonly measured fell into the following categories: 57% biochemical or clinical, 55% dietary intake, 49% anthropometry, 23% health or nutritional status (many studies used more than one outcome). The most common clinical conditions covered by the studies were cardiovascular disease, renal disease and diabetes, and the most common setting was a hospital outpatient clinic (twenty-four out of the sixty-nine studies).

To address the second question, preliminary searches indicated that papers which considered patients' views stated that they were measuring quality of life, so this was included as a search term. The terms diet, dietetic, nutrition, diet therapy and medical nutrition therapy were combined with patient-centred, quality of life, SF36 and EuroQol (the latter two being the names of two specific questionnaires completed by patients), using the same databases as the first search. Twenty-two studies involving dietetic treatment were found in which the authors claimed that patients' views were considered or that quality of life was measured as an outcome. These papers were read and independently assessed as to whether patients were involved in determining outcome measures. In most cases, patients simply completed a pre-existing questionnaire without input into the items to be included. Some of these questionnaires did not focus on food and diet and were therefore inappropriate for use in dietary studies. Furthermore, there was confusion between measurement of health status and quality of life. Five studies were found where the investigators used quality of life instruments that involved patients' input into their design (Retterstol *et al.* 1996; DCCT Research Group, 1996; Bradley *et al.* 1999; Faro, 1999; MacCracken & Scalis, 1999). However, little detail was given about patient(s)' involvement and whether this influenced future care.

Future work should address the issue of patient involvement in the determination of outcome and how this then influences future care.

Bradley C, Todd C, Gorton T, Symonds E, Martin A & Plowright R (1999) *Quality of Life Research* **8**, 79–91.

DCCT Research Group (1996) *Diabetes Care* **19**, 195–203.

Faro (1999) *Pediatric Nursing* **25**, 247–253, 286, 267–269.

MacCracken KA & Scalis JC (1999) *Journal of the American Dietetic Association* **99**, 1554–1558.

Retterstol K, Stugaard M, Gorbitz C & Ose L (1996) *American Journal of Cardiology* **78**, 1369–1374.

Scottish Executive (2001) *Patient Focus and Public Involvement*. Edinburgh: Scottish Executive.

Changes in fasting plasma lipid concentrations after advice to follow the current dietary guidelines in healthy postmenopausal women. By S.R. AREFHOSSEINI, S. HIGGINS and C.A. EDWARDS, *Department of Human Nutrition, University of Glasgow, Glasgow G3 8SS*

The prevalence of coronary heart disease is four times higher in women after menopause, due to the loss of protection by reproductive hormones. In the UK, the current dietary guidelines promote a diet high in carbohydrate and low in fat for adults, including postmenopausal women (Department of Health, 1991). However, there is some evidence that in the short term, a diet high in carbohydrate may adversely influence plasma lipid concentrations (Jeppesen *et al.* 1997).

Eight healthy postmenopausal women (age 56.1 (SD 6.9) years); BMI 25.2 (SD 2.9) kg m⁻² participated in the study. Habitual diet was assessed by a 7-day weighed intake. On the basis of the results, subjects were advised to alter their habitual diet so that it comprised 50–55%, 30% and 15% energy from carbohydrate, fat and protein, respectively. Subjects were asked to follow this diet for 4 weeks, in a free-living situation. Fasting blood samples were obtained at baseline and after 1 and 4 weeks of the dietary intervention. Plasma total cholesterol, high-density lipoprotein (HDL) cholesterol and triacylglycerol (TAG) concentrations were measured using commercial kits (Sigma, UK). Low-density lipoprotein (LDL) cholesterol was estimated using the equation of Friedewald *et al.* (1972).

Dietary intake	Baseline (n 8)		After 1 week (n 8)		After 4 weeks (n 7)	
	Mean	SD	Mean	SD	Mean	SD
Energy (MJ)	9.2	2.1	7.1*	0.9	6.7*	0.9
% energy from						
Carbohydrate	39.4	7.6	48.5*	5.8	50.2*	4.5
Starch	21.6	5.8	27.9*	5.0	29.9*	2.8
Sugars	18.0	7.3	21.3*	7.8	21.2*	3.4
Total fat	36.0	7.6	24.8*	4.1	24.8*	1.8
SFA	13.5	4.3	7.4*	1.8	7.9*	1.4
MUFA	12.8	3.6	7.9*	1.2	8.4*	0.5
Plasma lipids (mmol/l)						
Total cholesterol	5.11	0.68	4.93	0.62	4.86	0.54
LDL cholesterol	2.92	0.76	2.79	0.66	2.78	0.62
HDL cholesterol	1.77	0.18	1.70	0.11	1.59*	0.22
TAG	0.92	0.11	0.96	0.11	1.06	0.40

SFA saturated fatty acids; MUFA monounsaturated fatty acids.

*Mean values significantly different from baseline $P < 0.05$. One subject did not complete the study.

Energy intake, percentage energy from total fat, SFA and MUFA were significantly lower ($P < 0.05$) and percentage energy from carbohydrate, starch and sugars were significantly higher ($P < 0.05$) following 1 and 4 weeks of dietary intervention. HDL cholesterol concentrations were significantly reduced ($P < 0.05$) following 4 weeks of dietary intervention.

These results show that relatively short-term changes in diet in accordance with the current dietary recommendations may have adverse effects on fasting plasma lipid concentrations. More longer term and realistic (i.e. free-living) studies are needed to investigate the influence of the current dietary guidelines on plasma lipids, in order to establish safe and appropriate dietary guidelines for postmenopausal women.

Department of Health (1991) *Dietary Reference Values for Food, Energy and Nutrients for the United Kingdom*. Report on Health and Social Subjects No. 41. London: HMSO.

Friedewald WT, Levy RI & Fredrickson DS (1972) *Clinical Chemistry* **18**, 499–502.

Jeppesen J, Schaaf P, Jones C, Zhou MY, Chen YD & Reaven GM (1997) *American Journal of Clinical Nutrition* **65**, 1027–1033.

Nutrition risk in community-living older people from the MAVIS trial (Mineral and Vitamin Intervention Study): a comparison. By A.I. STEPHEN, A.C. MILNE, A. AVENELL and G. MCNEILL for the MAVIS TRIAL GROUP, *Health Services Research Unit, University of Aberdeen, Foresterhill, Aberdeen AB25 2ZD*

The National Diet and Nutrition Survey of the Elderly (NDNS) (Finch *et al.* 1998) found multiple nutritional deficiencies in people aged ≥ 65 years. Vitamin and mineral deficiencies may contribute to morbidity and mortality from infection. The MAVIS study is a randomised controlled trial of the effect of vitamin and mineral supplementation on infections in 910 community-living elderly people, excluding people already taking supplements. We examined whether the MAVIS participants were representative of elderly people in the UK in terms of anthropometry and nutrition risk.

We compared the age, sex and BMI from the MAVIS study with the equivalent data for free-living people aged ≥ 65 years from the NDNS. Fewer MAVIS participants were over 75 years old (32% v. 39%), and fewer were over 85 years old (4% v. 7.5%). More participants were men (53% v. 50%). MAVIS participants had significantly higher BMI than NDNS participants: the mean (SD) BMI was 28.0 (3.5) kg/m² for men compared with 26.5 (3.7) ($P < 0.001$). For women the mean (SD) BMI was 28.1 (4.7), compared with 26.8 (4.7) ($P < 0.001$).

The nutrition risk of the MAVIS population was compared with data from a study of predictors of micronutrient status in a random sample of people > 75 years old (McNeill *et al.* 2002). The screening tool developed and validated by McNeill *et al.* to predict nutritional status was used in the MAVIS study. It was modelled on the ability of individual questions to predict micronutrient deficiencies. Seventeen questions about diet, activity and motivation were found to be predictors of iron, folate, vitamin C and vitamin D deficiency, and were developed into a simple questionnaire to assess nutritional risk.

	No. (%) of participants > 75 years at high risk of nutritional deficiency	
	McNeill <i>et al.</i> * n=291	MAVIS (2003) Men n=153 Women n=138
All participants > 75 years	76 (19)	12 (8)
Iron	53 (18)	12 (8)
Folate	18 (6)	9 (6)
Vitamin C	64 (16)	22 (14)
Vitamin D	60 (15)	13 (8)

*Unpublished data.

The proportions of those over 75 years classified as at nutritional risk in MAVIS were very similar to those of McNeill *et al.* but the MAVIS participants were younger (14% aged > 85 years v. 24%). They had higher average BMI: the mean (SD) BMI was 27.2 kg/m² (3.4) for MAVIS men and 25.3 (3.1) for men in McNeill ($P < 0.001$). For MAVIS women, mean (SD) BMI was 27.0 (4.1) v. 26.0 (3.8) for McNeill's women ($P < 0.05$). More participants were classified as at risk of vitamin D deficiency in the MAVIS study (20% v. 15%), χ^2 tests showed that women in the MAVIS study were significantly more at risk of iron deficiency ($P < 0.001$) and of vitamin D deficiency ($P < 0.001$) than men in the MAVIS study.

The MAVIS study participants were a self selected group (11% of the general population) who did not routinely take vitamin and mineral supplements. This may explain some of the differences found in their age, sex and BMI distribution when compared with NDNS and McNeill's participants.

Finch S, Doyle W, Lowe C, Bates C, Prentice A, Smithers G & Clarke P (1998). *National Diet and Nutrition Survey*. London: HMSO.
McNeill G, Vyvyan J, Peace H, McKie L, Seymour G, Hendry J & MacPherson I (2002). *British Journal of Nutrition* **88**, 555–561.

Intestinal transport of [2-¹⁴C]quercetin-4'-glucoside across the rat small intestine. By R. RAJIKAN¹, A. CROZIER², S.T. CALDWELL³, R.C. HARTLEY³ and C.A. EDWARDS¹,
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Flavonoids are found ubiquitously in plants (Middleton *et al.* 2000) and consumed by humans and animals as an important part of their diet. In plants, the flavonoid quercetin is present conjugated with sugar moieties (Haslam, 1998). Quercetin-4'-glucoside is one of the major quercetin conjugates in onion (Moon *et al.* 2000). Quercetin glucosides are thought to be highly bioavailable compared with free quercetin (Hollman *et al.* 1995). Hydrolysis of glucosides by the brush border membrane enzyme, lactase phloridzin hydrolase (LPH), was suggested as an important step prior to transportation across the intestinal membrane (Day *et al.* 2000). The intestinal sodium-dependant glucose transporter (SGLT1) has also been suggested to have an important role in the absorption of quercetin glucosides (Wolfgram *et al.* 2002).

In this study, the involvement of the intestinal sodium-dependant glucose transporter (SGLT1) and the enzyme lactase phloridzin hydrolase (LPH) in the absorption of radiolabelled quercetin-4'-glucoside (¹⁴CQ4G) was investigated in rat small intestine *in vitro*. Segments of rat small intestine were inverted and incubated for 1 h in a modified Krebs solution; 25 μ M ¹⁴CQ4G was then added to the mucosal solution and absorption monitored for 1 h. The addition of EDTA protected degradation of the labelled quercetin glucoside in the Krebs buffer. However, only 1.23 SD(0.16%) of the initial dose of ¹⁴CQ4G was detected inside the intestinal segment (serosal) within 1 h. There was evidence of metabolism of the glucoside. Intact ¹⁴CQ4G and free quercetin as well as several metabolites were found in both serosal and mucosal solutions after 60 min.

Incubation with Na⁺-free solution significantly reduced the transport of ¹⁴CQ4G across the intestinal membrane (0.77 [SD 0.23%], $P < 0.01$) as well as the inclusion of glucose (1.04 [SD 0.17%], $P < 0.05$) or phloridzin in the incubation solution (0.70 [SD 0.22%], $P < 0.01$). When phloridzin was included, more free quercetin was detected in the mucosal side after 60 min incubation. The results indicated that both SGLT1 and LPH play a role in the transport pathway of ¹⁴CQ4G across the intestine but the total amount absorbed over 1 h was not large and there was evidence of metabolism of the parent compound. It is not yet clear if this metabolism is due to small intestinal or microbial cells.

Day AJ, Cañada FJ, Diaz JC, Kroon PA, McLauchlan R, Faulds CB, Plumb GW, Morgan MRA & Williamson G (2000). *FEBS Letters* **468**, 166–170.
Haslam E (1998). *Practical Polyphenols: From Structure to Molecular Recognition and Physiological Action*. Cambridge: Cambridge University Press.
Hollman PCH, De Vries JHM, Van Leeuwen SD, Mengelers MJB & Katan MB (1995). *American Journal of Clinical Nutrition* **62**, 1276–1282.
Middleton E Jr, Kandaswami C & Theoharides TC (2000). *Pharmacological Reviews* **52**, 673–751.
Moon J-H, Nakata R, Oshima S, Inakuma T & Terao J (2000). *American Journal of Regulatory, Integrative and Comparative Physiology* **279**, R461–R467.
Wolfgram S, Block M & Ader P (2002). *Journal of Nutrition* **132**, 630–635.

Relationships between plasma phospholipid oleic and linoleic acid status and soluble intercellular adhesion molecule 1 concentration in healthy adults. By A.A. WINDER¹, E.A. MILES¹, S. KEW¹, T. BANERJEE¹, Y.E. FINNEGAN², A.M. MINIHANE³, C.M. WILLIAMS⁴, P.C. CALDER¹, ¹Institute of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO16 7PX and ²Hugh Sinclair Unit of Human Nutrition, School of Food Biosciences, The University of Reading, Whiteknights, Reading RG6 6AP

Intercellular adhesion molecule 1 (ICAM-1) is expressed on the surface of leucocytes and endothelial cells and plays a role in the movement of leucocytes between body compartments. ICAM-1 can be cleaved from the cell surface and circulates in the bloodstream as soluble ICAM-1 (sICAM-1). This process may be indicative of inflammation and/or endothelial dysfunction, and sICAM-1 concentrations are elevated in cardiovascular disease and in acute inflammatory disorders (for references see Miles *et al.* 2001). There is little information about the relationship between fatty acid status and sICAM-1 concentrations in humans.

Blood was collected from healthy subjects in the fasted state. Subjects were men (n 89) and women (n 68) aged 26 to 71 years (mean (SEM) 53.1 (0.9)) with a BMI of 26.5 (0.3) kg/m². Mean fasting plasma triacylglycerol (TAG) and cholesterol concentrations were 1.7 (0.1) and 5.7 (0.1) mmol/l, respectively. Plasma phospholipid (PL) fatty acid composition was determined by standard techniques and sICAM-1 concentrations were measured by ELISA.

Mean sICAM-1 concentration was 422 (13) ng/ml. sICAM-1 concentrations were not different between men and women and were not influenced by age, BMI, plasma cholesterol concentration or smoking status. There was a significant association between plasma TAG and sICAM-1 concentrations (ρ 0.165, $P=0.045$). Furthermore, there were significant associations between the proportions of oleic acid and linoleic acid in plasma PL and sICAM-1 concentration (ρ -0.299, $P<0.001$ and ρ -0.261, $P=0.016$, respectively). The negative relationship between linoleic acid in plasma PL and sICAM-1 concentration was only borderline significant when plasma TAG was controlled for ($r=-0.166$, $P=0.05$). In contrast, the negative relationship between oleic acid in plasma PL and sICAM-1 concentration remained highly significant when plasma TAG was controlled for ($r=0.356$, $P<0.001$) and when gender, age, BMI, TAG, plasma cholesterol and smoking status were all controlled for ($r=0.360$, $P<0.001$). This is the first time that this association has been described.

These results indicate that plasma oleic acid status strongly influences processes that control sICAM-1 concentration. This is consistent with studies investigating oleic acid and ICAM-1 expression on leucocytes in animals (Sanderson & Calder, 1998) and humans (Yaqoob *et al.* 1998), and suggests that oleic acid may down-regulate cellular inflammation.

The plasma samples used in this study were generated as part of a study funded by the Department for Environment, Food and Rural Affairs, Roche Vitamins and Unilever Research Vlaardingen under the Agri-Food LINK programme (MAFF-LINKAFQ111).

Miles EA, Thies F, Wallace FA, Powell JR, Hurst TL, Newsholme EA & Calder PC (2001) *Clinical Science* **100**, 91–100.
Sanderson P & Calder PC (1998) *Immunology* **94**, 79–87.
Yaqoob P, Knapper JA, Webb DH, Williams CM, Newsholme EA & Calder PC (1998) *American Journal of Clinical Nutrition* **67**, 129–135.

Use of calibrated heart rate monitors to estimate CO₂ production rate during the ¹³C-mixed triacylglycerol (MTG) breath test. By C. SLATER¹, T. PRESTON² and L.T. WEAVER¹, ¹University of Glasgow, Division of Developmental Medicine, Yorkhill Hospitals, Glasgow G3 8SJ and ²Isotope Biochemistry Laboratory, Scottish Universities Environmental Research Centre, East Kilbride, G75 0QF

The ¹³C-mixed triacylglycerol (MTG) breath test is a non-invasive measure of intraluminal fat digestion (Vantrappen *et al.* 1989). It is a useful measure of exocrine pancreatic insufficiency in children with cystic fibrosis (CF) and could be used to optimise pancreatic enzyme replacement therapy (PERT) in these children (Amari *et al.* 1997). The test has not been widely adopted in children because it lacks specificity i.e. too many false-positive results. Results are expressed as cumulative percentage dose recovered (cPDR), which requires knowledge of CO₂ production rate (VCO_2) as well as breath ¹³C enrichment. A predicted resting value of VCO_2 is often used (van Dijk-van Aalst *et al.* 2001), but even if VCO_2 is measured (Amari *et al.* 1998), this may not be the appropriate value unless the subject is confined to bed and fasts for the duration of the test. Use of resting VCO_2 when the subject is not at rest leads to an underestimation of true PDR.

After an overnight fast, six children with CF, ten healthy control children and eight healthy adults ingested 10 mg/kg ¹³C-MTG baked in a biscuit composed of oats, butter and honey (Slater *et al.* 2002). A drink of unsweetened orange juice or water was taken with the meal. Children with CF did not take their PERT with this meal. A light lunch, composed of foods with low ¹³C natural abundance (Morrison *et al.* 2000) was taken 4 h after the test meal. Children with CF took their usual dose of PERT with this meal. Heart-rate monitors were worn throughout the test. Breath samples were collected at baseline, and every 20 min for 6 h. Resting VCO_2 (RMR) was measured 3 h after the test meal using a ventilated hood indirect calorimeter. At the end of the test, heart rate (HR) was calibrated against VCO_2 by making simultaneous measurements with the subject lying on a bed, sitting, standing and during a continuous series of increasing workloads on a bicycle ergometer. Data from the calibration procedure were downloaded to an Excel spreadsheet and a sigmoid model was fitted (Slater *et al.* 2001). PDR was calculated (1) using predicted VCO_2 from body surface area (Shreeve *et al.* 1970), constant; (2) using measured resting VCO_2 , constant; (3) using VCO_2 predicted from HR around the time of the sample, variable. Physical activity level (PAL) was defined as HR-predicted cPDR/measured resting cPDR (method 3/method 2).

	Adult cPDR (6 h)	Child cPDR (6 h)	CF cPDR (6 h)
Method 1: VCO_2 predicted from body surface area	33.6 (29–43)	28.0 (18–40)	11.1 (4–29)
Method 2: VCO_2 measured resting	30.5 (26–33)	28.0 (17–43)	11.2 (5–32)
Method 3: VCO_2 predicted from heart rate	41.5 (27–45)	42.2 (33–62)	16.8 (6–42)
PAL=(Method 3/Method 2)*	1.3 (1.0–1.6)	1.6 (1.0–2.2)	1.3 (1.2–1.5)

Median (range). The CF group included one child, who was not pancreatic insufficient. *Adults remained seated throughout the test; seven control children performed the test in their own homes PAL 1.7 (1.0–2.2), three at the hospital (PAL 1.3 (1.3–1.4)); all CF children came to the hospital.

Children taking part in 'rainy-day' activities in their own homes had a more variable PAL than those coming to the hospital. The 6-h cPDR calculated from RMR in healthy children is the same as that observed previously (Amari *et al.* 1997). The use of calibrated heart-rate monitors to estimate non-resting VCO_2 eliminated false-positive results in healthy children. The positive predictive value in adults using predicted RMR was 0.63 (Vantrappen *et al.* 1989). In children in this study it was 0.71 using RMR and 1.00 using VCO_2 predicted from HR. This method improved the specificity, and can be used to determine the reference range for ¹³C-breath tests in children and the appropriate PAL to use under realistic conditions i.e. non-resting and with food intake.

Amari S, Harding M, Coward WA, Evans TJ & Weaver LT (1997) *Archives of Disease in Childhood* **76**, 394–351.
Amari S, Coward WA, Harding M & Weaver LT (1998) *British Journal of Nutrition* **79**, 541–545.
Morrison DJ, Dodson B, Slater C & Preston T (2000) *Rapid Communications in Mass Spectrometry* **14**, 1321–1324.
Shreeve VW, Ceras E & Luft R (1970) *Acta Endocrinologica* **65**, 155–169.
Slater C, Preston T, Weaver LTW (2001) *Proceedings of the Nutrition Society* **60**, 8A.
Slater C, Preston T, Morrison DJ, Weaver LT (2002) *Proceedings of the Nutrition Society* **61**, 66A.
van Dijk-van Aalst K, Van Den Driessche M, van der Schoor S, Schifflers S, Van't Westende T, Ghooys Y & Veeraman-Wauters G (2001) *Journal of Pediatric Gastroenterology and Nutrition* **32**, 579–585.
Vantrappen GR, Rutgeerts PJ, Ghooys YF & Hiele MI (1989) *Gastroenterology* **96**, 1126–1134.

Effects of an 8-day exercise programme on energy balance in males and females matched for % body fat. By R. McLAUGHLIN^{1,2}, D. MALKOVA² and M. NIMMO¹, ¹Strathclyde Institute for Biomedical Sciences, University of Strathclyde and ²Human Nutrition Department of Glasgow University, Glasgow G3 7ER

Adiposity is strongly gender related. Despite a similar food provision many more women become obese than men (Gallagher *et al.* 2000). Results comparing energy balance in men and women during exercise are scarce. It has been suggested that females may preserve their energy balance more strongly than males either by decreasing spontaneous activity (SA), defined as total energy expenditure (TEE) minus the energy cost of exercise, or by increasing energy intake (EI). Therefore, the aim of this study was to examine effects of an 8-d exercise programme on TEE, SA and EI in males and females matched for % body fat.

Subjects were eight healthy, sedentary men (age: 23±1 years, BMI: 25.3 (SD5.3) kg m⁻², body fat: 18.6(SD 7.4)%, \dot{V}_{O_2} peak: 44(SD 8) ml min⁻¹ kg⁻¹) and nine eumenorrhoeic women (age: 24 (SD 4) years, BMI: 21.8(SD 1.6) kg m⁻², body fat: 19.0(SD 5.5)%, \dot{V}_{O_2} peak: 41 (SD 6) ml min⁻¹ kg⁻¹). All measurements of females were taken during the follicular phase of the menstrual cycle.

TEE and its components, sleeping energy expenditure (SEE), sedentary energy expenditure (SEDEE) and active energy expenditure (AEE) were estimated over an 8-d control period, and an 8-d exercise period. Body composition was determined prior to the control period using hydrostatic weighing (Benke & Wilmore, 1974). Body mass (BM) and basal metabolic rate (BMR), determined using Deltatrac (Datex Instrumentation Corporation, Helsinki), were measured the day before and the day after control and exercise periods. During days 1, 3, 5 and 7 of the exercise period, subjects exercised on a cycle ergometer at the load which elicited 90% of lactate threshold. In both males and females, exercise was sufficient to expend 2.09 MJ. Subjects were required to wear heart-rate (HR) monitors and record their activity during the waking hours of both control and exercise periods. Calculations on TEE were based on individual relationships of \dot{V}_{O_2} and V_{CO_2} to HR during active and inactive conditions (Moon & Butte, 1996). Sleeping metabolic rate was estimated to be 95% of BMR (Goldberg *et al.* 1988).

Over the period of 8 d, the energy expenditure of exercise (EEX), excluding basal metabolic rate, amounted to 8.37 MJ in both males and females. This led to a significant increase in TEE: in males (control, 82.43 (SE 3.73) MJ; exercise, 95.2 (SE 4.92) MJ) and in females (control, 68.98(SE 5.21) MJ; exercise, 78.32 (SE 4.96) MJ). TEE minus EEX in the exercise period was not different from TEE in the control period in either males or females. SEE, SEDEE and AEE did not differ between control and exercise periods in either gender. Males showed no change in BM over the control (pre-control, 83.4 (SE 5.6); post-control, 83.1 (SE 5.3) kg) or exercise period (pre-exercise, 83.4(SE 5.3); post-exercise, 83.4 (SE 5.3) kg). For females there was no difference in BM over the control period (pre-control, 62.6 (SE 2.1); post-control, 62.9 (SE 2.3) kg). However, females showed a significant decrease in BM over the exercise period (pre-exercise, 62.4 (SE 1.9); post-exercise, 61.7 (SE 2.0) kg).

In conclusion, exercise over an 8-d period increased TEE without a compensatory decrease in spontaneous activity in both men and women. However, a negative energy balance was induced only in increasing dietary intake.

Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR & Sakamoto Y (2000) *American Journal of Clinical Nutrition* **72**, 694–701.
Benke AR & Wilmore IH (1974) Evaluation and regulation of body build and composition. Englewood Cliffs, NJ: Prentice Hall.
Goldberg GR, Prentice AM, Davies HL & Murgatroyd PR (1988) *European Journal of Clinical Nutrition* **42**, 137–144.
Moon JK & Butte NF (1996) *Journal of Applied Physiology* **81**, 1754–1761.

Alterations in faecal microflora and short-chain fatty acid concentrations following consumption of enteral formula by healthy subjects. By K. WHELAN¹, P.A. JUDD², V.R. PREEDY¹ and M.A. TAYLOR³, ¹Department of Nutrition and Dietetics, King's College London, London SE1 9NN, ²Lancashire Postgraduate School of Medicine and Health, University of Central Lancashire, Preston PR1 2HE and ³School of Biomedical Sciences, University of Nottingham, Nottingham NG7 2UH

Enteral tube feeding (ETF) is a common method of nutritional support for patients in both hospital and community settings. Diarrhoea is a common complication with a pathogenesis that includes an increased risk of *Clostridium difficile* infection (Bliss *et al.* 1998) and an abnormal secretion of water into the colonic lumen that is reversed by short-chain fatty acids (SCFA) (Bowling *et al.* 1993). ETF formulas have traditionally been low-residue (fibre-free); however, formulas containing fibre and the prebiotic carbohydrate fructo-oligosaccharide (FOS) are now available. The aim of this study was to investigate the effect of oral consumption of enteral formula (±fibre and FOS) on the faecal microflora and SCFA concentrations in healthy subjects in a prospective, randomised, double-blind, cross-over design.

Ten healthy subjects were randomly assigned to consume a low-residue enteral formula (Nutren 1.0, Nestlé) or a fibre and FOS-containing enteral formula (Nutren Fiber, Nestlé) as a sole source of nutrition for 2 weeks. Following a 6-week washout period, the formulas were crossed over and subjects consumed formula (±fibre and FOS) for another 2 weeks. Total 3 d faecal collection was conducted at baseline and at the end of each formula consumption phase. Major bacterial groups were quantified using fluorescent *in situ* hybridisation using the probes EUB 338 (all bacteria), Bif 164 (bifidobacteria) and EREC 482, Chis 150 and Clit 135 (major clostridia). Faecal SCFA concentrations were measured using gas-liquid chromatography and faecal pH was measured using a pH electrode.

	Low-residue formula		Fibre and FOS formula	
	Baseline	During	Baseline	During
Total bacteria (log ₁₀ bacteria/g dry faeces)	11.31 (0.02)	10.98 (0.07)*	11.33 (0.03)	11.17 (0.05)*†
Bifidobacteria	9.46 (0.25)	9.05 (0.46)	9.69 (0.21)	10.41 (0.16)*†
Clostridia	11.02 (0.04)	10.71 (0.25)	11.12 (0.04)	10.71 (0.13)*
Total SCFA (µmol/g dry faeces)	378 (59)	220 (39)*	472 (61)	332 (42)†
Acetate	218 (31)	137 (24)*	271 (36)	220 (30)†
Propionate	72 (12)	36 (8)*	92 (14)	58 (12)†
Butyrate	59 (17)	19 (4)*	72 (13)	25 (4)*
Faecal pH	6.85 (0.13)	7.80 (0.12)*	6.68 (0.1)	7.59 (0.08)*†

Values are mean (SEM)
* Significantly different from baseline ($P<0.05$).
† Significantly different from during low-residue formula ($P<0.05$).

Consumption of the low-residue formula resulted in a major reduction in total faecal bacteria, which was partially prevented by supplementation with fibre and FOS (51% v. 32% reduction, $P=0.005$). Furthermore, the fibre and FOS formula resulted in an increase in bifidobacteria and a reduction in clostridia. The low-residue formula caused a reduction in the concentrations of major SCFA, whereas the fibre and FOS formula prevented significant reductions in SCFA except for butyrate. Consumption of both formulas caused an increase in faecal pH; however, faecal pH was lower when subjects consumed the fibre and FOS formula.

Consumption of low-residue enteral formula results in adverse changes to the faecal microflora, SCFA concentrations and pH. Supplementation of the formula with fibre and FOS partially prevents some of these changes. Whether such changes are involved in the pathogenesis of ETF diarrhoea and whether fibre and FOS-containing formulas have similar effects on the flora of patients receiving ETF remains to be investigated.

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Bliss DZ, Johnson S, Savik K, Clabots CR, Willard K & Gerding DN (1998) *Annals of Internal Medicine* **129**, 1012–1019.
Bowling TE, Rammundo AH, Grimble GK & Silk DBA (1993) *Lancet* **342**, 1266–1268.