

*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.*

## **PROCEEDINGS OF THE NUTRITION SOCIETY**

### **ABSTRACTS OF COMMUNICATIONS**

*A Scientific Meeting was held at the University of Southampton on Tuesday–Friday, 2–5 August 1994, when the following paper was presented. This paper arrived too late for inclusion in Volume 54 no. 2.*

**Validation of a food-frequency questionnaire in a small sample of children aged 12-24 months.** By M.A. HIRVING<sup>1</sup>, M.S. LAWSON<sup>2</sup>, C. GOODHART<sup>3</sup> and C. DOGAN<sup>3</sup>, <sup>1</sup>*School of Life Sciences, University of North London N7 8DB*, <sup>2</sup>*Medical Unit, Institute of Child Health, London WC1 N1EH* and <sup>3</sup>*Statham Grove Surgery, London N16 9DP*

The frequency of Fe-deficiency anaemia in pre-school age children can be as high as 30% in some UK populations (Wharton, 1989), and results in significant developmental delay. A rapid and easily administered means of identification of those at particular risk who may require advice and haematological testing could be an important preventative measure. A food-frequency questionnaire (FFQ) with supplementary questions has been developed and used in conjunction with an on-going blood screening programme for children aged 12-24 months in north London. The FFQ comprised eighteen foods which impact on Fe status with seven frequency categories. In order to test whether the FFQ accurately reflected food consumption, a 5 d weighed intake (WI) was carried out by six families 7-30 d after completion of the FFQ.

Food	Servings/week				Method agreement* (%)
	WI		FFQ		
	Mean	SD	Mean	SD	
Meat	4.2	3.0	4.8	2.5	66
Fish	1.5	1.4	1.7	1.3	50
Bread	5.2	2.8	5.3	2.6	100
Cereals	6.2	1.6	6.2	2.0	83
Pulses	2.2	2.6	4.8	2.5	50
Milk	10.8	5.2	10.5	5.9	66
Vegetables	9.8	4.8	7.3	3.8	66
Fruit/juice	12.8	2.9	12.8	2.9	66
Mean	—		—		68

\*Agreement to within  $\pm 2$  servings per week.

There was an overall tendency for parents to overestimate consumption frequency, with pulses being most frequently misclassified. Foods eaten frequently (more than once daily) were also more likely to be incorrectly estimated. The mean correlation (Pearson's  $r$ ) between the methods was 0.78 ( $P < 0.0001$ ), which is greater than that found by other authors when assessing intake of nutrients or foods (Mullen *et al.* 1984; Gelissen & Roberts, 1992).

FFQ have been used to assess intake and predict risk factors in adults but their use has not been tested in very young children. It can be concluded that the FFQ can be used to predict food consumption frequency in children aged 12-24 months, although some allowance should be made for possible underestimation of consumption of frequently eaten food items.

Gelissen, I.C. & Roberts, D.C. (1992). *Journal of Human Nutrition and Dietetics* **5**, 215-223.

Mullen, B.J., Krantzler, N.J., Grivetti, L.E., Schutz, H.G. & Meiselman, H.L. (1984). *American Journal of Clinical Nutrition* **39**, 136-143.

Wharton, B.A. (1989). *Acta Paediatrica Scandinavica* **361** Suppl., 5-11.

*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.*

## **PROCEEDINGS OF THE NUTRITION SOCIETY**

### **ABSTRACTS OF COMMUNICATIONS**

*A Scientific Meeting was held at the Royal Society of Medicine, London, on Friday, 17 January 1995, when the following papers were presented.*

**Vitamin D activity of meat.** By S.M. LEE<sup>1</sup>, D.H. BUSS<sup>1</sup>, and D. HATTON<sup>2</sup>, <sup>1</sup>Ministry of Agriculture, Fisheries and Food, Nobel House, 17 Smith Square, London SW1P 3JR, and <sup>2</sup>Laboratory of the Government Chemist, Queens Road, Teddington, Middlesex TW11 0LY

Although it has generally been considered that meat contains very little vitamin D, low intakes of meat have been associated with rickets and osteomalacia in Asians in the UK (Henderson *et al.* 1987, 1990). This has led to suggestions that meat may contain more vitamin D and vitamin D metabolites than previously supposed. Preliminary work detected the presence of the vitamin D metabolite 25-hydroxycholecalciferol (25(OH)D<sub>3</sub>), but not of cholecalciferol itself, in beef and chicken (Ministry of Agriculture, Fisheries and Food, 1994). Further analyses have now been carried out as part of a large survey of the nutrient content of retail cuts of carcass meat. Cholecalciferol and 25(OH)D<sub>3</sub> in selected raw and cooked cuts were determined using reverse-phase HPLC. Preliminary results are presented here.

	<i>n</i>	µg/kg edible portion			
		Cholecalciferol		25(OH)D <sub>3</sub>	
		Mean	Max.	Mean	Max.
Beef	7	<1	<1	1.2	1.8
Veal	1	12	-	0.3	-
Pork	9	3	11	0.8	1.2
Lamb	11	3	5	0.5	0.8

The results for the one veal sample analysed suggest that veal is high in cholecalciferol (possibly from feed) but low in 25(OH)D<sub>3</sub> (the form present in blood). In contrast, beef contains little or no cholecalciferol but higher levels of 25(OH)D<sub>3</sub>. There were no obvious trends with cooking or amount of fat in the cut. Cholecalciferol levels in pork varied from not detected to 11 µg/kg but were about 3 µg/kg in most samples and were higher in fat than in lean. Levels of 25(OH)D<sub>3</sub> were mostly about 0.8 µg/kg but the metabolite was not detected in one sample. Cholecalciferol was not detected in four lamb samples but was present at levels of about 5 µg/kg in six samples. The metabolite 25(OH)D<sub>3</sub> was present at levels of up to 0.8 µg/kg with more in lean than in fat.

If it is assumed that 25(OH)D<sub>3</sub> has a potency five times that of cholecalciferol, the vitamin D activity for an average portion of meat (say 90-150 g) could be about 1 µg. For example, an average pork chop would contain about 1.2 µg vitamin D activity, while a medium portion of roast lamb would contain about 0.6 µg. Thus, current estimations of vitamin D intake (about 3 µg/d) might be increased by 20% or more and intakes in high consumers of meat by even more. These findings have particular relevance to groups in the UK population for whom dietary sources are important because of insufficient exposure to sunlight or reduced synthesis in the skin (e.g. Asians, elderly people). Further work to determine levels in different types of poultry and other foods is under way.

Henderson, J.B., Dunnigan, M.G., McIntosh, W.B., Motaal, A.A., Gettinby, G. & Glekin, B.M. (1987). *Quarterly Journal of Medicine* 63, 413-425.

Henderson, J.B., Dunnigan, M.G., McIntosh, W.B., Motaal, A.A. & Hole, D. (1990). *Quarterly Journal of Medicine* 76, 923-933.

Ministry of Agriculture, Fisheries and Food (1994). *The British Diet: Finding the Facts, 1989-1993. Food Surveillance Paper No. 40.* London: H.M. Stationery Office.

**Current selenium content of foods and an estimation of average intake in the United Kingdom.**

By M.A. BUTCHER<sup>1</sup>, P.A. JUDD<sup>1</sup>, C. CAYGILL<sup>2</sup>, S. PEACH<sup>3</sup> and A.T. DIPLOCK<sup>3</sup>, <sup>1</sup>*Department of Nutrition and Dietetics, King's College London, Campden Hill Road, London W8 7AH*, <sup>2</sup>*PHLS-CDSC, 61 Colindale Avenue, London NW9 5HT*, <sup>3</sup>*Free Radical Research Group, UMDS, Guy's Hospital, St Thomas Street, London SE1 9RT*

As part of a study investigating the role of diet in gastric cancer development we have measured the Se content of a range of commonly eaten foods. Currently multiple samples of over 100 foods have been measured including those foods described as important Se contributors in the UK diet. From these we have selected the foods previously analysed by Thorn *et al.* (1978), which they used to estimate Se intakes in the UK at that time, and compared them with the current Se levels.

Se measurements were made using a modification of the method of Olson (1969), i.e. wet digestion, followed by derivatization with diammononaphthalene and fluorometric measurement.

Direct comparison of the results shows that the majority of the foods now have higher Se levels than in 1978. This is particularly true for meat; beef contained 0.03 µgSe/g in 1978 compared with 0.14 µg/g in the present study; fats: margarines previously contained <0.01 µg/g but now contain 0.13 µg/g; eggs: 0.06 µg/g in 1978 compared to 0.32 µg/g currently. Values for foods in the cereal group are comparable with those measured by Thorn *et al.*, though some lower values were found in this study, e.g. breadmaking flour, 0.42 µg/g in Thorn's study compared with 0.14 µg/g in the present study and wholemeal flour, 0.53 µg/g in 1978 compared to 0.07 µg/g now.

Using these data and additional analyses for commonly eaten foods, i.e. bread and chicken, we have attempted to estimate the 'average' Se intake in the UK using MAFF figures for 1992.

Food Group	Se Content (µg/g)		Estimated weight of food eaten (g/day)	Se Intake	Se Intake
	Range	Mean		Present Study (µg/person per d)	Thorn <i>et al.</i> (µg/person per d)
Cereals	0.038-0.073	0.054	209	11	30
Meat	0.064-0.249	0.181	135	24	17
Fish	0.206-0.396	0.330	20	7	5
Dairy and eggs	<0.01-0.313	0.127	40	5	4
Fats	0.128-0.158	0.141	35	5	1
Vegetables	<0.01-0.143	0.022	313	7	2
Fruit	0.020-0.027	0.023	133	3	1
Total				62	60

The total intake today is equivalent to that in 1978 but the sources of the Se in the diet have changed. In 1978 50% of the Se intake came from the consumption of cereals but in the current study that proportion has fallen to 18%. Many of the other food groups contribute far more to the total Se intake, particularly meat (up to 39% of the intake from 28% in 1978), fats (up to 8% from 1.7%) and vegetables (up to 11% from 3%). The results suggest that the change in the contributions made by the different food groups may affect some sections of the population, e.g. those on vegetarian (and particularly vegan) diets or people on low incomes may now have lower Se intakes. This study also confirms the findings of Barclay & MacPherson (1993) of falling Se levels in flour used in the UK.

This research was supported by the World Cancer Research Fund.

Barclay M.N.I. & MacPherson A (1993). *British Journal of Nutrition* **68**; 261-270.

Ministry of Agriculture, Fisheries and Food (1992). *Household Food Consumption and Expenditure*. H.M.S.O.

Olson O.E. (1969). *Journal of the Association of Official Analytical Chemists* **52**; 627-634.

Thorn J, Robertson J, Buss D.H and Bunton N.G (1978). *British Journal of Nutrition* **39**; 391-396.

**Food patterns and nutrient intake in short-stature children.** By CATHERINE M. POLLARD<sup>1</sup>, STEPHEN A. WOOTTON<sup>1</sup> and PETER R. BETTS<sup>2</sup>, <sup>1</sup>*Institute of Human Nutrition, University of Southampton, Southampton SO16 7PX* and <sup>2</sup>*Department of Paediatrics, Southampton General Hospital, Southampton SO16 6YD*

In a previous study we demonstrated that children of short stature with and without growth hormone treatment exhibited higher energy intakes and energy expenditure at rest when expressed per unit body weight than children of normal stature (Smith *et al.* 1992). The present study determined whether these changes in energy metabolism were associated with differences in the types of food consumed and nutrient intake. Weighed food intake was recorded over 7 d in (1) short children age 9-10 years who were <-2.0 SD for height (8M/7F; *n* 15), (2) short children receiving growth hormone (Genotropin 30 IU/m<sup>2</sup>/week; 8M/7F; *n* 15) and (3) children of normal stature, age- and sex-matched (9M/7F; *n* 16). Each item of food and drink was assigned to a major food group category according to Paul & Southgate (1978) and the relative contribution made by each food group to total energy intake was determined (%E). Nutrient intakes were determined using computerized food composition tables (Microdiet).

	Short stature		Short treated		Normal stature	
	Mean	SD	Mean	SD	Mean	SD
Energy (MJ/d)	6.23 <sup>a</sup>	1.03	8.18 <sup>b</sup>	1.24	7.72 <sup>b</sup>	1.2
Calcium (mg/d)	505 <sup>a</sup>	140	634 <sup>ab</sup>	142	802 <sup>b</sup>	293
Zinc (mg/d)	4.57 <sup>a</sup>	1.37	6.11 <sup>b</sup>	1.47	6.96 <sup>b</sup>	1.25
Confectionery (%E)	19.6 <sup>a</sup>	8.4	20.8 <sup>b</sup>	5.1	13.1 <sup>a</sup>	9.0
Crisps/chips (%E)	13.2 <sup>a</sup>	4.9	12.3 <sup>a</sup>	5.8	7.90 <sup>b</sup>	5.9
Veg/fruit (%E)	4.9 <sup>a</sup>	2.9	5.6 <sup>a</sup>	3.3	9.9 <sup>b</sup>	5.1

Mean values within a row with unlike superscript letters were significantly different  $P < 0.05$ .

There were no differences between the groups in the relative proportions of energy derived from carbohydrate and fat although the contribution from protein was slightly lower in both short-stature groups than normals ( $P < 0.05$ ). For most nutrients, the differences between groups expressed in absolute terms could be attributed to differences in energy intake (i.e. comparable nutrient densities). However the Ca and Zn intakes of both short-stature groups were lower than those of normals when expressed either in absolute terms or corrected for differences in energy intake (Ca 81 and 78 mg/MJ v. 103 mg/MJ both  $P < 0.05$ ; Zn 0.73 and 0.70 mg/MJ v. 0.90 mg/MJ both  $P < 0.05$ ). Both short-stature groups obtained a greater proportion of their energy from confectionery, crisps and chips than the normal-stature group with less energy coming from fruit and vegetables. These results suggest that the higher energy intakes of short-stature children may be associated with differences in the pattern of food intake which may in turn influence micronutrient intake, particularly Ca and Zn. Further studies are required to determine the factors that influence the pattern of food intake in these children.

Paul, A.A. & Southgate D.A.T. (1978). *McCance & Widdowson's The Composition of Foods*. 4th Ed. London: H.M. Stationery Office.

Smith, C.M., McCaughey E.S., Betts P.R., Bond S.A., Wootton S.A. & Jackson A.A. (1993). *Proceedings of the Nutrition Society* 52, 98A.

**Diversity of diets amongst South Asian Muslims in Britain. By Tashmin KASSAM KHAMIS, JANE E THOMAS, and PATRICIA A JUDD. Department of Nutrition and Dietetics, King's College London, London W8 7 AH**

Evidence suggests that diet may contribute to different patterns of diseases between the South Asian and host communities. However our understanding of the causative role of diet and the consequent opportunity for disease prevention is handicapped by a fundamental ignorance of both the composition of traditional foods of the various south Asian communities in the UK and their diverse eating habits. Three South Asian groups in London, originating from Bangladesh, Pakistan and East Africa (Ismailis) were investigated in order to develop a nutrient composition database on the commonest traditional dishes consumed and to identify food habits and the factors affecting food choice. Data were obtained by 7 d menu records or, in cases of illiteracy, by repeated 24 h recalls. Questionnaires were also administered. The study of ninety-two households in total (291 subjects aged 12 years and over) showed that the most commonly consumed traditional dishes (those dishes consumed at least once during the recording period by a minimum of 20% of households) were different amongst the different Muslim groups. Preliminary results are discussed with analysis still ongoing.

In the Bangladeshi group traditional dishes consisted mainly of rice and fish imported from Bangladesh, chicken and lamb curries, often with vegetables added, stir fried vegetable "bhajis" and massoor dhal. For the Pakistani group "roti" (unleavened bread) was the most common staple, although rice was also used. Lamb and chicken were the preferred meats, with curried vegetables and a large variety of dhals and "mithai" (sweetmeats) also popular. Ismailis ate both rice and "roti", preferring chicken, beef and lamb meats. Consumption of vegetable curries and dhals was less common but fried snacks (eg "ganthia", "thepla", "bhajias") were popular.

The younger generation in each group was shown to be more acculturated in its food habits than the older generation, though differences were seen between the groups. For example, outside eating of non-traditional foods was more common to children than parents in all groups and more significant differences were seen in the Bangladeshi and Pakistani groups. This is illustrated in the table which shows the percentage of each group and generation consuming "fast foods".

Generation	Bangladeshis (N 100)	Pakistanis (N 108)	Ismailis (N 83)
Older	48	47	90
Younger	90	86	100

Fewer children practised religious food observances than their parents. Differences between the older and younger generation in social activities, lifestyles and language proficiency pointed to more contact by the younger group with the host community. The degree of acculturation of eating habits appears to be related to the level of exposure to the host culture. People of the older generation, shown to be less exposed to the host community, were the main food purchasers and preparers in the home and as a result meals eaten at home by all groups tended to be more traditional. These generational differences have important consequences for health promotion targeting.

Variation between the groups was also seen in the nutrient composition of dishes with the same name (Kassam *et al.* 1993). It is vital to distinguish between different South Asian groups and collection of sub-ethnic, group specific food composition data is necessary to provide an informed basis for the provision of dietary advice.

This work was supported by the Aga Khan Foundation UK, Dept. of Health and MAFF.

Kassam, T.N., Judd, P.A. & Thomas, J.E. (1993). *XV International Congress of Nutrition, Abstract 291*. London, Smith - Gordon.

**A feasibility study of a monounsaturated fatty-acid-rich diet in middle-aged subjects.** By J.A. TREDGER, C. CULVERWELL, J. KNAPPER, J. WRIGHT & C.M. WILLIAMS, *The Nutrition and Food Science Research Centre, University of Surrey, Guildford GU2 5XH*

Some doubt has been expressed about the dietary feasibility and consumer acceptability of current dietary guidelines for fat intake (Department of Health, 1991), which recommend population intakes for total and saturated fatty acids of 33% and 10% food energy respectively. As an alternative dietary strategy we are studying the feasibility of and metabolic responses to a diet in which significant amounts of saturated fatty acids are substituted by monounsaturated fatty acids (MUFA) and have carried out a pilot study in middle-aged volunteers.

Eight healthy volunteers (six males, two females) were recruited to undertake a dietary intervention of a high MUFA diet for a period of 1 month. Substitute milk (Dairy Crest), margarine (Olive Gold), extra virgin olive oil for cooking and a selection of cakes and biscuits produced using a combination of milk, margarine and olive oil, were provided on a weekly basis for daily consumption. In addition each subject was provided with frozen recipe meals for 20 out of 28 d of the study. Recipe meals were evaluated by preference testing against meals prepared using standard recipe oils and fats before commencing the study. A diet diary was kept by volunteers for each day of the study and a weighed diet record was undertaken for 2 d each week. Fasting blood samples were collected at the start and at 2 and 4 weeks of the study for measurement of plasma triacylglycerol (TAG), cholesterol and glucose concentrations.

Mean scores for the preference testing for the MUFA-enriched meals (6.49(SD 0.93)) were not significantly different from those for the standard meals (6.67(SD 0.89)). The percentage energy as fat, percentage energy as SFA and percentage energy as MUFA were 43, 6 and 24 respectively for MUFA-enriched meals compared to 43, 22 and 13 for the standard meals.

Evaluation of the dietary intakes of the subjects showed that during the study, MUFA intake increased from 11 to 18% energy, total fat intake decreased from 38 to 32% dietary energy, with SFA decreasing from 16 to 7% of energy. Biochemical evaluations are shown in the Table.

	Baseline		4 weeks		Change from Baseline	
	Mean	SD	Mean	SD	Mean	SD
Plasma TAG (mmol/l)	1.09	0.37	0.90	0.23	-0.20 *	0.18
Plasma cholesterol (mmol/l)	5.91	1.30	4.56	0.71	-1.41 **	1.20
Plasma glucose (mmol/l)	6.52	1.52	6.11	1.12	+0.46	1.57

Significant difference \* $p < 0.05$ , \*\* $p < 0.01$ .

During the study there was a significant decrease in fasting plasma TAG and cholesterol concentrations. However dietary evaluation showed that percentage energy intake from total fat also decreased during the intervention period, so that the observed changes cannot be attributed solely to the target intervention. In future studies more attention will need to be placed on the maintenance of individual habitual energy intakes, by providing a wider range of fat-substituted foods than has been possible in the present study.

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: H.M. Stationary Office.



**An evaluation of the feasibility of sip-feed supplementation in undernourished, acutely sick, elderly patients.** By J.J. REILLY<sup>1</sup>, M. MACKINTOSH<sup>1</sup>, J. POTTER<sup>2</sup> and M.A. ROBERTS<sup>2</sup> <sup>1</sup>University of Glasgow Department of Human Nutrition, Yorkhill Hospitals, Glasgow G3 8SJ and <sup>2</sup>Victoria Geriatric Unit, 100 Mansionhouse Road, Glasgow G41 3DX

We have previously shown that, in a heterogenous population of elderly patients admitted to hospital with acute illness, (a) undernutrition is prevalent (Potter *et al*, 1995); (b) hospitalization is characterised by negative energy balance and further deterioration in nutritional status (Klipstein-Grobusch *et al*, 1995); (c) Undernutrition is associated with adverse clinical outcome (Potter *et al*, 1995).

The aims of the present study were to test if a novel means of providing nutritional support to these patients could: achieve successful consumption of supplement; permit quantification of consumption; lead to improvements in energy balance characteristics and/or nutritional status during hospitalization. During an 8-week period all new admissions to the unit (n 128) received an anthropometric nutritional assessment within 2d of admission. Those meeting published criteria for undernutrition (BMI <25th centile relative to UK reference values (Burr and Phillips, 1984) and/or corrected arm muscle area < 16.0 cm<sup>2</sup> men; < 16.9 cm<sup>2</sup> women (Lipski *et al*, 1993; n 37) and considered at risk were randomised to receive either standard care (controls, n 17) or prescribed 300 ml of 6.3 kJ/ml sip-feed supplement (intervention group, n 20) given as 3 x 100 ml aliquots each day at drug rounds. Consumption of supplement was recorded by nursing staff. In all patients body weight, triceps skinfold and mid arm circumference were measured within 2d of admission and at 14d (or discharge if earlier). Energy intake was measured by weighed dietary record on day 3 or 4 and day 11 or 12 of admission in all patients.

In the intervention group 15 patients consumed all prescribed supplement during their hospital stay. One patient consumed an average of 100 ml supplement/d while the remaining four refused all supplementation. In the control group median energy intake (EI) was 102 (range 43-280) kJ/kg per day, and median EI: predicted BMR/ratio 1.0 (range 0.6-2.0). In the intervention group median voluntary food energy intake was 106 (range 12-236) kJ/kg per day and median food EI: predicted BMR/ratio 1.0 (range 0.2-2.0). With inclusion of energy intake from supplements median energy intake in the intervention group increased to 155 kJ/kg/per d and median EI: predicted BMR/ratio to 1.5. No significant differences in group mean changes in weight or corrected arm muscle area were observed between intervention or control group, though a trend to reduced deterioration in nutritional status in the intervention group was noted.

We conclude that compliance with this simple regime of sip-feed supplement provision is generally good in these patients. Furthermore, compliance can be quantified adequately. Supplementation may have some benefits in preventing the deterioration in nutritional status characteristic of hospitalization in this patient population by producing changes from negative to positive energy balance. Further research is necessary in order to assess the functional and clinical effects of such an intervention.

We acknowledge the assistance of the Department of Dietetics, Victoria Geriatric Unit.

Burr, M.L. & Phillips, K.M. (1984). *British Journal of Nutrition* 51: 165-169.

Klipstein-Grobusch, K., Reilly, J.J., Potter, J., Edwards, C.A. & Roberts, M.A. (1995). *British Journal of Nutrition* 73: 323-334.

Lipski, P.S., Torrance, A., Kelly, P.J. & James, O.F.W. (1993). *Age and Ageing* 22: 244-255.

Potter, J., Klipstein-Grobusch, K., Reilly, J.J. & Roberts, M.A. (1995). *Age and Ageing*, In the Press.

**Coordinated response of liver and muscle protein synthesis to fasting and refeeding in 12-month-old rats.** By LAURENT MOSONI, THIERRY MALMEZAT, CHRISTIANE OBLED and PHILIPPE PATUREAU MIRAND, *Laboratoire d'Etude du Métabolisme Azoté, INRA, Theix, France*

The effect of fasting on protein metabolism has been well characterized (loss of body proteins, inhibition of tissue protein synthesis, stimulation of protein degradation). However, most experiments have been performed in young growing animals and, although marked changes occur in protein metabolism during ageing, few experiments have been performed in adults. From these studies, it seems that age-related differences in the adaptation to starvation are that older rats have a higher capacity to spare muscle proteins, and a lower sensitivity of muscle protein synthesis not only to food deprivation, but also to refeeding (Goodman *et al.* 1981; Baillie & Garlick, 1992). By measuring protein and RNA content, as well as *in vivo* protein synthesis rates using a flooding dose of valine (with 50% [1-<sup>13</sup>C]valine), we analysed more precisely how muscle and liver protein synthesis adapted to 113 h fasting and to 6 h refeeding in mature animals using 12-month-old male Sprague-Dawley rats (n=6 per group).

	Tibialis anterior muscle				Liver			
	Fed	Fasted	Refed	SE	Fed	Fasted	Refed	SE
Total protein (g)	0.200	0.188	0.185	0.005	3.09	2.07*	2.45*†	0.10
Total RNA (mg)	1.00	0.60*	0.58*	0.04	119	96	107	5
FSR (%/ d)	5.35	4.17*	3.63*	0.16	55.1	40.5*	52.2†	0.8
Cs (mg RNA/ g protein)	4.97	3.25*	3.12*	0.19	38.5	46.3*	43.6*	0.9
K <sub>ma</sub> (mg protein/d per mg RNA)	11.2	12.5	11.7	0.4	14.5	8.8*	12.0*†	0.3

FSR, fractional synthesis rates; Cs, capacity for protein synthesis, K<sub>ma</sub>, ribosomal efficiency.

\* Significantly different from fed rats, P≤0.05.

† Significantly different from fasted rats, P≤0.05.

The results summarized in the Table confirmed that even after 113 h without food, 12-month-old rats could spare muscle proteins during fasting at the expense of liver proteins, which were markedly depressed. Protein synthesis was significantly decreased in both tissues, due to a loss of ribosomes in muscle (as reflected by total RNA measurements), and due to a drop in ribosomal efficiency in liver (whereas liver ribosomes were maintained). After 6 h refeeding, liver protein mass had already significantly increased, not only because of a stimulation of protein synthesis and ribosomal efficiency, but also probably as a result of a marked inhibition of protein degradation. Muscle protein synthesis was not stimulated by refeeding, which reflects an age-related insensitivity of muscle ribosomal efficiency. However, this lack of stimulation allowed amino acids to be used in other tissues like liver. In conclusion, coordinated responses of liver and muscle protein synthesis, based on opposite control of ribosome number, allowed a sparing of muscle proteins during fasting, and a rapid recovery of liver proteins during refeeding.

Baillie, A.G.S. & Garlick, P.J. (1992). *American Journal of Physiology* **262**, E1-E5.

Goodman, M.N., McElaney, M.A. & Ruderman, N.B. (1981). *American Journal of Physiology* **241**, E321-E327.

**Diurnal cycling in urea-nitrogen hydrolysis.** By T.S. MEAKINS and A.A. JACKSON, *Department of Human Nutrition, University of Southampton, Southampton SO16 7PX*

Based on urinary N excretion, protein intake has been taken to determine the amplitude of diurnal cycling of protein turnover (Quevedo *et al* 1994). Further, from the pattern of urinary N excretion, diurnal changes in urea production have been imputed (El Khouri *et al* 1994) which do not stand up to critical analysis. Recalculation of these data indicates a relatively constant rate of urea formation over the day, with large changes in the hydrolysis of urea N between day (0% production) and night (50% production) (Jackson 1994). In the present study the pattern of hydrolysis of urea N in the colon over a 24 h period has been determined.

Seven adult males were given diets containing 126 kJ/kg per d and 0.95 g protein/kg per d for 5 d. Over the final 33 h, urea kinetics were measured for each 3 h period using the prime/intermittent oral dose method (Jackson *et al* 1984), with analysis for the final 24 h period.

Urea	24 h Period		03.00 - 15.00 hours		15.00 - 03.00 hours	
	Mean	SEM	Mean	SEM	Mean	SEM
Production(mgN/kg perh)	7.65	0.3	7.51	0.5	7.80	0.4
Hydrolysis(mgN/kg perh)	4.71	0.3	3.70	0.3	5.74*	0.5
Excretion (mgN/kg perh)	2.94	0.4	3.81	0.5	2.06*	0.6

\* Mean values for 15.00-03.00 hours were significantly different from 03.00-15.00 hours,  $P < 0.05$  (paired rank test).

Protein was taken in every 3 h from 06.00 to 21.00 hours. However, urea production was virtually constant throughout the 24 h period. Excretion of urea in urine was lowest in the morning, rising to a peak at about 16.00 hours and falling during the evening, and, when compared with the average for the entire 24 h period, was consistently above average from 15.00 to 24.00 hours and consistently below average between 03.00 and 12.00 hours. Hydrolysis of urea by the colonic microflora mirrored the changes in excretion. Six to nine hours after feeding the hydrolysis of urea-N decreased and was on average 26% of urea production. Six hours after feeding had ceased, salvage increased to an average of 50% of production. We have previously argued that the rate of urea-hydrolysis and salvage is primarily determined by the N substrate available for metabolism to the colonic microflora. As small-intestinal transit is about 6 h, our results would support this suggestion, and indicate that the proportion of urea production which does not pass to the colon is excreted.

We have previously considered that the hydrolysis and salvage of urea-N is a fundamental aspect of metabolic adaptation over a period of days, to lowered N intake. The present study demonstrates that it may also be of significance in enabling the body to sustain homeostasis in the short term, in response to a diurnal cycle in food intake. If true, there are clear implications for the calculation of the non-protein respiratory exchange ratio by indirect calorimetry and in the derivation of protein degradation in studies of protein turnover.

El-Khouri A.E. Fukagawa N.K. Sanchez M. Tsay R.H. Gleason R.E. Chapman T.T. & Young V.R. (1994). *American Journal of Clinical Nutrition* **59**, 1000-1011.

Jackson A.A. (1994). *American Journal of Clinical Nutrition* **60**, 977-978.

Jackson A.A., Picou D., Landman J. (1984). *Human Nutrition Clinical Nutrition* **38C**, 339-354.

Quevedo M.R., Price G.M., Halliday D., Pacy P.J. & Millward D.J. (1994). *Clinical Science* **86**, 185-193.

**The blood pressure response of normotensive rats and rats with maternal-diet-induced hypertension to diets of differing fatty acid composition.** By S.C. LANGLEY-EVANS and A.A. JACKSON. *Department of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO16 7PX*

In the human population maternal diet in pregnancy is a primary determinant of risk of hypertension and cardiovascular disease (Barker *et al.* 1993). Postnatal environmental influences including diet, obesity and smoking have a further effect, over and above the *in utero* programming influences of maternal dietary factors. A rat model of maternal-diet-induced hypertension has been developed, in which exposure of the fetus to maternal low protein diets leads to a hypertensive state with early onset (Langley-Evans *et al.* 1994). The present study examines the differential responses of rats with differing prenatal dietary experiences to postnatal diets containing either coconut oil or maize oil.

Three female Wistar rats were fed on a 180 g casein/kg diet for 2 weeks prior to conception and throughout pregnancy. A further three females were fed on a 90 g casein/kg diet for the same period. On giving birth all rats were transferred to a standard laboratory chow diet, which was used to wean the offspring at the age of 3-4 weeks. At 6 weeks of age fourteen female rats from the 180 g casein/kg diet group and eight from the 90 g casein/kg dietary group had blood pressure determined (day 0 measurement). Half the rats in each group were then fed on a 90 g coconut oil/kg diet or a 90 g maize oil/kg diet for 6 weeks. Each diet contained a further 10 g corn oil/kg (Mulrooney & Grimble, 1993). Blood pressure was determined after 1, 2, 3 and 6 weeks.

Maternal diet g casein/kg	Postnatal diet	Systolic blood pressure (mm Hg)					
		Day 0			Week 6		
		Mean	n	SEM	Mean	n	SEM
180		127	14	5			
	Maize Oil				122	7	8
	Coconut Oil				157*	7	8
90		135 <sup>+</sup>	8	7			
	Maize Oil				140	4	11
	Coconut Oil				138	4	14

\* Mean value was significantly different from that for maize oil,  $P < 0.05$ . <sup>+</sup> Mean value was significantly different from that for 180 g casein/kg,  $P < 0.05$ . Three-way ANOVA indicated a significant interaction of maternal and postnatal diet,  $P < 0.01$ ,  $F = 7.26$ .

Rats exposed to 90 g casein/kg diet *in utero* had high blood pressure relative to those exposed to the 180 g casein/kg maternal diet, consistent with earlier reports (Langley-Evans *et al.* 1994). The normotensive (180 g casein/kg exposed) rats fed on maize oil maintained a stable blood pressure over the 6 weeks of the study. Those fed on coconut oil increased systolic blood pressure by approximately 30 mmHg. Hypertensive rats (exposed to 90 g casein/kg maternal diet) did not alter blood pressure when fed on either maize or coconut oil diets.

The results are consistent with the hypothesis that in addition to programming blood pressure in later life, exposure of the rat fetus to maternal low-protein diets influences the nature of the response to postnatal dietary changes.

Barker, D.J.P., Gluckman, P.D., Godfrey, K.M., Harding, J.E., Owens, J.A. & Robinson, J.S. (1993). *Lancet* **341**, 935-941.

Langley-Evans, S.C., Phillips, G.J. & Jackson, A.A. (1994). *Clinical Nutrition* **13**, 319-324.

Mulrooney, H.M. & Grimble, R.F. (1993). *Clinical Science* **84**, 105-112.

**Rats with hypertension induced by *in utero* exposure to maternal low-protein diets, fail to increase blood pressure in response to a chronically high salt intake.** By S.C. LANGLEY-EVANS and A.A. JACKSON. *Department of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO16 7PX*

Observations in the human population suggest a strong involvement of maternal diet in determining fetal growth and the programming of hypertension *in utero* (Barker *et al.* 1993). A rat model of maternal-diet-induced hypertension appears to mirror strongly the epidemiological evidence (Langley & Jackson, 1994). In humans, programming effects of maternal diet are overlaid by postnatal environmental influences, one of which is a high Na intake. Dietary NaCl has been strongly linked with hypertension, notably in sensitive individuals (Stamler, 1993) and rat studies clearly demonstrate hypertensive effects of Na (Friedman *et al.* 1990).

Three female Wistar rats were fed on a 180 g casein/kg diet for 2 weeks before conception and throughout pregnancy. A further three females were fed on a 90 g casein/kg diet for the same period. On giving birth all rats were transferred to a standard laboratory chow diet, which was used to wean the offspring at the age of 3-4 weeks. At 6 weeks of age six male rats from each maternal dietary group had blood pressure determined using an indirect tail cuff method (day 0 measurement). The rats were then provided with 15 g/l NaCl in place of drinking water for 7 days. Blood pressure was determined on days 1, 3, 5 and 7 and compared with six water-drinking controls, prenatally exposed to 180 or 90 g casein/kg diet. In a separate experiment the same rats were housed in metabolic cages, with access to either water or NaCl solution as the only drink, and fluid intake, urinary output and urinary Na excretion determined.

Maternal diet (g casein/kg)	Treatment	Systolic blood pressure (mm Hg)					
		Day 0			Day 5		
		Mean	n	SEM	Mean	n	SEM
180	Water	139 <sup>a</sup>	6	10	136 <sup>a</sup>	6	4
	NaCl	137 <sup>a</sup>	6	5	176 <sup>b*</sup>	6	10
90	Water	154 <sup>b</sup>	6	7	145 <sup>ca</sup>	6	1
	NaCl	152 <sup>b</sup>	6	1	139 <sup>aa</sup>	6	2

<sup>a,b,c</sup> Mean values within a column with unlike superscript letters were significantly different,  $P < 0.05$ .

\* Mean value was significantly different from that of water control  $P < 0.05$ .

Normotensive rats exposed to the 180 g casein/kg diet *in utero* significantly increased fluid intake, urinary output, Na excretion and systolic blood pressure in response to saline drinking. Hypertensive rats exposed to the 90 g casein/kg diet *in utero* also increased fluid intake, urinary output and Na excretion, but failed to increase systolic blood pressure while drinking 15 g/l NaCl.

Rats with maternal-diet-induced hypertension appear to be insensitive to the hypertensive effects of Na. This insensitivity does not appear to stem from a more rapid clearance of excess Na.

Barker, D.J.P., Gluckman, P.D., Godfrey, K.M., Harding, J.E., Owens, J.A. & Robinson, J.S. (1993). *Lancet* **341**, 935-941.

Friedman, S.M., McIndoe, R.A. & Tanaka, M. (1990) *Journal of Hypertension* **8**, 61-66.

Langley, S.C. & Jackson, A.A. (1994) *Clinical Science* **86**, 217-222.

Stamler, J. (1993) *Annals of the New York Academy of Science* **676**, 122-156.

**Pharmacological adrenalectomy during pregnancy abolishes maternal-diet-induced hypertension in the rat.** By S.C. LANGLEY-EVANS, G.J. PHILLIPS and A.A. JACKSON. *Department of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO16 7PX*

Glucocorticoid hormones have been implicated in the programming of hypertension *in utero* (Benediktsson *et al.* 1993). In the maternal-diet-induced hypertensive rat model, a reduced activity of the placental enzyme 11 $\beta$ -hydroxysteroid dehydrogenase in rats consuming low-protein diets, has been proposed to over-expose the fetus to corticosterone (Phillips *et al.*, 1994). In the present study pharmacological adrenalectomy of pregnant rats, using the adrenal 11 $\beta$ -hydroxylase inhibitor metyrapone (Baram and Schultz, 1990), is used to evaluate the role of corticosteroids in the initiation of hypertension *in utero*.

Thirteen female Wistar rats were fed on a 180 g casein/kg diet for 2 weeks before conception and throughout pregnancy. A further fifteen females were fed on a 90 g casein/kg diet for the same period. Twice daily for 14 d from conception, six rats in each group were injected subcutaneously with 10 mg metyrapone/kg body weight in arachis oil. The remaining rats were injected twice daily with arachis oil. On day 14 of pregnancy eight rats in each dietary group, four treated with metyrapone and four vehicle-injected controls, were killed for assessment of maternal and fetal plasma corticosterone concentrations. The remaining rats proceeded to full term and on giving birth all rats were transferred to a standard laboratory chow diet, which was used to wean the offspring at the age of 3-4 weeks. At 7 weeks of age a random selection of the male and female pups had blood pressure determined, using an indirect tail cuff method.

Maternal diet g casein/kg	Treatment	Body weight (g)			Systolic blood pressure (mm Hg)		
		Mean	<i>n</i>	SEM	Mean	<i>n</i>	SEM
180	Vehicle	156	12	6	129	12	5
	Metyrapone	169	8	18	144*	8	5
90	Vehicle	141 <sup>+</sup>	19	4	158 <sup>+</sup>	19	6
	Metyrapone	133 <sup>+</sup>	8	5	139*	8	3

\* Mean values were significantly different from vehicle-treated controls,  $P < 0.05$ . <sup>+</sup> Mean values were significantly different from 180 g casein/kg diet,  $P < 0.05$ .

Seven-week-old rats exposed to 90 g casein/kg maternal diets *in utero* were of significantly lower body weight than rats exposed to the 180 g casein/kg diet. Metyrapone treatment had no significant effect on body weight. Vehicle-injected rats exposed to the low-protein diet *in utero* had significantly elevated blood pressure relative to the vehicle-injected 180 g casein/kg diet-exposed control group. The hypertension of the low-protein-diet-exposed group was abolished by metyrapone treatment, but in the normotensive group metyrapone treatment significantly elevated blood pressure.

The results would suggest that within the first 2 weeks of pregnancy in the rat, glucocorticoids are important determinants of future blood pressure in the offspring. The effects of glucocorticoids are modulated by the maternal diet and may be either hypertensive or hypotensive.

Baram, T.Z. & Schultz L. (1990). *Life Sciences* 47, 485-489.

Benediktsson, R., Lindsay, R.S., Noble, J., Seckl, J.R. & Edwards C.R.W. (1993). *Lancet* 341, 339-341.

Phillips, G.J., Langley-Evans, S.C., Benediktsson, R., Seckl, J.R., Edwards, C.R.W. & Jackson, A.A. (1994). *Proceedings of the Nutrition Society* 53, 170A.

**Factors influencing creatine retention in man.** By A.L. GREEN, I.A. MACDONALD and P.L. GREENHAFF, *Department of Physiology and Pharmacology, The Medical School, Nottingham University NG7 2UH*

Insulin stimulates muscle creatine (CR) uptake *in vitro* (Haughland & Chang, 1975). Dietary CR supplementation, at a rate of 20 g/d for 5 d, increases muscle total CR concentration by about 20 % in man, and uptake is further increased if exercise is performed during supplementation (Harris *et al.* 1992). The aim of the present study was to investigate the effects of carbohydrate (CHO) ingestion (in an attempt to raise plasma insulin concentration), together with exercise, on CR disposal following CR ingestion in man.

Twenty-two men were randomly divided into four groups (A-D). On d 1, fasted subjects gave a blood sample and then consumed: A, 5 g CR in 250 ml low-calorie hot orange, *n* 6; B and C, 5 g CR in 250 ml hot orange + 500 ml Lucozade, *n* 6; D, 250 ml low-calorie hot orange, *n* 4. Arterialized-venous blood samples were then obtained at 20 min intervals for the next 4.5 h, while subjects remained in a supine position. For the remainder of the d, and throughout d 2, subjects ingested the above preparations at 4 h intervals (total daily CR dose 20 g) and on the morning of d 3, reported back to the laboratory and underwent the same procedures as on d1. In addition, subjects in group C performed 1 h of cycling exercise at 70 %  $\dot{V}O_{2\max}$  each day immediately before fluid ingestion. All subjects weighed and recorded their dietary intake throughout the study (subjects in groups B + C consumed a prescribed high-CHO diet) and undertook 24 h urine collections on d 1 and d 3. Plasma and urine CR were measured using HPLC and serum insulin was measured using a radioimmunoassay technique.

	Day 1						Day 3					
	A		B		C		A		B		C	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
AUC (mmol/l/min)	2.83	0.30	0.88 <sup>††</sup>	0.11	0.96 <sup>†</sup>	0.46	2.64	0.23	0.95 <sup>*</sup>	0.45	1.06 <sup>†</sup>	0.21
Urinary CR (g)	9.5	1.2	5.0 <sup>*</sup>	0.8	4.8 <sup>*</sup>	0.9	11.9	1.1	5.7 <sup>†</sup>	1.2	8.5 <sup>*</sup>	1.1
Peak insulin (mIU/l)	7.8	1.3	72.0 <sup>††</sup>	11.2	70.0 <sup>††</sup>	12.2	9.5	2.0	84.2 <sup>††</sup>	11.5	58.3 <sup>††</sup>	5.9

Significantly different from group A (ANOVA, post-hoc unpaired *t* test): \* *P*<0.05; † *P*<0.01; †† *P*<0.001.

Plasma CR concentration peaked within 90 min following CR ingestion and declined towards resting values during the remaining 180 min. The area under the plasma CR-time curve (AUC) was lower in group B and C when compared with A. Urinary CR content (g) was also lower in groups B compared with group A. No difference was seen between groups B and C when comparing AUC or urinary CR content. Following carbohydrate ingestion, serum insulin levels peaked within 30 min in groups B and C and returned to the pre-ingestion concentration over the remaining 240 min. Plasma insulin concentration did not change in groups A and D over the course of the experiment.

Thus, CR retention is likely to be increased when CHO is consumed in conjunction with CR; this may be due to an insulin mediated increase in muscle CR uptake. The extent of CR retention does not appear to be further increased when exercise is performed before CR and CHO supplementation. This latter finding could be interpreted to mean that the previously reported stimulatory effect of exercise on muscle CR uptake (Harris *et al.* 1992), may have been achieved by exercise increasing insulin sensitivity.

Ethical approval was granted for this study which was supported by the Ministry of Defence.

Harris, R.C., Söderlund, K. & Hultman, E. (1992). *Clinical Science* **83**, 367-374.

Haughland, R.B. & Chang, D.T. (1975). *Proceedings Society of Experimental Biology and Medicine* **148**, 1-4.

**The influence of treadmill walking on the lipaemic and metabolic responses to a high-fat meal in women aged 35 to 50 years.** By N.V. TSETSONIS, D.M. ROCHE and A.E. HARDMAN, *Department of Physical Education, Sports Science and Recreation Management, Loughborough University, Loughborough Leics LE11 3TU*

One prolonged bout of walking diminishes the lipaemic response to a fatty meal in young adults (Aldred *et al.* 1994). However, as postprandial lipaemia increases with age (Krasinski *et al.* 1990) and may be influenced by body composition, this effect may not be evident in other subject groups. The purpose of the present study was to examine the effect of one bout of moderate walking on the lipaemic and metabolic responses to a high-fat meal in women aged 35 to 50 years.

Twelve healthy women, aged 44 (SEM 1.2) years, body fat 29.1 (SEM 1.0) %, waist:hip ratio 0.7 (SEM 0.02) and maximal O<sub>2</sub> uptake ( $\dot{V}O_{2\max}$ ) 33.4 (SEM 1.5) ml/kg per min took part in two trials (control, exercise) in a balanced design. Each trial was conducted over 2 d. On the afternoon of day 1, 90 min treadmill walking was performed at 60.6 (SEM 1.6) %  $\dot{V}O_{2\max}$ . On the control trial, subjects did not perform any exercise on day 1. On day 2 subjects came to the laboratory after an overnight fast for an oral fat tolerance test (OFTT), and baseline expired air and venous blood samples were obtained in the fasted state. They then ingested a test meal (1.7 g dietary fat/kg subject's fat-free mass, 67% energy from fat). Further expired air and blood samples were obtained at intervals during the next 6 h. Dietary and exercise habits were controlled for 3 d before each OFTT. Serum was analysed for triacylglycerol (TAG), total cholesterol, high-density-lipoprotein cholesterol and its subfractions, insulin and free fatty acid concentrations. The lipaemic response (area under TAG v. time curve, normalized to the zero hour level), peak TAG concentration and maximal TAG increase (mean of two highest values minus the fasting value) were adopted as indices of lipaemia. Comparisons between trials were made using two-way ANOVA, adopting a 5% level of significance.

Fasting TAG concentration and all indices of lipaemia were lower for the exercise trial (Table), but no differences were found between trials in the cholesterol or insulin responses. In the fasted state, respiratory exchange ratio (R) was lower for the exercise trial (0.73 (SEM 0.02) v. 0.80 (SEM 0.02)). Average R over the 6 h observation period was also lower for the exercise trial (0.80 (SEM 0.02) v. 0.84 (SEM 0.02)). The change in O<sub>2</sub> uptake over time differed between trials, values for 1 and 2 h after the meal being higher for the exercise trial.

Trial	Fasting [TAG] (mmol/l)		Lipaemic response (mmol/l·h)		Peak [TAG] (mmol/l)		Max. [TAG] increase (mmol/l)	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Exercise	0.61*	0.09	3.09*	0.53	1.50*	0.19	0.80*	0.14
Control	0.73	0.10	3.74	0.60	1.77	0.21	0.96	0.15

\* Significantly different from control,  $p < 0.05$ .

These results suggest that, in healthy, normolipidaemic middle-aged women, walking at 60%  $\dot{V}O_{2\max}$  attenuated the lipaemic response to a high-fat meal consumed 15 h later. Walking also increased fat oxidation and caused a difference in the O<sub>2</sub> uptake response to the meal.

Aldred, H.E., Perry, I.C. & Hardman, A.E. (1994). *Metabolism* **43**, 836-841.

Krasinski, S.E., Cohn, J.S., Schaefer, E.J. & Russell, R.M. (1990). *Journal of Clinical Investigation* **85**, 883-892.



**The effect of 13 weeks' endurance running training followed by 9 d detraining on fasting and postprandial triacylglycerol concentrations.** By S.L. HERD<sup>1</sup>, C.J. CAIRNS<sup>1</sup>, K.S. MOORE<sup>1</sup>, A.E. HARDMAN<sup>1</sup> and L.H. BOOBIS<sup>2</sup>, <sup>1</sup>*Department of Physical Education, Sports Science and Recreation Management, Loughborough University, Leicestershire LE11 3TU, and* <sup>2</sup>*Department of Surgery, Sunderland District General Hospital, Tyne and Wear SR4 7TP*

Research suggests that endurance-trained individuals exhibit a lower lipaemic response than their sedentary peers (Cohen *et al.* 1989). The purpose of the present study was to ascertain whether the difference in response is attributable to the chronic effects of endurance training or to the residual effect of the last training session.

Fourteen physically active, normolipidaemic adults volunteered to participate in the study. Eight (five women, three men) completed a progressive running training programme of 13 weeks, followed by 9 d detraining. Six (three women, three men), serving as control subjects, maintained their habitual lifestyle. For runners, mean age was 21.9 (SEM 1.6) years and body mass index 24.6 (SEM 0.8) kg/m<sup>2</sup>. Corresponding values for controls were 24.2 (SEM 2.1) years and 23.3 (SEM 0.7) kg/m<sup>2</sup> respectively.

Changes in endurance fitness were assessed using treadmill tests and changes in body fatness by anthropometry. Each subject undertook four oral fat tolerance tests (OFTT) i.e. before the start of the exercise programme (pre-training) and 15 h, 60 h and 9 d after the final running session (post-training). The weighed food intake technique was employed to describe habitual dietary practice the initial OFTT and to control food intake before subsequent tests. All subjects refrained from exercise for 2 d before each test. Runners abstained from all exercise after the last training session until the end of the study. On all four occasions, after an overnight fast, a cannula was introduced into a forearm vein and the first blood sample was drawn. The test meal, comprising cereals, fruit, nuts, chocolate and whipping cream (1.2 g fat/kg body mass), was consumed and further blood samples were drawn hourly for 6 h. Serum was analysed for lipid and lipoprotein variables. The total lipaemic response was determined as the area under the triacylglycerol (TAG) v. time curve. The incremental lipaemic response was the total lipaemic response normalized to the 0 h level. A Mann-Whitney *U* test was employed to compare changes over time between runners and controls, adopting a 5% level of significance.

Following the running programme, changes in maximal O<sub>2</sub> uptake ( $\dot{V}O_{2\max}$ ) and body fatness differed between runners and controls ( $\dot{V}O_{2\max}$ , runners +3.2 (SEM 1.1) ml/kg per min v. controls -1.3 (SEM 1.2) ml/kg per min; body fatness, runners -1.9 (SEM 0.5)% v. controls -0.5 (SEM 0.2)%). For runners, indices of lipaemia, indicated in the Table below, tended to increase during the detraining period but no significant differences were observed.

	Fasting [TAG] (mmol/l)				Total lipaemic response (mmol/l.h)				Incremental lipaemic response (mmol/l.h)			
	Runners		Controls		Runners		Controls		Runners		Controls	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Pre-training	1.15	0.19	0.70	0.08	10.32	1.54	6.14	0.71	3.74	0.83	1.97	0.44
15 h post	0.72	0.09	0.71	0.08	7.23	1.12	6.37	0.61	2.88	0.69	2.12	0.42
60 h post	0.92	0.09	0.62	0.12	9.92	1.20	5.68	0.81	4.39	0.81	1.96	0.27
9 d post	1.03	0.15	0.79	0.10	10.59	1.13	7.29	0.85	4.39	0.95	2.57	0.65

The proposition that the low lipaemic response which characterizes endurance-trained young adults may reflect the residual effects of the last training session justifies further examination.

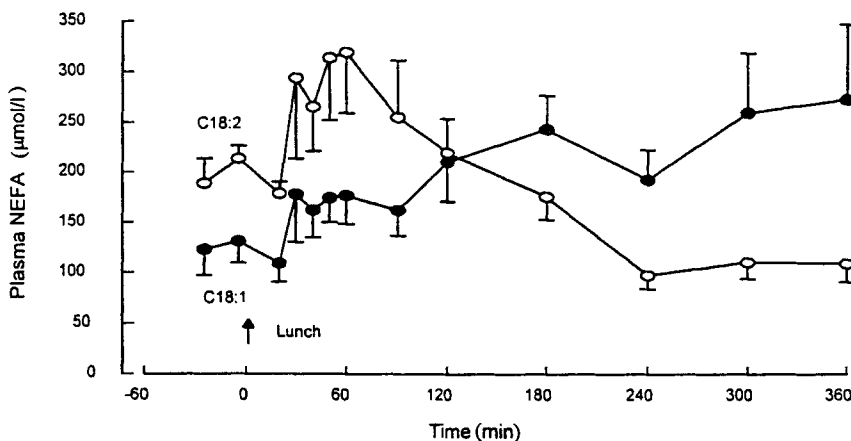
This research was supported by the British Heart Foundation.

Cohen, J.C., Noakes, T.D. & Spinnler Benade, A.J. (1989). *American Journal of Clinical Nutrition* 49, 443-447.

**The origin of plasma non-esterified fatty acids in the postprandial period.** By B.A. FIELDING, J. CALLOW, R. OWEN, J.S. SAMRA and K.N. FRAYN, *Oxford Lipid Metabolism Group, Radcliffe Infirmary, Oxford OX2 6HE*

The net flow of fatty acids within adipose tissue is precisely controlled by the regulation of lipoprotein lipase (LPL) and hormone sensitive lipase (HSL) to produce net fatty acid release in the fasting state and net fatty acid retention in the fed state (Frayn *et al.* 1994). In response to the ingestion of dietary fat, LPL releases fatty acids from plasma triacylglycerol (TAG) so that they may be taken up by adipose tissue for re-esterification. LPL appears to operate continuously, generating a pool of fatty acids of which some are always released into the venous plasma. In the postprandial state, a proportion are diverted into the tissue for esterification and storage.

In order to study this, we have used two sequential test meals with specific fat composition to enable natural dietary fatty acids to be traced into the plasma non-esterified fatty acid (NEFA) profile. Seven normal subjects (two male) ate a fat-free evening meal the day before the study. After an overnight fast of at least 12 h, the subjects consumed a high-fat breakfast consisting of 54 g fat (safflower oil), 12 g protein and 140 g carbohydrate as a cooked oat 'flapjack'. Five hours later at 12.00 hours, the subjects ate a second test meal (lunch) containing 61 g fat (olive oil and fresh avocado), 13 g protein and 35 g carbohydrate. The predominant fatty acids in the first test meal, expressed as g 100 g total fatty acids, were C18:2, 68 and C18:1, 19 and those in the second meal were C18:2, 8 and C18:1, 74.6.



The concentration of C18:2 in plasma NEFA showed a large peak at about 50 - 60 min after the second test meal (see Figure). This peak exactly mirrors a peak in the concentration of C18:2 in plasma chylomicron TAG which has been 'carried over' from the previous meal (Fielding *et al.* 1994) and this may illustrate the 'spillage' of NEFA into the plasma as a result of the hydrolysis of dietary TAG by adipose tissue LPL.

Frayn, K.N., Shadid, S., Hamrani, R., Humphreys, S.M., Clark, M.L., Fielding, B.A., Boland, O. & Coppack, S.W. (1994). *American Journal of Physiology* 266, E308-E317.

Fielding, B.A., Owen, R., Callow, J. & Frayn, K.N. (1994). *Proceedings of the Nutrition Society* (In the Press).

**The role of cortisol in regulation of lipolysis in normal subjects after an overnight fast.** By J. S. SAMRA<sup>1</sup>, M. L. CLARK<sup>1</sup>, S. M. HUMPHREYS<sup>1</sup>, K.N. FRAYN<sup>1</sup> and I. A. MACDONALD<sup>2</sup>, <sup>1</sup>*Oxford Lipid Metabolism Group, Radcliffe Infirmary, Oxford OX2 6HE and* <sup>2</sup>*Department of Physiology and Pharmacology, Medical School, Queen's Medical Centre, Nottingham NG7 2UH*

Lipolysis is the process by which stored body fat is broken down to provide metabolic fuel in the form of non-esterified fatty acids (NEFA) and glycerol. This process is modulated by a number of hormones. The possible role of cortisol and growth hormone in stimulating lipolysis has come into prominence because microdialysis work has shown that at rest alpha adrenergic effect is inhibitory on lipolysis (Amer *et al.* 1990).

We have investigated the role of cortisol in regulating lipolysis by measurement of arterial venous difference across a subcutaneous adipose tissue depot. We obtained arterialized blood from a vein draining a hand warmed in a box at 65 ° and venous blood from superficial inferior epigastric vein draining the adipose tissue of anterior abdominal wall. Six healthy subjects (median age 34 (range 24-49) years ; median BMI 24.55 (range 20.33-25.34) kg/m<sup>2</sup>) were studied on two occasions after an overnight fast. During the first morning either metyrapone was given to block cortisol production or a control study was carried out. The veno-arterial (v-a) differences were measured for triacylglycerol (TAG), NEFA, glycerol and glucose across the adipose tissue bed and adipose tissue blood flow was measured by the clearance of <sup>133</sup>Xe (Larsen *et al.* 1966). The net effect of adipose tissue lipoprotein lipase (LPL) and hormone-sensitive lipase (HSL) activity can be measured by studying arteriovenous TAG, NEFA and glycerol differences (Frayn *et al.* 1994).

	Control		Metyrapone	
	Mean	SE	Mean	SE
v-a difference				
NEFA (µmol/l)	500	59	324 **	81
TAG (µmol/l)	- 41.5	15.5	- 27.6	7.9
Glycerol (µmol/l)	153.6	22.5	86.6 *	20.9
Glucose (mmol/l)	- 0.025	0.038	- 0.045	0.055
Blood flow (ml/100g .min)	4.86	0.69	5.37	1.14
Cortisol (nmol/l)	343	24	191 **	16

Mean values were significantly different from controls \*  $P < 0.05$ , \*\*  $P < 0.01$ .

Normal plasma cortisol concentrations averaged from 03.00 to 14.00 hours were suppressed with 4-hourly doses of metyrapone. After 18 h starvation the v-a blood differences for NEFA and glycerol (indicative of HSL activity) were significantly different in the control study than after the cortisol blockade, but the blood TAG differences (indicative of LPL activity) were not significant. There were no significant differences in adipose tissue blood flow. These results suggest that cortisol is one of the many hormones that regulates lipolysis in normal subjects after an overnight fast through its action on HSL.

Amer, P., Kreigholm, E., Engfeldt, P. & Bolinder, J. (1990) *Journal of Clinical Investigation* **85**, 893-898.

Larsen, O.A., Lassen, N.A. & Quaade, F. (1966). *Acta Physiologica Scandinavica* **66**, 337-345.

Frayn, K.N., Shadid, S., Hamrani, R., Humphreys, S.M., Clark, M.L., Fielding, B.A., Boland, O. & Coppack, S.W. (1994). *American Journal of Physiology* **266**, E308-E317.

**The 24 h energy expenditure and urinary catecholamines of healthy young men given medium-chain triacylglycerols incorporated into a typical "Western" diet: a dose-response study in a human respiratory chamber.** By A.G. DULLOO, M. FATHI, N. MENSI, and L. GIRARDIER, *Department of Physiology, Faculty of Medicine, Centre Médical Universitaire, and Hôpital Cantonal Universitaire de Genève, 1 rue Michel-Servet, 1211 Geneva 4, Switzerland*

The importance of dietary fat types in human energy balance and in the control of body composition is poorly understood, despite numerous indications that the role of dietary fat in altering thermogenesis and as a factor in susceptibility to fat and lean tissue accretion depends upon its fatty acid composition (Dulloo, 1993; Dulloo *et al.* 1995). In this context, medium-chain triacylglycerols (MCT) have consistently been shown to be utilized less efficiently than long-chain triacylglycerols (LCT) in laboratory animals, but in humans a greater stimulatory effect of MCT than LCT has been demonstrated over only a few hours after single meal ingestion of large amounts of MCT; its influence on daily energy expenditure (EE) when consumed under a more "normal" pattern of food intake is not known.

The studies reported here were undertaken to determine whether mild-to-moderately high amounts of MCT ingested together with typical "Western" foods eaten at breakfast, lunch and dinner could stimulate thermogenesis to the extent of increasing EE over a 24 h cycle. At the same time, the role of catecholamines in mediating the thermogenic effects of MCT was assessed from urinary excretion rates of adrenaline, noradrenaline, and dopamine. Using a respiratory chamber, 24 h EE and 24 h urinary catecholamines were measured in eight healthy and normal-weight young men on four separate occasions which were randomized between four different combinations of MCT and LCT in a double-blind design. These were incorporated into fruit yogurts which provided an additional 10 g fat at breakfast, lunch and dinner, and consumed with their weight maintenance diet (15% protein, 40% fat, and 45% carbohydrates by energy) in the following ratio: MCT:LCT (g/g) 0:30, 5:25, 15:15 and 30:0. Within-subject measurements were conducted under identical conditions of energy intake and activity pattern.

The results indicate a significant increase in 24 h EE with increasing MCT:LCT ratio (ANOVA,  $P < 0.001$ ), with the diet providing 30 g MCT stimulating daily EE by 5%: this corresponds to a mean/median absolute increase in daily EE of 500 kJ, with individual values varying between 268 kJ and 756 kJ. No statistically significant differences were observed in respiratory quotient nor in urinary N losses across diets, but 24 h urinary noradrenaline was significantly increased (ANOVA,  $P < 0.025$ ), whereas adrenaline and dopamine were unaltered. The net energy value of MCT, in moderate-to-high amounts, is calculated to be 20.9 kJ/g (5 kcal/g), and hence much less than values of 29-33 kJ/g (7-8 kcal/g) previously assumed for MCT, and the value of 37.7 kJ/d (9 kcal/g) for other fats.

Taken together, these results suggest that MCT as part of typical "Western" diets may play a role in the control of human body composition by enhancing daily EE, and that this thermogenic effect is mediated in part through activation of the sympathetic nervous system.

This study was supported in part by the Swiss National Research Foundation and in part by Arkopharma Laboratories.

Dulloo, A.G. (1993). *Nutrition* 9, 366-372.

Dulloo, A.G., Mensi, N., Seydoux, J. & Girardier, L. (1995). *Metabolism* 44, 273-279.

**Long-chain but not medium chain triacylglycerols increase factor VII coagulant activity.** By NAJAT YAHIA<sup>1</sup>, G.J. MILLER<sup>2</sup> and T.A.B. SANDERS<sup>1</sup>. <sup>1</sup>*Department of Nutrition and Dietetics, King's College, University of London, Campden Hill Road, London W8 7AH and* <sup>2</sup>*MRC Epidemiology and Medical Care Unit, Wolfson Institute of Preventive Medicine, Medical College of St Bartholomew's Hospital, Charterhouse Square, London EC1M 6BQ*

Elevated levels of factor VII coagulant (VIIc) activity are associated with increased risk of fatal ischaemic heart disease. We have previously shown that VIIc activity is increased 7 h after a meal containing 90 g fat in healthy subjects. No such effect was noted with an isoenergetic low fat, high carbohydrate test meal. This elevation in VIIc activity was related to the degree of postprandial lipaemia. However, it is also possible that a high carbohydrate test meal suppresses the activation of VIIc. Medium chain triacylglycerols (MCT) are absorbed and transported to the liver via the portal vein and do not lead to postprandial lipaemia (Swift *et al.*, 1992). We postulated that the consumption of MCT would not increase factor VIIc activity. In order to test this hypothesis, four volunteers consumed test meals containing either 90 g olive oil or 90 g MCT on separate days 1 week apart. Venous samples were obtained in the fasting state and 3 h and 7 h after the test meal and plasma triacylglycerol concentrations and VIIc activity were determined. The results were as follows:

	90 g olive oil		90 g MCT	
	Mean	SE	Mean	SE
Change from fasting triacylglycerol at 3 h (mmol/l)	1.58*	0.630	-0.14	0.07
Change from fasting VIIc at 7 h (% standard)	11.25*	3.28	-6.0	7.38

\* $P < 0.05$  compared with fasting sample.

All the subjects experienced moderate abdominal discomfort 1-3 h after consuming the MCT test meal but not after the olive-oil test meal. This may have been because MCT is a potent stimulator of cholecystokinin release (Remedios *et al.*, 1992). We would caution other workers about using such a high dose of MCT in future. Plasma triacylglycerol concentrations and factor VIIc activity levels were significantly elevated by olive oil but not by MCT. These results suggest that only long-chain fatty acids which lead to chylomicron formation are able to activate factor VIIc in the post-prandial state.

T.A.B.S. acknowledges a grant from Ministry of Agriculture Fisheries and Food.

De Grassi, T., Miller, G.J. & Sanders, T.A.B. (1994). *Proceedings of Nutrition Society* **53**, 79A.

Remedios, T.M., Mitsuhiro, F., Sung-in, Y. & Jun-ichi, O. (1992). *Journal of Nutrition* **122**, 1702-1705.

Swift, L.J., Hill, J.O., Peter, J.C. & Greene, H.L., (1992). *American Journal of Clinical Nutrition* **5**, 881-886.

**Effects of elaidic acid (18:1 *n*-9 *trans*) and oleic acid (18:1*n*-9 *cis*) on plasma and hepatic cholesterol concentrations in the hamster.** By MARY B.C. ASCOTT, LUCIE POLLARD and T.A.B. SANDERS, *Department of Nutrition and Dietetics, King's College, University of London, Campden Hill Road, London W8 7AH*

*Trans* monounsaturated fatty acids are the predominant isomeric fatty acids in human diets and their consumption has been associated with an increased risk of cardiovascular disease (Willett *et al.* 1993). Elaidic acid (18:1*n*-9 *trans*) compared with oleic acid (18:1*n*-9 *cis*) was found to decrease the HDL:LDL ratio in human subjects (Abbey & Nestel, 1994). We have previously found that hamsters fed on ethyl oleate accumulate cholesterol in their livers and secrete VLDL particles with a higher cholesterol:protein ratio than those fed ethyl palmitate or myristate (Sanders *et al.* 1993). The present study compares a diet rich in ethyl oleate with a diet in which a large proportion of the ethyl oleate was replaced by ethyl elaidate. Eighteen adult male hamsters were randomly allocated to two groups of nine to receive semi-purified casein-based diets containing (g/kg): cholesterol 0.6; sunflower oil 20; test fat 100 (95% ethyl oleate or 72% ethyl elaidate and 23% ethyl palmitate). Food intakes were restricted to 20 g/day and were similar in both groups throughout the study. On day 20 they received only 5 g, and on day 21 the animals were anaesthetized and exsanguinated and tissues obtained for analysis. Plasma lipoproteins were separated by ultracentrifugation and hepatic and lipoprotein cholesterol concentrations were determined using enzymatic assays. The results were as follows:

	Oleate n 9		Elaidate n 9	
	Mean	SE	Mean	SE
Plasma cholesterol (mmol/l)	4.96	0.39	4.85	0.29
HDL:LDL cholesterol	4.20	0.27	3.30*	0.33
Hepatic cholesterol (mmol/l)	33.40	3.25	19.70**	1.70

\* $P < 0.05$ ; \*\* $P < 0.01$  compared with oleate group

Elaidic acid partially displaced oleic acid in adipose tissue, hepatic cholesteryl esters, triacylglycerols, and VLDL cholesteryl esters as expected. Plasma cholesterol concentrations were similar in both groups but the HDL:LDL ratio was significantly lower for the elaidate group. This observation is consistent with enhanced cholesterol transfer protein activity which has been reported in man (Abbey & Nestel, 1994). The VLDL cholesterol:protein ratio was similar in both groups. Hepatic total and esterified cholesterol concentrations were higher in the oleate-fed group compared with the elaidate fed group. We conclude that elaidic acid behaves differently from both saturated fatty acids and oleic acid in its effects on cholesterol metabolism in the hamster. Further studies are required to explain why oleic acid increases cholesterol accumulation in the liver.

Abbey, M., Nestel, P.J. (1994). *Atherosclerosis* **106**, 99-107.

Sanders, T.A.B., Sandaradura, S. & Slattery, F. (1993). *Proceedings of the Nutrition Society* **52**, 364A.

Willett, W.C., Stampfer, M.J., Manson, J.E., Colditz, G.A., Speizer, F.E., Rosner, B.A., Sampson, L.A. &

Henneken, C.A. (1993). *Lancet* **341**, 581-585.

**Beneficial effects of plant volatile oils on the retinal lipids during ageing.** By ZSUZSA RECSAN<sup>1</sup>, GIAMPIERO PAGLIUCA<sup>2</sup>, LASZLO G. PENZES<sup>1</sup>, STANLEY G DEANS<sup>1</sup>, RAYMOND C. NOBLE<sup>1</sup> and MARCO V. PIRETTI<sup>2</sup>, <sup>1</sup>*Department of Biochemical Sciences, SAC Auchincruive, Ayr KA6 5HW*, <sup>2</sup>*Department of Veterinary Biochemistry, University of Bologna, Bologna, Italy*

Age-related macular degeneration (AMD) is one of the leading causes of severe visual impairment. Numerous risk factors are known to be involved, major amongst them being nutritional. Higher levels of circulating micronutrients with antioxidative capabilities are suggested to reduce the risk of AMD. Since antiquity medicinal and culinary plants have been credited with a range of beneficial properties for the well-being of man (Deans & Waterman, 1993). Most recently, investigations into aspects of lipid metabolism in the liver of ageing mice have shown that dietary supplementation with specific plant volatile oils can result in pronounced beneficial effects on tissue levels of polyunsaturated fatty acids (PUFA; Deans *et al.* 1993). Rats were fed daily with 3.5 mg of a selection of plant volatile oils over an 18 month period. At 28 months of age, the retinas were excised and the major lipid and fatty acid composition (phospholipid, triglyceride, cholesterol ester) was determined. The Table shows the percentage composition of the unsaturated fatty acids in the retinas from rats fed with plant volatile oils. Each result is the mean of six measurements.

Fatty acid	Control	SED	Clove	SED	Nutmeg	SED	Pepper	SED	Thyme	SED
Arachidonic	12.4	0.89	16.0**	0.16	16.0*	0.98	18.5***	0.61	17.5***	0.13
Docosahexænoic	39.2	2.75	52.6***	0.24	51.6	5.43	47.7*	0.18	45.8*	0.26
Polyunsaturate :	1.47		2.52		2.43		2.30		2.01	
Monounsaturate ratio										

Mean values were significantly different from control ; \*P<0.05, \*\*P<0.01, \*\*\*P<0.001.

There was no difference in the percentage distribution of the retinal lipid fractions listed above between the rats. Phospholipid accounted for 24.2 (SE±4.1) % total lipid. Treatment with the volatile oils significantly increased the proportions of the total and individual PUFA and the polyunsaturated:monounsaturated ratios. In the case of treatment with clove, pepper and thyme volatile oils, there were significant increases in the levels of both arachidonic and docosahexænoic (DHA) acids, while in the case of nutmeg oil, the increase was confined to arachidonic acid. The increased levels of PUFA were accompanied by significant reductions in the levels of linoleic acid.

It is well recognized that a reduction in retinal levels of DHA is a feature of ageing (Rotstein *et al.* 1987). An increased destruction of polyunsaturates would most notably involve the formation of free radicals and peroxidation products. The present observations sustain the belief of the involvement of an antioxidant rôle in retinal function during ageing. The volatile oils of clove, nutmeg, pepper and thyme all afforded considerable protection in the maintenance of PUFA levels, in particular the highly labile DHA. The results are in entire agreement with data obtained from investigations into the lipid composition of ageing mice administered oil of thyme, where an identifiable high concentration of antioxidant capacity resulted in the maintenance of enhanced levels of DHA.

Deans, S.G., Noble, R.C., Péznes, L.G. & Imre, S.G. (1993). *Age* 17, 71-74.

Deans, S.G. & Waterman, P.G. (1993). *Volatile Oil Crops*. London : Longman

Rotstein, N.P., Boscherio, I., Giusto, N.M. & Avelano, M.I. (1987). *Lipids* 22, 253-260.

**Impact of plant volatile oils upon enzymes involved in lipid peroxidation.** By STANLEY G DEANS<sup>1</sup>, ALLAN MACPHERSON<sup>1</sup>, RAYMOND C NOBLE<sup>1</sup>, LASZLO G PENZES<sup>2</sup> and SANDOR G IMRE<sup>3</sup>, <sup>1</sup> Department of Biochemical Sciences, SAC Auchincruive, Ayr KA6 5HW, <sup>2</sup> Institute of Gerontology, Semmelweis Medical University, Budapest, Hungary, <sup>3</sup> Debrecen University of Medicine, Debrecen, Hungary

Ageing is associated with a substantial decrease in the levels of polyunsaturated fatty acids (PUFA) in tissues with consequential adverse effects on a range of biological functions (Wade & Tsumita, 1984). As part of an array of controlling factors, peroxidation is a major factor leading to a reduction in PUFA levels (Pézenes *et al.* 1988). Antioxidant activities are prominent among the claimed benefits of culinary and medicinal plants (Deans *et al.* 1993a).

Groups of young (4 month) and old (16 month) mice were fed on a standard laboratory diet supplemented *via* their drinking water by gavage, with 360 µg of volatile oils for periods of 6 and 19 weeks respectively. Age-matched controls received only drinking water. The volatile oils, selected by their *in vitro* antioxidant performance (Deans *et al.* 1993b), were bitter almond (*Amygdalus communis*), clove (*Eugenia caryophyllatus*), nutmeg (*Myristica fragrans*), black pepper (*Piper nigrum*) and thyme (*Thymus vulgaris*), obtained by steam distillation.

Glutathione peroxidase (GSHPx : EC 1.11.1.9) concentrations were measured in whole blood samples and were found to be significantly lower in old mice as shown in the Table.

Treatment	Control	Almond	Clove	Nutmeg	Pepper	Thyme	SED
Young	116	125	122	116	130	130	11.2
Old	63	42	69	65	87*	89*	4.3

Mean values were significantly different from control, \*P<0.03.

No significant effect of treatment was detected in the young rodents, while pepper and thyme treatments resulted in elevated levels of GSHPx in old mice compared with their age-matched controls. Lipid peroxidation capacity, as measured by malondialdehyde in the plasma, also exhibited little treatment effect with the young mice, but was significantly lower in the pepper-treated old mice than in their controls.

These results confirm the beneficial effects to be realized with a number of these culinary and medicinal plant volatile oils and suggest that they are due to their antioxidant properties preventing undesirable lipid peroxidation (Deans *et al.* 1994).

Deans, S.G., Noble, R.C., MacPherson, A., Pézenes, L.G. & Imre, S.G. (1994). *Aspects of Ageing and Disease*. Vienna : Facultas Wien.

Deans, S.G., Noble, R.C., Pézenes, L.G. & Imre, S.G. (1993a). *Age* 17, 71-74.

Deans, S.G., Noble, R.C., Pézenes, L.G. & Beregi, E. (1993b). *Role of Free Radicals in Biological Systems*. Budapest : Akademiai Kiado.

Pézenes, L.G., Noble, R.C., Beregi, E., Imre, S.G., Izsak, J. & Regius, O. (1988). *Mechanisms of Ageing and Development* 45, 75-92.

Wade, E. & Tsumita, T. (1984). *Mechanisms of Ageing and Development* 27, 287-294.



**Liver and kidney volume and their relationship to metabolic rate at rest.** By G. McNEILL<sup>1,2</sup>, M.A. FOSTER<sup>3</sup>, J. LOVE<sup>1</sup> and V. ANTFANG<sup>3</sup>, <sup>1</sup>*Rowett Research Institute, Aberdeen AB2 9SB, and Departments of <sup>2</sup>Medicine and Therapeutics and <sup>3</sup>Biomedical Physics and Bioengineering, University of Aberdeen, Aberdeen AB9 2ZD*

Basal metabolic rate (BMR) is known to be related to fat-free mass (FFM), of which the largest components by mass are muscle and bone. At rest, the most metabolically active tissues are the visceral organs such as liver, kidney, heart and brain. These tissues account for approximately 5% by weight but are believed to account for about 60% of energy expenditure at rest. The aim of the present study was to assess whether differences in liver and kidney volume could explain some of the variance in BMR/kg FFM between healthy subjects.

Thirty women aged 18-63 years, weight 48.2-76.5 kg, were recruited for measurements of liver, left kidney and spleen volume by magnetic resonance imaging using the Aberdeen Mark II imager. This imager operates at 0.08 T, thus the resolution was more limited than that of newer generation imagers. Measurements of the cross sectional area of these tissues were made over horizontal sections 16mm thick at every 20mm from the top of the diaphragm to the bottom of the left kidney. All measurements were made in the post-absorptive state. The volume of each tissue (cm<sup>3</sup>) was calculated as  $\Sigma (2 \times \text{cross-sectional area (cm}^2))$ . In two women who underwent four repeat measurements of tissue volume the CV of estimates of liver volume were 3.0 and 3.6%; of left kidney volume 2.8 and 3.8%, and of spleen volume 4.9 and 4.0%. BMR was measured using a ventilated hood indirect calorimeter; sleeping metabolic rate (SMR) was measured using whole-body indirect calorimeter chambers, and FFM was calculated from body fat estimated from four-site skinfold thickness.

The mean values for liver, left kidney and spleen volume in the thirty women were 1187 (SD 186), 120 (SD 29) and 136 (SD 50) cm<sup>3</sup> respectively. Liver and kidney volume tended to increase with FFM ( $r$  0.52 and  $r$  0.55;  $p < 0.005$ ). There was no significant relationship between spleen volume and FFM. The Table shows the correlation coefficients ( $r$ ) of BMR and SMR with FFM, liver, kidney and spleen volume.

	FFM (kg)	FFM (kg) + liver (cm <sup>3</sup> )	FFM (kg) + kidney (cm <sup>3</sup> )	FFM (kg) + spleen (cm <sup>3</sup> )
BMR (kJ/d)	$r$ 0.87 ***	$r$ 0.87 ***	$r$ 0.89 ***	$r$ 0.87 ***
SMR (kJ/d)	$r$ 0.79 ***	$r$ 0.81 ***	$r$ 0.79 ***	$r$ 0.81 ***

\*\*\*  $p < 0.0001$ .

These results provide no evidence that the volumes of liver, kidney or spleen explain any of the variance in energy expenditure at rest between individuals over and above that explained by FFM alone.

**Waist circumference predicts intra-abdominal fat mass better than waist:hip ratio in women.** By T.S. HAN<sup>1</sup>, G. McNEILL<sup>1</sup>, P. BARAS<sup>2</sup> and M.A. FOSTER<sup>2</sup>, *Departments of <sup>1</sup>Medicine and Therapeutics and <sup>2</sup>Biomedical Physics and Bioengineering, University of Aberdeen, Aberdeen AB9 2ZD*

Intra-abdominal fat mass (IFM) is believed to be more strongly associated with an increased risk of metabolic abnormalities such as hypertriglycerolaemia and insulin resistance than subcutaneous fat mass. The most suitable method for measuring intra-abdominal fat mass (IFM) *in vivo* is magnetic resonance imaging (MRI), but the equipment is expensive and not readily available for large studies. This study aimed to determine which anthropometric measurement was most closely correlated with IFM estimated by MRI.

Four cross-sectional images of the abdomen at equal distances between the xiphisternum and the anterior iliac crest were made by MRI using the Aberdeen Mark II imager which operates at 0.08T in twenty women of mean age 34.4 (SD10.0) years and BMI 25.2 (SD4.5) kg/m<sup>2</sup>. Intra-abdominal fat area was drawn on the computer screen five times for each image, and the CV of repeated areas from one image ranged from 3.1 - 3.5 %. Five subjects underwent repeat scans of one image: the CV of these was 2.3%. The mean intra-abdominal fat area in each image was used in conjunction with the distance between images to estimate volume using a truncated cone model. The volume was converted to mass by assuming that fat has a density of 0.9 kg/l and that 80% of adipose tissue is fat. Anthropometric measurements made included waist and hip circumference, saggital and transverse waist diameter (SWD and TWD), abdominal and supra-iliac skinfolds. Waist circumference was defined as the minimum circumference between the lower rib margin and the iliac crest in the mid axillary line, and hip circumference as the maximum circumference over the buttocks. Waist:hip ratio (WHR) and body mass index (BMI) were also calculated for each woman.

The Table shows the correlation coefficients (*r*) between the anthropometric variables and the IFM estimated by MRI.

	Circumferences			Diameters		Skinfolds		Ratios	
	Waist	Hip	Thigh	SWD	TWD	Abdo	Sup-il	WHR	BMI
<i>r</i>	0.89**	0.69*	0.67*	0.88**	0.88**	0.80**	0.75*	0.57*	0.85**

\**p* < 0.05 \*\**p* < 0.01

Waist circumference and SWD and TWD each accounted for about 78% of the variance in IFM in these women. The correlation between BMI and IFM was almost as good (*r*<sup>2</sup> 0.72). The lowest correlation was between WHR and IFM (*r*<sup>2</sup> 0.33). This suggests that waist circumference alone may be a better predictor of IFM in women than the more widely used WHR.