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## **PROCEEDINGS OF THE NUTRITION SOCIETY**

### **ABSTRACTS OF COMMUNICATIONS**

*A Scientific Meeting was held at the University College, Cork, on Wednesday–Friday, 22–24 June 1994, when the following paper was presented. This paper arrived too late for inclusion in Volume 53 no. 3.*

**Nutritional concerns of carers for, and nutritional status of, individuals with cerebral palsy in a residential home.** By A. FAYNE<sup>1</sup>, M. MORAN<sup>2</sup> and S. SUGRUE<sup>1</sup>, <sup>1</sup> *Dublin Institute of Technology, Kevin Street, Dublin 8.* and <sup>2</sup> *Cerebral Palsy Ireland, Sandymount, Dublin 4, Republic of Ireland.*

Cerebral palsy affects 2.4 per 1000 Irish population and is the most common cause of physical disability. Feeding problems often occur and many children and adolescents with cerebral palsy are small, underweight and stunted compared with age-standardized norms.

The purpose of this study was to assess the nutritional status of a small sample (n 8), of children and adolescents (age range 8 - 18 years) with cerebral palsy, in a residential home, and also to identify the specific nutritional concerns of those caring for them.

Information was gathered by means of a 3-d weighed food intake, anthropometric measurements (weight, tibial length, triceps skinfold thickness, and mid-arm muscle circumference), chart review, staff interview and interviewer- assisted questionnaire.

Results from this heterogenous group indicated that at least three of the eight subjects had difficulty consuming adequate diets due to the presence of mechanical feeding problems such as asymmetric tonic neck reflex, imperfect lip closure, poor bite reflex, abnormal tongue thrust and weak head and neck control. Other factors which appeared to adversely affect food intake included aspiration (n 2), vomiting (n 1), spillage (n 4), lack of speech (n 2) and lack of independent mobility (n 4). The lack of specific anthropometric and dietary standards made detailed conclusions difficult to draw. Measured weights indicated that three subjects were underweight for age, one overweight, and two stunted. The dietary energy intakes of some residents appeared much lower than others, ranging from 4.75-14.17 MJ/24h (54-153% of the estimated average requirement for age) which was not always reflected by the anthropometric measurements. Differences between these results and the care-staffs' perception of the subjects' nutritional status were numerous and many previously unidentified nutritional problems were noted by the study.

In conclusion, each member of this very heterogenous group had specific nutritional requirements which appeared to vary according to classification e.g. athetoid or spastic and physical activity. Further research is required on the incidence of feeding problems, dietary intake, nutritional status and nutritional requirements of all classification groups of cerebral palsy.

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## **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 papers were presented. These papers arrived too late for inclusion in Volume 53 no. 3.*

**Body Mass Index, body composition and age in rural Nepali men and women.** By V.R. TUFFREY<sup>1</sup>, S.S. STRICKLAND<sup>1</sup>, S.J. ULIJASZEK<sup>2</sup>, and G.M. GURUNG<sup>3</sup>. <sup>1</sup>*Centre for Human Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT*, <sup>2</sup>*Department of Biological Anthropology, University of Cambridge, Downing Street, Cambridge CB2 3DZ* and <sup>3</sup>*Centre for Nepal and Asian Studies, Tribhuvan University, Kathmandu, Nepal*

It has been proposed that body mass index (BMI = weight(kg)/height(m)<sup>2</sup>) be used to classify degrees of chronic energy deficiency (CED) in adults (Ferro-Luzzi *et al.* 1992), but the validity of BMI as an indicator of CED has been questioned. The interpretation of the index is confounded by the effects of body proportion (Norgan, 1994) and varies in terms of fatness and energy stores between the sexes, between populations (Norgan, 1990) and with age (Strickland & Ulijaszek, 1993).

Anthropometric measurements including skinfold thicknesses were made on adults of mongoloid (M) and non-mongoloid (NM) ethnic groups, from three hill villages in Nepal. By two way ANOVA, values of the Cormic index (sitting height/stature) differed by ethnic group (*n* 62 M, 71 NM, *P*<0.01) and not by sex (*n* 61 men, 72 women). Polynomials were fitted to obtain centiles by Healy's method (Healy *et al.* 1988) of body composition variables by age and BMI. The Table gives 50th centile values of variables derived from measurements in three seasons. The fall of BMI with age was greater for women than men. Interpretation of BMI in terms of percentage fat mass (%FM) varied with sex, and only slightly by ethnic group. Over the BMI range observed, values of %FM increased by 30-40%.

	<i>n</i>	BMI (kg/m <sup>2</sup> )	Fat free mass (kg)	Fat mass (kg)	%FM
Age 20 to 25 years					
M men	41	20.7	47.7	5.3	10.1
NM men	28	19.3	45.3	3.9	8.5
M women	59	21.5	37.0	10.5	22.3
NM women	53	20.0	36.6	9.6	20.7
Age 70 to 75 years					
M men	17	19.7	42.5	6.9	14.1
NM men	12	18.2	37.9	5.6	11.5
M women	14	17.8	29.0	8.7	22.8
NM women	3	17.6	28.2	8.2	22.7
BMI 16 to 17					
M men	15		37.6	4.5	10.5
NM men	23		37.7	4.3	10.4
M women	26		28.4	7.7	21.1
NM women	31		29.4	6.7	18.5
BMI 23 to 24					
M men	24		48.2	10.4	17.6
NM men	3		51.0	8.1	13.8
M women	39		37.9	14.3	27.6
NM women	13		37.9	15.3	28.6

The findings suggest that within sexes, the interpretation of BMI in terms of energy stores may not vary between ethnic groups living in the same environment, despite differences in body proportion.

This work was funded by the Overseas Development Administration.

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Ferro-Luzzi, A., Sette, S., Franklin, M. and James, W.P.T. (1988). *European Journal of Clinical Nutrition* **46**, 173-186.

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Strickland, S.S. & Ulijaszek, S.J. (1993). *European Journal of Clinical Nutrition* **47**, 9-19.

Recruitment bias to a randomized controlled trial of obesity treatment, and reasons for drop out. By C. J. WATTS, C. D. SUMMERBELL and J. S. GARROW, *Rank Department of Human Nutrition, St Bartholomew's Hospital Medical College, Charterhouse Square, London EC1M 6BQ*

Failure to recruit and randomize patients to a trial may destroy the representativeness of the sample studied and so produce a selection bias. Furthermore, drop-out of some of the randomized patients from the analysis may destroy the unbiased comparison of treatments.

We examined recruitment bias and drop-out in a randomized controlled trial of three dietary treatments for adult obesity (Watts *et al.* 1994); milk plus one discretionary food (group 1), milk only (group 2), standard energy-controlled diet (group 3). The results presented are those collected at baseline (visit 1) and after 4 weeks into the trial (visit 2).

Of fifty-nine new patients referred to the clinic between 1 November 1993 and 13 May 1994, eighteen were not randomized into the trial; six claimed a dislike of or aversion to milk, four were below the age of 18 years, three lived too far away to attend the clinic regularly (every 4 weeks for 16 weeks), and two were not willing to change their diet in order to lose weight. One patient was already being seen by a dietitian in another department of the hospital and so was referred back to her, and one patient refused to take part in the study because she "did not want to be a guinea pig". Finally, one patient, who had been referred because she was too large to fit into an MRI scanner, was not randomized into the trial because she had a history of short-term memory loss of unknown origin.

Of the forty-one patients who were randomized into the trial, two were discharged before their second visit; one became aware of the fact she was pregnant (group 2) and the other had subsequently been referred for a thyroidectomy by her General Practitioner (group 3). Another patient (group 3), who was too busy to complete the baseline questionnaires at clinic and instead took them home to post back later, never posted them back and indeed we never saw him again.

Of the remaining thirty-eight patients randomized to the trial, four did not come back for a second appointment within 6 weeks and were discharged from the trial (group 1,  $n$  2; group 3,  $n$  2), and five have still to attend. Of the twenty-nine patients who have completed visit-2 questionnaires, no weights were available for three. These three patients could not attend their clinic appointments for various reasons but were sent questionnaires which they completed and returned.

These results show that both selection and systematic bias are likely to have occurred in this randomized controlled trial of three dietary treatments for adult obesity. In terms of selection bias, adults who were not randomized ( $n$  14) did not differ from those who were entered into the trial ( $n$  41) in terms of age (mean 40.5 (SD 12.0) years  $\nu$  41.6 (SD 13.2) years), sex (13 females:1 male  $\nu$  31 females:10 males) or BMI (mean 41.4 (SD 9.5)  $\nu$  44.6 (SD 8.7)). In terms of systematic bias, seven patients have so far dropped out of the trial. Preliminary analysis does not suggest that these patients had different characteristics to those who are still enrolled ( $n$  34) in terms of age (mean 39.4 (SD 15.8) years  $\nu$  43.8 (SD 9.0) years), sex (5 females:2 males  $\nu$  26 females:8 males) or BMI (mean 48.4 (SD 6.5)  $\nu$  43.8 (SD 9.0)), but group allocation may be predictive of drop-out; more patients allocated to group 3 dropped out of the study ( $n$  4) compared with those in other treatment groups. Although we realise that the numbers are small, this group effect may be due to a lack of motivation by patients who know that they have been randomized to a standard treatment rather than a 'special' diet. This has obvious implications for the design and bias of randomized controlled trials of dietary treatments for many diseases and conditions.

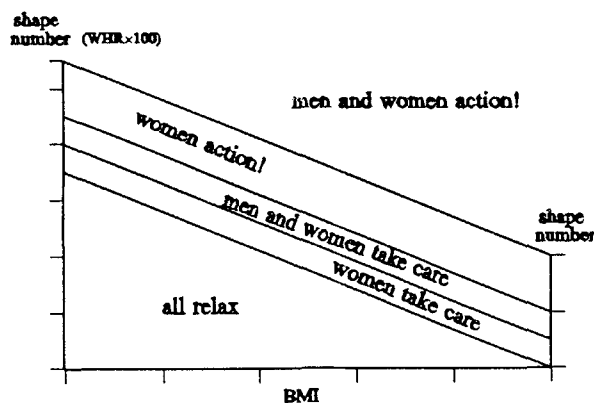
**The need for a new shape chart for assessing the risks of obesity.** By M.A. ASHWELL, *British Nutrition Foundation, High Holborn House, 52-54 High Holborn, London WC1V 6RQ*

Consistent evidence has now accumulated showing that the relative distribution of fat in the body can be as significant as the amount of fat in the body in its association with diseases such as diabetes, coronary heart disease (CHD) and some cancers. The relative distribution of fat in the body can be measured most simply in the community using the waist hip ratio (WHR); this ratio has been shown to be a reasonable proxy for the ratio of internal to external fat measured by scanning methods.

The Health Surveys for England for 1991 and 1992 (White *et al* 1993; Breeze *et al* 1994) have provided nationally representative data for both BMI and WHR in nearly 7000 men and women. They are the first surveys to assess the prevalence of obesity (12% men and 16% women have a BMI exceeding 30) and also to categorize people according to both BMI and WHR; 7% men and 5% women have a BMI exceeding 30 and a high WHR (exceeding 0.95 for men and 0.85 for women). Furthermore, the 1992 survey showed that the people with both high BMI and high WHR were most likely to have higher values for the classical risk factors for CHD, blood pressure, cholesterol and fibrinogen (Ashwell, 1993) as well as having more psychosocial problems.

The Health of the Nation (Department of Health, 1992) has set the reduction in the level of obesity as one of its priority targets. By the year 2005, the prevalence of obesity should be 6% for men and 8% for women. Such a drastic reduction from current levels requires preventive measures and prioritization for treatment.

A prototype shape chart is being evaluated (see Figure) to make it easier to identify those in the highest risk groups i.e. high BMI and high WHR so that treatment can be prioritized to this group but also to indicate those people who should be encouraged to take most care.



Successful weight reduction in these people should preferentially reduce internal fat deposits and improve their disease risk profile. Thus, a step towards the achievement of the Health of the Nation obesity targets would be accompanied by a step towards the achievement of the targets to reduce CHD and stroke at the same time.

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**Intrauterine and postnatal influences on the biochemical antecedents of atherosclerotic vascular disease in children.** By T. FORRESTER, F. BENNETT, R. WILKS, D. SIMEON, M. ALLEN, P. SCOTT, A. MORRIS and M. STEWART, *Tropical Metabolism Research Unit, University of the West Indies, Kingston, Jamaica*

The risk factors for atherosclerotic vascular disease include hypertension, diabetes, elevated serum cholesterol and obesity. Elevated cholesterol and obesity in childhood track into adult life (Freedman *et al.* 1989). Recent reports also implicate growth failure in utero and in early postnatal life as other potent risk factors for adult cardiovascular disease (Barker & Martyn, 1992).

In the present retrospective cohort study, we examined the relationships between maternal, fetal and postnatal factors and serum cholesterol concentration (Chol) and percentage glycosylated haemoglobin (GlyHb) as a proxy for glucose intolerance in children. Of 2983 eligible schoolchildren aged 6 to 16 years, 2332 (78%) were recruited. Blood pressure, anthropometric variables, Chol and GlyHb were measured. Complete antenatal and delivery data for 1610 subjects were obtained from the University Hospital records, and fetal anthropometry was available for a subgroup of 636. Multiple regression was used to identify the independent relationships between the dependent variables Chol and GlyHb, and maternal, fetal and childhood characteristics.

GlyHb was related to placental weight (PWT) and inversely related to crown heel length (CH) at birth. In childhood, triceps skinfold thickness (TSF) was also directly related to GlyHb. Cholesterol was inversely related to maternal body mass index in trimester 1 (MATBMI). It was directly related to the child's TSF and inversely to height (HT).

	Regression Slope	SE	t	P
<b>GlyHb</b>				
CH	-0.038	0.010	-3.673	0.0003
PWT (Kg)	0.535	0.267	2.004	0.045
TSF (mm)	2.026	0.709	2.856	0.005
<b>CHOL</b>				
MATBMI	-0.011	0.004	-2.646	0.008
TSF (mm)	2.669	0.736	3.627	0.0003
HT (cm)	-0.008	0.003	-2.445	0.008

Regression equations with GlyHb and Chol as dependent variables, n 636.

These results suggest that intrauterine stunting (CH) and an increased placental weight predict glucose intolerance. Fatness in childhood (TSF) is another predictor. For serum cholesterol concentration, the major influences are maternal nutritional status early in pregnancy (MATBMI) as well as stunting (HT) and fatness in childhood.

- Barker, D.J.P. & Martyn, C.N. (1992). *Journal of Epidemiology and Community Health* **46**, 8-11.  
 Freedman, D.S., Srinivasan, S.R. Harsha, D.W., Webber, L.S. & Berenson, G.S. (1989).  
*American Journal of Clinical Nutrition* **50**, 930-939.

**Intrauterine and postnatal influences on blood pressure in childhood.** By T. FORRESTER, R. WILKS, F. BENNETT, D. SIMEON, M. ALLEN, P. SCOTT, A. MORRIS AND M. STEWART, *Tropical Metabolism Research Unit, University of the West Indies, Kingston, Jamaica*

The variance of blood pressure (BP) and risk of hypertension in adults are related to several recognized risk factors including weight, activity, dietary salt, alcohol and psychosocial stress. BP in childhood tracks into adulthood, the predictive power increasing with age at first measurement (Lauer *et al.* 1991). Intrauterine growth retardation has also been reported as a potent contributor to the variance of BP and risk of hypertension (Barker & Martyn, 1992; Law *et al.* 1993). Thus, childhood associations between BP and the recognized risk factors for high blood pressure, might indicate an increased risk of developing hypertension in adulthood.

In the present retrospective cohort study, we measured BP in 2332 schoolchildren, aged 6-16 years, and examined the relationships between their BP and their anthropometry, birth weight, placental weight, maternal BP and weight during pregnancy. Complete antenatal and delivery records were obtained for 1610 of these subjects from the University Hospital of the West Indies. Stepwise multiple regression was used to identify the independent associations between BP and presumed risk factors.

	Regression Slope	SE	t	P
Weight (kg)	0.295	0.043	6.864	0.0000
Maternal BPS (mm Hg)	0.249	0.043	5.872	0.0000
Birth wt. (kg)	-2.554	0.874	-2.922	0.003

Regression equation, BPS as dependent variable, n 1610.

Systolic BP (BPS) in children was related to the child's current weight. Maternal BPS was also strongly predictive of child's BPS. BPS was inversely related to birth weight, a 1 kg increase in birth weight being associated with a 2.6 mm Hg fall in systolic blood pressure. Similar, but weaker and inconsistent relationships were shown for diastolic pressure.

These findings suggest that familiarity (maternal BPS), childhood overweight (weight), and growth retardation in utero (birth weight) are major predictors of BP in children.

Barker, D.J.P. & Martyn, C.N. (1992). *Journal of Epidemiology and Community Health* **46**, 8-11.

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**Manipulation of postprandial triacylglycerol concentration and coagulation factor VII activity with low-fat diet and / or fish-oil supplementation.** By H.M. ROCHE and M.J. GIBNEY, Unit of Nutrition and Dietetics, Department of Clinical Medicine, Trinity College Medical School, St. James's Hospital, Dublin 8, Republic of Ireland

Coagulation factor VII activity (FVIIc) is related to postprandial plasma triacylglycerolaemic (TAG) response and this in turn is related to fasting plasma TAG. High-carbohydrate diets tend to elevate fasting TAG while chronic fish-oil intake reduces both fasting and postprandial TAG. The present study examined the interactive effects of a chronic low-fat diet and low-dose fish-oil supplements on the postprandial TAG and FVIIc responses.

The study design incorporated four groups of eight healthy volunteers (3M, 5F). Two groups adhered to a low-fat diet and two groups to their usual dietary intake (full fat); one group in each fat level consumed 900 mg  $\omega$ -3 polyunsaturated fatty acid daily. The intervention period was 16 weeks. Postprandial plasma TAG and FVIIc were investigated following the consumption of a single fat meal (0.5 g fat / kg body weight). Statistical significance was investigated with repeated measures ANOVA. Results are presented below as TAG concentrations in mmol/l and FVIIc expressed as a percentage of reference plasma of known FVIIc. The postprandial response was quantified as the geometric mean area under the curve (Geomean).

DIET	Pre-Trial				Post-Trial			
	TAG Fasting	TAG Geomean	FVIIc Fasting	FVIIc Geomean	TAG Fasting	TAG Geomean	FVIIc Fasting	FVIIc Geomean
Low fat+fish oil	0.736	1.207	125.14	120.81	0.768	1.477	87.45	87.90
SD	0.309	0.520	40.15	29.57	0.406	1.006	21.95	17.63
Low fat-fish oil	1.155	2.188	144.90	140.77	1.330	2.228	104.67	98.47
SD	0.659	1.003	31.71	23.98	1.293	1.635	39.33	31.81
Full fat+fish oil	0.764	1.486	100.63	98.11	0.598	1.044	92.64	99.77
SD	0.279	0.453	17.60	25.13	0.201	0.134	34.57	34.57
Full fat-fish oil	0.735	1.306	93.09	87.86	0.809	1.298	94.82	88.48
SD	0.331	0.487	23.32	25.97	0.272	0.629	20.18	16.45

Repeated measures analysis demonstrated that the low-fat diet, without fish oil, caused significant increase of fasting plasma TAG, while concentrations were significantly reduced following fish-oil supplementation only ( $p=0.015$ ). There was a significant ( $p=0.049$ ) attenuating effect of chronic low-dose fish-oil intake on postprandial TAG response in the full-fat group. Both baseline FVIIc and the geometric area of the postprandial FVIIc response were significantly reduced ( $p=0.037$  and  $p=0.006$  respectively) in the low-fat groups.

Low fat diets significantly reduce fasting and postprandial factor VII activity and low-dose fish-oil supplements significantly reduce the hypertriacylglycerolaemic effect of low-fat diets.

This study was funded by NUTRISCAN Ltd.

**The influence of sulphur amino acids and enzymes associated with synthesis and recycling of reduced glutathione in maintaining glutathione status after tumour necrosis factor  $\alpha$  (TNF) injection in rats.** By E.A.L. HUNTER and R.F. GRIMBLE, *Department of Human Nutrition, University of Southampton, Southampton SO16 7PX*

The inflammatory effects of TNF are modulated by sulphur amino acid intake, an effect which may have important implications in the treatment of disease where the overexpression of cytokines is apparent. Maintenance of the antioxidant glutathione (GSH), via sulphur amino acid supplementation, ameliorates lung inflammation in TNF-treated rats fed on a low-protein diet (Hunter & Grimble, 1994). To elucidate the mechanism by which GSH is maintained we examined the activities of key enzymes of GSH recycling, glutathione reductase (GRed; *E.C.* 1.6.1.4) and glutathione peroxidase (GPx; *E.C.* 1.11.1.9), and synthesis,  $\gamma$  glutamylcysteine synthetase ( $\gamma$ GCS; *E.C.* 6.3.2.2), in both the fed and fasted state and investigated the effect of TNF on these enzymes in rats fed on low-protein diets supplemented with sulphur amino acids.

Groups of male Wistar rats ( $n$  15) received diets containing either 200 g casein/kg with 12 g alanine/kg and 8 g cysteine/kg (Normal Protein) or isonitrogenous low-protein diets containing 80 g casein/kg supplemented with 12 g alanine/kg (Alanine), 8 g cysteine/kg and 12 g alanine/kg (Cysteine) or 10 g methionine/kg and 12 g alanine/kg (Methionine). After 8 d one third of the animals from each group (T) received 50  $\mu$ g TNF/kg body weight intraperitoneally and were allowed free access to food for a further 24 h. On day 9 the second third of each group (P) were injected with sterile non-pyrogenic saline (9 g NaCl/l) and were pair-fed the intakes of the corresponding TNF group. The final third from each group (C) received no injection and no reduction in food intake on day 9. Liver and lung GSH, protein, GRed and GPx and liver  $\gamma$ GCS were measured 24 h after injection.

	Normal Protein			Cysteine			Low Protein Methionine			Alanine		
	C	P	T	C	P	T	C	P	T	C	P	T
<b>Liver</b>												
GSH( $\mu$ mol/g)	7.9 <sup>a</sup>	2.1 <sup>c</sup>	6.1 <sup>b</sup>	9.1 <sup>a</sup>	2.1 <sup>c</sup>	6.1 <sup>b</sup>	10.1 <sup>a</sup>	2.1 <sup>c</sup>	5.8 <sup>b</sup>	2.7 <sup>ab</sup>	2.2 <sup>b</sup>	2.9 <sup>a</sup>
GPx(U/g protein)	41 <sup>h</sup>	36 <sup>b</sup>	57 <sup>a</sup>	28 <sup>b</sup>	46 <sup>b</sup>	63 <sup>a</sup>	56 <sup>a</sup>	46 <sup>a</sup>	55 <sup>a</sup>	61 <sup>a</sup>	77 <sup>a</sup>	71 <sup>a</sup>
GRed(U/mg protein)	5.8 <sup>a</sup>	4.1 <sup>b</sup>	6.3 <sup>a</sup>	7.5 <sup>a</sup>	4.9 <sup>b</sup>	9.5 <sup>a</sup>	9.3 <sup>a</sup>	5.0 <sup>b</sup>	8.5 <sup>a</sup>	10.1 <sup>a</sup>	4.4 <sup>b</sup>	10.5 <sup>a</sup>
$\gamma$ GCS(U/mg protein)	0.9	1.2	1.1	1.0	1.0	1.0	1.2	1.2	1.0	1.0	1.2	1.2
<b>Lung</b>												
GSH( $\mu$ mol/g)	2.5 <sup>a</sup>	2.0 <sup>b</sup>	2.4 <sup>a</sup>	2.6 <sup>a</sup>	2.0 <sup>b</sup>	2.2 <sup>b</sup>	3.0 <sup>a</sup>	2.2 <sup>b</sup>	2.1 <sup>b</sup>	2.0 <sup>a</sup>	1.9 <sup>a</sup>	1.9 <sup>a</sup>
GPx(U/g protein)	36 <sup>a</sup>	45 <sup>a</sup>	41 <sup>a</sup>	39 <sup>b</sup>	67 <sup>a</sup>	62 <sup>a</sup>	53 <sup>b</sup>	43 <sup>c</sup>	71 <sup>a</sup>	38 <sup>b</sup>	61 <sup>ab</sup>	73 <sup>a</sup>
GRed(U/mg protein)	6.9 <sup>a</sup>	10.0 <sup>a</sup>	7.3 <sup>a</sup>	8.6 <sup>b</sup>	13.2 <sup>a</sup>	8.6 <sup>b</sup>	10.8 <sup>a</sup>	9.7 <sup>a</sup>	9.6 <sup>a</sup>	7.7 <sup>b</sup>	13.1 <sup>a</sup>	8.9 <sup>b</sup>

<sup>a, b, c</sup> Values in the same dietary group with unlike superscripts are significantly different (ANOVA):  $P < 0.05$ .

Whilst the Normal Protein rats grew at a rate of 7.6 g/d and the Cysteine and Methionine rats grew by 4.3 and 2.9 g/d respectively, the Alanine rats grew by 0.4 g/d. TNF treatment returns liver GSH concentration to that seen in the control groups, overcoming some of the effects of the reduction of appetite, in groups where there is sufficient sulphur amino acid in the diet. Maintenance of tissue GSH during inflammation may be due largely to recycling of glutathione rather than enhanced synthesis. An adequate dietary content of sulphur amino acid is essential for such maintenance.

The support of the Meat and Livestock Commission and the BBSRC is gratefully acknowledged.

**Urea kinetics in normal breast-fed infants measured in their home.** By H.A. STEINBRECHER, D.M. GRIFFITHS and A.A. JACKSON, *Institute of Human Nutrition, University of Southampton, Southampton SO16 7PX.*

Human milk is relatively low in protein, but contains large amounts of non-protein N, especially urea. Although urea kinetics have been measured in neonates during the postoperative period whilst being fed on an infant formula (Wheeler *et al.* 1991), there are no reports of measurements of urea kinetics in normal infants who were receiving their own mother's milk at home.

Urea kinetics were measured in the home in six infants, between 4 and 7 weeks of age, who were being breast-fed. Primed/intermittent oral doses of [<sup>15</sup>N<sup>15</sup>N] urea were given for 12 h, with the continuous collection of urine. Breast-milk samples were collected for the assessment of oral urea intake. Urea kinetics were determined from the plateau enrichment in urine of [<sup>15</sup>N<sup>15</sup>N] urea and [<sup>15</sup>N<sup>14</sup>N] urea, with appropriate allowance being made for the contribution to urea appearance from the ingested urea.

	Age (d)	Production mgN/kg/d	Excretion mgN/kg/d	Salvage mgN/kg/d
Mean	34	323	83	241
95% CI	28-40	150-526	25-140	45-468
Wheeler <i>et al.</i> (1991)	< 28	242	49	193

There was a wide variation amongst the infants for all aspects of urea kinetics, which might be accounted for by differences in physiological state and overall nutrient intake. As in the neonatal period, urea production was very high, and the proportion of urea produced which was excreted in the urine was relatively small (31%, 95% CI, 5-57). An average of 66% (95% CI, 43-95) of the urea-N produced was salvaged through the activity of the colonic microflora with over 90% of the N being retained within the metabolic pool of the body (10% was returned to urea formation).

This is the first study in which urea kinetics have been measured in normal infants in the home environment. They demonstrate that it is normal for the intensity of urea kinetics to be greater than that found in adults, with high rates of urea production and substantial salvaging of urea N. The data imply a high demand for nitrogen at this age and the practical implications of this process for the normal homeostasis and metabolic well-being during early life need to be determined.

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**Plasma antioxidant status in Peripheral Arterial Disease.** By C. O'BRIEN and M. NELSON, *Department of Nutrition and Dietetics, King's College, Campden Hill Road, London W8 7AH*

Peripheral arterial disease (PAD), which is atherosclerosis of the arteries supplying the lower limbs, has not yet been extensively studied in relation to diet. Recent results have shown that low vitamin E intakes are related to an increased risk of PAD (Donnan et al, 1993). Antioxidant vitamins such as vitamin E, ascorbic acid and  $\beta$ -carotene may help to inhibit the oxidation of low-density-lipoprotein (LDL)-cholesterol, oxidized LDL being more atherogenic than native LDL (Steinberg et al, 1989). Dietary Se may also contribute to antioxidant defence mechanisms via its incorporation into the antioxidant enzyme glutathione peroxidase (GPx; EC 1.11.1.9).

A paired case-control study was conducted to assess whether low antioxidant status is associated with an increased risk of PAD. Seventy-five cases were sex- and age-matched ( $\pm 2.5$  years) with seventy-five controls recruited either through GP practices or from hospital day wards. Plasma was analysed for vitamin E, ascorbic acid,  $\beta$ -carotene, total cholesterol and Se. Plasma vitamin E was also adjusted for total cholesterol. The following table shows their mean values separately by sex. Analysis of variance was used to assess differences between the two groups, and analysis of variance was also adjusted for pack-years of smoking.

Plasma Values	MALES (n 58)					FEMALES (n 17)				
	Cases		Controls		P adj	Cases		Controls		P adj
	Mean	SE	Mean	SE		Mean	SE	Mean	SE	
Ascorbic acid ( $\mu\text{mol/l}$ )	16.6	2.2	32.2	2.8***	*	15.8	4.7	40.8	5.1**	ns
$\beta$ -carotene ( $\mu\text{mol/l}$ )	0.45	0.04	0.58	0.1	ns	0.58	0.1	0.77	0.1	ns
$\alpha$ -tocopherol ( $\mu\text{mol/l}$ )	33.8	1.0	32.3	1.0	ns	34.5	2.6	33.4	1.6	ns
Total Cholesterol (mmol/l)	6.4	0.1	5.7	0.1***	***	6.8	0.2	6.5	0.3	ns
Vitamin E:cholesterol (mmol:mol)	5.3	0.2	5.8	0.1*	ns	5.1	0.3	5.1	0.1	ns
Selenium (ng/ml)†	73.2	2.8	79.4	2.6	*	79.9	2.2	76.7	3.5	ns

Significantly different from cases: \*  $P \leq 0.05$ , \*\*  $P \leq 0.01$ , \*\*\*  $P \leq 0.001$ , ns = not significant, † For males, n 55, P adj = P adjusted for pack-years.

The results show that plasma ascorbic acid levels, vitamin E:cholesterol ratios and total cholesterol levels were significantly lower in male cases than in controls. Ascorbic acid levels were also significantly lower in female cases. Thirteen cases had experienced myocardial infarction compared with only five subjects in the control group (chi-squared  $P \leq 0.01$  for males only) and there were also more smokers in the case group compared with the controls (chi-squared  $P \leq 0.001$  for both males and females). These results show that poor antioxidant status may contribute to the aetiology of PAD, although there is some confounding due to smoking and the presence of coronary atherosclerosis.

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**Effect of maternal glucose infusion during late gestation on the metabolic and hormonal environment at parturition in sheep.** By L. CLARKE, M.A. LOMAX and M.E. SYMONDS, *Department of Biochemistry and Physiology, University of Reading, Whiteknights, Reading, RG6 2AJ*

Underfed, winter shorn pregnant ewes exhibit raised plasma concentrations of thyroid hormones and an increased rate of glucose production (Symonds *et al.* 1988). These metabolic adaptations in response to chronic maternal cold exposure benefit the ewe by preventing pregnancy toxæmia as well as enhancing lamb birth weight (Symonds *et al.* 1986). The present study investigates the extent to which glucose infusion over the final 5-7 d gestation alters the maternal metabolic and hormonal environment during late gestation and during birth in underfed, unshorn ewes.

Nineteen unshorn ewes were fed on 60% of the energy requirements for maintenance and pregnancy (0.2-0.25 kg barley concentrate: 1.0-1.25 kg chopped hay) and infused with either glucose (GLU: *n* 10) at a rate of 0.62 mmol/min or saline (SAL: *n* 8). Maternal blood samples were taken at intervals of 1 h over an 8 h period on 145 d gestation (G) and during parturition (P:147 d) and analysed for glucose, 3-hydroxybutyrate (3HB), non-esterified fatty acids (NEFA), triiodothyronine (T<sub>3</sub>) and thyroxine (T<sub>4</sub>) as described by Clarke *et al.* (1994).

		Glucose (mmol/l)		3HB (mmol/l)		NEFA (mmol/l)		T <sub>3</sub> (nmol/l)		T <sub>4</sub> (nmol/l)	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
G	SAL	2.57*	0.14	2.32**	0.53	1.68**	0.22	1.87*	0.06	45**	2
	GLU	3.92	0.54	0.80	0.15	0.66	0.12	2.07	0.07	61	5
P	SAL	6.71†††	1.10	1.84	0.54	2.28	0.21	2.66††	0.20	65†	9
	GLU	5.90	1.50	1.34††	0.28	2.26††	0.35	2.59	0.20	66	9

Significantly different (analysis of variance) GLU v SAL: \* *P*<0.05, \*\* *P*<0.01.

G v P: † *P*<0.05, †† *P*<0.01, ††† *P*<0.001

During gestation glucose infusion resulted in higher plasma concentrations of glucose, T<sub>3</sub> and T<sub>4</sub> in conjunction with lower plasma concentrations of NEFA and 3HB. Between 145 d gestation and parturition significant increases in plasma concentrations of glucose, T<sub>3</sub> and T<sub>4</sub> were observed only in the saline-infused ewes. In contrast, plasma concentrations of NEFA and 3HB only increased in the glucose-infused group over this period.

It is concluded that chronic maternal glucose infusion in underfed, unshorn ewes results in a similar metabolic and hormonal profile to that observed in shorn pregnant ewes. However, this markedly alters the endocrine and homeorrhetic maternal adaptations associated with normal parturition at term.

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**Weight loss improves thrombotic and rheological risk factors for ischaemic heart disease.** By C.R. HANKEY<sup>1</sup>, A. RUMLEY<sup>2</sup>, G.D.O. LOWE<sup>2</sup> and M.E.J. LEAN<sup>1</sup>. *University Department of Human Nutrition<sup>1</sup> and Department of Medicine<sup>2</sup> Glasgow Royal Infirmary, Glasgow G31 2ER.*

Overweight subjects are at increased risk for ischaemic heart disease with epidemiological risk factors relating to blood flow (rheology), (Bottiger & Carlson, 1980) and haemostasis (thrombosis) (Meade *et al.* 1980). Overweight people have elevated whole blood and plasma viscosity (Ernst *et al.* 1986). Improvements in plasma and whole blood viscosity in overweight slimmers have previously been shown (Parenti *et al.* 1988).

The present study used established methodology to measure haemostatic and rheological parameters in a 12 week weight loss programme to evaluate the effect of dietary and weight change in 45 healthy overweight subjects (38 females 7 male), mean age 47 (SD 9.6) years. Diet prescription was based on estimated energy requirements (Schofield *et al.* 1985), with over 50% energy from carbohydrate, less than 30% energy from fat and less than 20% from protein and daily energy deficit of 2510 kJ. Prescribed energy intakes were 6703 kJ (SD 1623) /day. Reported energy intakes were 5745 (SD 1402) kJ, 31.8 (SD 6.1) % energy from fat, 45.2 (SD 5.9) % energy from carbohydrate and 22.2 (SD 2.8) % energy from protein, with no differences in reported intakes between weeks 1 and 12. Data were analysed using Student's paired t-test.

Weight loss resulted in significant reductions in both red cell aggregation and in factor VIIc activity, strong predictors of ischaemic heart disease (Heinrich *et al.*, 1994).

	Baseline		Week 12		Difference		P value
	Mean	SD	Mean	SD	Mean	SD	
Weight kg	93.4	19.5	88.9	19.1	-4.50	2.9	<0.001
BMI kg/m <sup>2</sup>	34.9	6.1	33.1	6.1	-1.8	1.5	<0.001
Whole blood viscosity mPas	3.06	0.4	3.01	0.38	-0.05	0.36	0.30
Plasma viscosity mPas	1.31	0.07	1.29	0.08	-0.02	0.07	0.39
Microhaematocrit %	43.1	3.7	43.5	3.6	+0.4	3.5	0.40
Red cell aggregation (arb. units)	4.44 <sup>a</sup>	1.4	4.03 <sup>a</sup>	1.3	-0.41	1.2	0.048
Fibrinogen g/l	3.26	0.9	3.45	0.7	+0.19	0.73	0.27
Factor VIIc IU/dl	114 <sup>b</sup>	21	108 <sup>b</sup>	21	-6	18.6	0.018

BMI, body mass index. a, b week 12 values significantly different (P<0.05) from baseline values.

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**Body composition changes after vertical band gastroplasty in obese females.** By N.J. FULLER, M.B. SAWYER and M. ELIA, *MRC Dunn Clinical Nutrition Centre, Cambridge CB2 2DH*

The aims of the present pilot study were twofold: firstly, to assess whether changes in body composition due to weight loss following elective vertical band gastroplasty surgery might differ from the 15% fat-free mass (FFM) and 85% fat (% body weight) indicated by Forbes (1987) for subjects with more than 65 kg initial body fat: secondly, to determine if traditional values applied to FFM density (DFFM) and hydration (HFFM) are appropriate in massive obesity and after major weight loss. Four weight-stable obese women (median age 45.5 (range 19-49) years; height 1.64 (SD 0.04) m) were studied both before gastroplasty (mean body mass index (BMI) 50.6 (SD 7.2) kg/m<sup>2</sup>) and again once stable weight had been attained, about 18 months later (BMI 35.1 (SD 3.8) kg/m<sup>2</sup>).

Total body water (TBW, kg) was assessed using deuterium- and <sup>18</sup>O-labelled water and body density by hydro-densitometry. Body fat (%), FFM (kg), DFFM (kg/l) and HFFM (%) were calculated using a three-component model incorporating these measurements (Fuller *et al.* 1992).

The Table shows body composition results before and after gastroplasty. Mean weight loss (41.9 kg) comprised 80 (SD 2.0) % fat and 20 (SD 2.0) % FFM. Absolute changes (kg) in gross body components (fat, FFM and TBW) were all strongly related to initial body and initial component weights ( $r > 0.81$ , in all cases).

	<u>Before gastroplasty</u>		<u>After weight loss</u>		<u>Difference</u>	
	Mean	SD	Mean	SD	Mean	SD
Body weight (kg)	136.3	24.3	94.4	13.6	-41.9	14.5
TBW (kg)	44.5	6.6	38.2	4.0	-6.4	3.9
FFM (kg)	61.5	8.2	53.1	5.1	-8.4	3.7
Fat (kg)	74.9	16.8	41.4	10.1	-33.5	10.9
Fat (%)	54.6	3.0	43.3	5.1	-11.3	2.6
DFFM (kg/l)	1.0996	0.0085	1.1009	0.0190	0.0012	0.0239
HFFM (%)	72.34	2.04	71.99	4.48	-0.36	5.66

The mean values for DFFM and HFFM obtained in the present preliminary study were close to those of the lean subjects in a previous study (Fuller *et al.* 1992), and showed no change with massive weight loss. Gross composition of the weight lost due to gastroplasty was not vastly different from that predicted by Forbes (1987).

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**An assessment of the validity of the 7 d weighed records (7dWR) of food intake in urban and rural Mexican women.** By C.P. SANCHEZ-CASTILLO<sup>1</sup>, S. BONNER<sup>2</sup>, MA. DE L. SOLANO<sup>1</sup>, N. LOPEZ<sup>1</sup> and G. McNEILL<sup>2,3</sup>. <sup>1</sup>*Instituto Nacional de la Nutrición Salvador Zubirán, Dept. de Fisiología de la Nutrición, México, D.F.* <sup>2</sup>*The Rowett Research Institute, Aberdeen AB2 9SB.* and <sup>3</sup>*Department of Medicine & Therapeutics, University Medical School, Aberdeen AB9 2ZD.*

Two methods have been proposed for validation of dietary records: urinary nitrogen (UN) / dietary nitrogen (DN) (Bingham and Cummings, 1985) and energy intake (EI) / basal metabolic rate (BMR) (Goldberg *et al.* 1991). The aims of the present study were: (1) to compare the agreement between the two methods, and (2) to assess the relative importance of undereating and underreporting.

Thirty-five urban and thirteen rural Mexican women aged 20-43 years were recruited. Two 7dWR of food intake were made, with body weight measured at the start and end of each week. Basal metabolic rate (BMR) was predicted from age and weight (WHO, 1985). Twenty-four hour urine collections (24 h UC) were made during the two 7dWR periods and in a further week without 7dWR. of the 24h UC in the rural and urban women, 32 and 40% were incomplete by the PABA check method (Bingham and Cummings, 1983). Only complete collections were used for the calculation of UN/DN. The Table shows the records classified as invalid by the two methods.

	Urban (n 35)		Rural (n 35)	
	Week 1	Week 2	Week 1	Week 2
	% (n)	% (n)	% (n)	% (n)
UN/DN >91%	48.5 (17)	37.1 (13)	21.1 (3)	23.1 (3)
EI/BMR <0.92	14.3 (5)	14.3 (5)	0 (0)	0 (0)
Both UN/DN >91% and EI/BMR <0.92	14.3 (5)	11.4 (4)	0 (0)	0 (0)

The proportion of invalid records was higher in urban than rural women, and was higher by UN/DN than by EI/BMR. Nine out of the ten invalid records by EI/BMR were also invalid by UN/DN. The weight change was not significantly different from zero except in week 1 in the urban women, with a gain of 297 (SD 680) g/week (P<0.05). UN excretion in the week without dietary records was not significantly different from that in the other 2 weeks in either urban (p=0.55) or rural (P=0.96) women. This suggests that the women were more likely to be underreporting than undereating during the 7dWR.

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**Pattern of production of selected crops and their influence on child nutrition in Malawi.** By M.O. Mpoma<sup>1</sup>, B.M. Margetts<sup>1</sup>, A.A. Jackson<sup>1</sup>, Kandaya<sup>2</sup> and J. Milner<sup>2</sup>, <sup>1</sup>*Institute of Human Nutrition University of Southampton SO16 17PX.* and <sup>2</sup>*Office of the President and Cabinet, Department of Economic Planning and Development, Malawi*

Food security as a major determinant of malnutrition has received increasing attention in the last decade. Malawi is a small country, with a population of 9 million people, of whom 85% live in the rural areas. The economy is predominantly agriculture which accounts for 37% of gross domestic product, 90% of total export and 85% of total labour force. The smallholder agricultural sector is the largest (Malawi government 1989 - 1992). Despite the reported improvements in national crop production, child malnutrition is still very high with 56% of all children under the age of 5 years stunted (Malawi Government, 1981). The present investigation examines the association between crop production and malnutrition in Malawi. Information from the period 1983-1993 was abstracted from the Malawi Surveillance Programme and we examined the association between pattern of production and pattern of reported levels of malnutrition among children zero to 5 years old. The analysis focused on yields of maize, rice, groundnuts, sorghum, millet, cassava, cotton and pulses. Mean crop production was calculated and production figures were plotted to establish patterns of production over the 10 years. Available energy was calculated from the total food production. Over the 10 years harvest declined by 54% for groundnuts and 41% for cassava; there were no changes in production of maize, sorghum and millet and there have been slight increases of 1.1% in rice production and 46% in cotton production. Weight-for-age values of children below 80% Havard standard were obtained from the Health Surveillance programme where children's weight are monitored. Although the method of data collection is standard, the clinic data consistently provides lower figures than controlled survey results (Chiligo 1985). Therefore the clinic data were only used to establish patterns of prevalence of underweight of under five years old children.

A regression analysis with underweight as the dependent variable and controlling for the crops in question plus ADD (agriculture development division), year, energy and food production was done. The results showed that 40% of the changes in underweight could be explained by the variables in the equation (with a *P* value of 0.02) explaining the significant link between crop production and nutritional status. The correlation was statistically significant with production of rice (-0.8).

The results suggest that there is a relationship between crop production and nutritional status in children zero to 5 years old in Malawi. The overall effect is statistically significant. Causal inferences cannot be drawn from these data. This analysis, however has provided pointers for future investigation of underweight in this agricultural community.

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**Gastrointestinal disease, diarrhoea and growth faltering in Gambian infants.** By P.G. LUNN, C.A. NORTHROP-CLEWES and R.M. DOWNES. *MRC Dunn Nutrition Centre, Cambridge, CB4 1XJ and Keneba, The Gambia.*

Growth faltering during infancy is a frequent observation in most developing countries and in many places diarrhoeal disease has generally been believed to be a major cause. This assumption however, has been challenged by Briend et al. (1989) who concluded from his studies in Bangladesh that diarrhoeal disease was not responsible for poor growth and that programmes aimed at alleviating this disease would not improve child growth. In an attempt to clarify the situation, the relationship between diarrhoea and growth have been investigated in terms of both short- and long-term growth in a longitudinal study of 119 infants, aged 2-15 months, living in a rural area of The Gambia.

During the first 3 months of life infants grew well and their z-scores for length and weight, -0.781 and -0.252 respectively reflected values at birth. However, beyond this age, and coincident with the introduction of weaning foods, both length and weight growth slowed and by 15 months of age mean z-scores had fallen to -2.094 and -2.330 respectively.

The proportion of the time that infants had diarrhoeal disease (Table) increased during this period from 2.8% at 2-3 months to a peak 10.3% at 9-12 months and on average an episode of this illness caused a 92 g loss of expected weight gain during the month in which it occurred. Because of the high prevalence of diarrhoea, it was assumed that much of the long-term growth faltering would be explained by repeated episodes of this illness. However studies of long-term growth (on average over 8.5 months, range 3-13 months) failed to show any relationship between time with diarrhoea and growth in either length or weight.

Age (months)	n	% time with diarrhoea		Lactulose:mannitol ratio	
		Mean	SE	Mean	SE
2-3	108	2.79	0.68	0.222	0.020
3-6	221	5.98	0.65	0.323	0.019
6-9	221	8.06	0.89	0.367	0.016
9-12	161	10.31	1.25	0.481	0.025
12-15	149	8.89	1.19	0.435	0.029

Use of the dual sugar (lactulose/mannitol) intestinal permeability test to assess damage to the small intestine demonstrated a progressive deterioration of the mucosa with age (Table), but in contrast to the diarrhoea data, intestinal permeability was significantly related to long-term length and weight growth. After correction for age, regression coefficients were -0.659 and -0.625, ( $P < 0.0001$ ) for length and weight respectively and the  $r^2$  suggested that this measurement could account for up to 43% of observed growth faltering. There was no correlation between intestinal permeability and time with diarrhoea in the long term although a weak relationship,  $r = 0.118$ ,  $P < 0.001$ , ( $n = 879$ ) was present when data were assessed on a monthly basis.

The results indicate that long-term growth faltering of infants in developing countries can be related to gastrointestinal disease even when no relationship can be demonstrated with diarrhoea prevalence.

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