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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Golden Jubilee Meeting of the Nutrition Society was held at Robinson College, Cambridge on Monday, Tuesday and Wednesday, 1–3 July 1991 when the following symposium papers were presented.

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The nutritional knowledge, attitudes and practices of male HIV-positive homosexuals. By C. D. SUMMERBELL and B. GAZZARD, *HIV Unit, Kobler Centre, 369 Fulham Road, Chelsea, London SW10 9TH*

Dietetic advice for HIV-infected individuals focuses on maintaining or increasing body-weight and ensuring an appropriate vitamin and mineral intake (Peck & Johnson, 1990). The general healthy eating guidelines are less relevant to this patient group. Alternative nutritional advice is widely available, although many of these strategies are based on limited evidence (Dwyer *et al.* 1988). This study aimed to identify nutritional attitudes and practices of HIV-positive gay men and relate them to the source of information.

Patients were randomly recruited over a 10-week period from outpatient clinics and asked to complete a self-administered questionnaire. The patients were in various stages of disease: asymptomatic (*n* 62); symptomatic but no AIDS diagnosis (*n* 57); and AIDS (*n* 69). Intravenous drug users were excluded from the study.

Most (93·1%) patients had altered their diet in some way subsequent to knowledge of their HIV status. Many had altered their diet in the direction of the healthy eating guidelines for the general population, as shown in the Table. The Table also shows that a large number of patients had adopted alternative sources of advice. There was a strong relationship between patients' attitudes towards the importance of specific foods and supplements in their diet and their altered consumption levels ($P<0·01$).

	Change in consumption levels subsequent to HIV diagnosis (% of total sample)			
Foods and supplements	More	Same	Less	Don't know
Cheese	13·8	61·7	21·8	2·7
Fish	27·7	60·6	9·6	2·1
Crisps	9·6	54·3	33·0	3·2
Skimmed milk	27·7	46·3	19·1	6·9
Sweeteners	6·4	56·4	23·9	13·3
Cakes	16·0	57·4	23·4	3·2
Garlic	25·5	63·8	9·0	1·6
Wholemeal bread	30·9	58·5	7·4	3·2
Butter	13·3	59·6	24·5	2·7
PUFA margarine	17·6	60·1	10·1	12·2
Vitamin supplements	46·8	41·0	4·3	8·0
Mineral supplements	26·6	55·3	5·3	12·8
Energy supplements	27·7	54·8	5·9	11·7

Those patients who had seen a dietitian (46·8%) were consuming more cakes ($P<0·001$), butter ($P<0·001$) and energy supplements ($P<0·05$), and less wholemeal bread ($P<0·01$) than before their HIV diagnosis, and had a more appropriate knowledge of the importance of nutrition in HIV disease ($P<0·05$), compared to those who had not seen a dietitian. Patients with AIDS were more likely to have seen a dietitian compared to those in the earlier stages of HIV disease ($P<0·001$).

The provision of professional nutritional advice should be a high priority during all stages of HIV disease.

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The effects of dietary energy source and tryptophan on hormones of the entero-insular axis and glucose in the early weaned pig after an intragastric infusion of glucose. By A. A. PONTER[1,2], B. SÈVE[2], N. O. CORTAMIRA[2], D. N. SALTER[1] and L. M. MORGAN[3], [1]*AFRC, Institute of Grassland and Environmental Research, Church Lane, Shinfield RG2 9AQ,* [2]*INRA, Station de Recherches Porcines, Saint-Gilles, 35590 L'Hermitage, France* and [3]*Department of Biochemistry, University of Surrey, Guildford, Surrey GU2 5XH*

The concentrations of the insulinotropic hormone gastric inhibitory polypeptide (GIP) are increased in response to glucose (Morgan *et al.* 1988) after a high-fat compared with a high-carbohydrate diet. High-fat diets also cause insulin resistance (Nagy *et al.* 1990) and tryptophan-deficient diets impair the disposal of glucose (Wittman, 1976). The present study was designed to investigate the effects of dietary energy source and tryptophan level on the response to a glucose load.

Forty-two piglets (seven litters of six piglets) weaned at 10 d were used. Within each litter six dietary regimens were represented. Two semi-purified diets were formulated to be deficient in tryptophan (113 mg/MJ). One high in carbohydrate (CO; 2·4% fat, 15·6 MJ DE/kg) and the other high in fat (FO; 22·6% fat, 18·6 MJ DE/kg). Two further diets were produced from each of the basal diets by the addition of tryptophan to give adequate (160 mg/MJ; C1, F1) and excessive (205 mg/MJ; C2, F2) levels with respect to requirements. The diets were given on the same DE basis in liquid form (feed:water; 1:3) by intragastric catheter. After 13 d, an intragastric load of glucose (3·28 g/kg$^{0.75}$) was given and blood samples were taken at intervals for 120 min and analysed for GIP, insulin and glucose.

The mean integrated (0–120 min) GIP, insulin and glucose concentrations are shown in the Table.

Tryptophan levels . . .		0	1	2	Mean	SED	*P* value
GIP	C	1949^{b}	2462^{ab}	2042^{b}	2151	E=226·0	E = NS
(ng/l per min)	F	2951^{a}	1958^{b}	2357^{ab}	2422	T=276·8	T = NS
	Mean	2451	2210	2200		E × T=391·5	E × T=0·037
Insulin	C	25·3	18·3	23·7	22·3	E=3·26	E=0·06
(mU/l per min)	F	34·9	20·6	30·2	28·6	T=3·99	T=0·035
	Mean	30·1^{a}	19·4^{b}	26·9^{ab}		E × T=5·64	E × T = NS
Glucose	C	1·74	0·74	1·33	1·27^{b}	E=0·374	E=0·001
(mmol/l per min)	F	3·32	1·82	2·83	2·66^{a}	T=0·458	T=0·034
	Mean	2·53^{a}	1·28^{b}	2·08^{ab}		E × T=0·648	E × T = NS

^{a,b} Values with different superscript letters were significantly different (ANOVA). Tryptophan levels 0, 133 mg/MJ; 1, 160 mg/MJ; 2, 205 mg/MJ. C, high-carbohydrate diet 15·6 MJ DE/kg; F, high-fat diet 18·6 MJ DE/kg; E, energy source effect (C or F), T; tryptophan level effect (0 or 1 or 2), E × T, energy source and tryptophan interaction. NS, not significant.

The high-fat diets caused changes in insulin and glucose concentrations in response to a glucose load, these changes are indicative of tissue insulin resistance. Plasma GIP concentrations were affected by an interaction between energy source and tryptophan, which may reflect changes in absorption rate or sensitivity of GIP release to glucose. In conclusion, piglets adapted to a tryptophan-adequate diet were able to control their plasma glucose concentrations more effectively than those adapted to a tryptophan-deficient diet.

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Effect of vitamin E and related compounds on mitochondrial Ca^{2+}-retention capacity. By J. PHOENIX and R. GUIDOUX, *Nestlé Research Centre, Nestec Ltd. (LABIOR), CH–1350 Orbe, Switzerland*

Previous studies with isolated intact rat soleus muscles have shown vitamin E to be protective against muscle damage caused by an elevation of intracellular Ca^{2+}. Results with various related compounds suggest that protection is afforded by the hydrocarbon phytyl chain and unrelated to antioxidant ability (Phoenix *et al.* 1989, 1990). As mitochondria act as a safety device against toxic increases in intracellular Ca^{2+}, it was of interest to investigate the effect of these compounds on mitochondrial Ca^{2+} handling.

Rat liver mitochrondria were incubated at 25°, with succinate (+ rotenone) as substrate, in the presence of α-tocopherol, α-tocopherol acetate, phytol and Trolox C (6-hydroxy-2,5,7,8-tetramethylchroman-2-carboxylic acid) at different concentrations (0–200 μM). After loading with Ca^{2+} (100 μg-ion/g protein) at low Pi level (2 mM), Ca^{2+} release was elicited by raising the Pi concentration to 6–10 mM. Net Ca^{2+} movements across the inner membrane were evaluated from extramitochondrial measurements obtained with a pCa electrode inserted in the wall of the reaction vessel.

None of the compounds tested had any effect on the initial Ca^{2+} uptake. Dose-dependent effects were exerted by α-tocopherol and α-tocopherol acetate (ANOVA, $P<0{\cdot}0001$). α-Tocopherol was more potent than α-tocopherol acetate (e.g. at 150 μM: 28·4+5·7% Ca^{2+} release (n 6) c.f. 64·7+4·1% Ca^{2+} release (n 5) for α-tocopherol acetate). The Pi-induced Ca^{2+} release was unaffected by Trolox C but appeared to be delayed by phytol.

These results show that α-tocopherol may protect mitochondria from damage by Ca^{2+} and Pi through a non-antioxidant mechanism, which may possibly account for the increased tolerance of myocytes to Ca^{2+} loads.

F. Perroti and M. Bonzon are acknowledged for excellent technical assistance.

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Effect of different dietary fats on mammary tumour incidence and on the fatty acid composition of membrane phospholipids. By CHRISTINE M. WILLIAMS and K. MAUNDER, *The Nutritional Metabolism Research Group, The School of Biological Sciences, University of Surrey, Guildford GU2 5XH*

Numerous studies in experimental animals have established that mammary tumour growth and number are influenced by the type and level of fat in the diet (Welsch, 1987). In particular, *n*-6 fatty acids enhance, and *n*-3 fatty acids inhibit, the growth of carcinogen-induced and transplantable mammary tumours (Karmali, 1987). The inhibitory effects of the *n*-3 fatty acids have been attributed to the ability of eicosapentaenoic acid (EPA) to inhibit prostanoid metabolism through displacement of arachidonic acid (AA), from membrane phospholipids, and via competitive inhibition of cyclooxygenase. However, in mammary tumour tissues most studies have measured total or choline phospholipids and have failed to show that displacement of AA by EPA occurs in this tissue. We have suggested that measurement of inositol phospholipids may be of greater relevance, since in other hormone-sensitive tissues, this phospholipid is the most important pool of AA for cell-stimulated prostanoid release. Forty-eight Sprague-Dawley rats were randomly distributed to maize oil, olive oil or fish oil (MaxEPA) (40 g/kg) diets at 3 weeks. Half the animals in each group were given the carcinogen ethylnitrosourea (ENU) orally. Animals were examined weekly for appearance of tumours. When a tumour of palpable size was detected, the animal was sacrificed along with an animal in the appropriate control group. Tumour and normal tissues were analysed for the fatty acid compositions of ethanolamine, choline and inositol phospholipids as previously described (Williams *et al.* 1989).

Tumour incidence rates in maize oil, olive oil and MaxEPA groups were 62·5, 75 and 29% respectively ($P<0·05$, MaxEPA *v.* maize oil and olive oil). There were no significant differences in the fatty acid compositions of inositol phospholipids between any of the dietary groups; stearic acid (C18:0) content of olive oil-fed animals was significantly higher in tumour compared with non-tumour tissues (41·0 (SD 9·4) *v.* 28·3 (SD 5·7); $P<0·01$).

In ethanolamine phospholipids, AA content was progressively decreased, maize oil > olive oil > MaxEPA, for both tumour and normal tissues, with statistically significantly lower amounts of AA in MaxEPA compared with maize oil-fed animals (7·8 (SD 2·8) *v.* 13·3 (SD 4·3); $P<0·05$). In maize oil-fed animals tumour tissue contained significantly less linoleic acid (2·0 (SD 0·4) *v.* 5·9 (SD 3·0); $P<0·05$), with numerically greater amounts of AA (18·0 (SD 7·6) *v.* 13·2 (SD 4·3); NS).

Of the phospholipids studied, inositol phospholipids showed the least and ethanolamine phospholipids the most marked differences according to dietary fatty acids; choline phospholipids were intermediate between the two (data not shown). The data presented suggest that if the effects of dietary fat on mammary tumorigenesis are mediated through alteration in prostanoid metabolism, the locus for this effect is not the inositol phospholipid fraction.

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Quantitative ^{13}C nuclear magnetic resonance spectroscopy: validation and application to studies of lipid metabolism. By S. C. CUNNANE, R. MCDONAGH, S. NARAYAN and T. ALLMAN, *Department of Nutritional Sciences, Faculty of Medicine, University of Toronto, Canada M5S 1A8*

^{13}C nuclear magnetic resonance spectroscopy (^{13}C NMR) has found wide application in tracing ^{13}C glucose metabolism in animals (Cohen, 1987). Using ^{13}C NMR, we have previously reported qualitative aspects of [^{13}C]acetate incorporation into human plasma lipids (Cunnane *et al.* 1989). We now report validation of similar NMR data against results obtained by quantitative capillary gas liquid chromatography (GLC). [2-^{13}C]Na acetate (MSD Isotopes, Montreal) was injected into a 40 g rat (200 mg intraperitoneally) or into a 2 kg rabbit (500 mg intravenously into portal vein). The animals were then killed under carbon dioxide gas and the livers removed (rat, 24 h later; rabbit, 1 min later). A chloroform:methanol (2:1) extract of liver total lipids containing about 100 mg lipid was prepared and quantitative ^{13}C NMR spectra obtained (Bruker 7·05; 5000 scans; 10 sec inter-pulse delay; gated-decoupling; NOE-suppression). These spectra were compared to those from the liver lipid extract of a rat or rabbit not injected with [2-^{13}C] acetate (natural abundance ^{13}C). Quantitative natural abundance ^{13}C NMR spectra of total lipid extracts of foods and rat adipose tissue samples were also obtained and the amount of lipid and fatty acid classes (% and mg/g; saturated, monounsaturated, *n*–6 and *n*–3 polyunsaturated) determined and compared to results obtained by GLC. Determination of the fatty acid composition of the foods and rat adipose tissue samples by quantitative ^{13}C NMR provided results for individual fatty acids which were highly positively correlated with those obtained by GLC (r 0·94 for mg/g; r 0·98 for % composition; n 48). 24 h after giving [2-^{13}C]acetate, ^{13}C enrichment in rat liver total lipids was distributed mainly in saturated and monounsaturated acyl carbons but also in olefinic carbons of arachidonic acid as well as in glycerol carbons of triacylglycerol and phospholipid. In rabbit liver, even after 1 min, [2-^{13}C] acetate was incorporated into C2 and C3 of saturated or monounsaturated fatty acids and into C9 of oleic acid. We believe these are the first ^{13}C NMR data describing fatty acid synthesis which have been quantitatively validated against GLC. The advantages of ^{13}C NMR over other methods of tracing ^{13}C metabolites are that it resolves ^{13}C enrichment of individual lipid carbons and provides quantitative metabolic data with minimal sample preparation. We are currently evaluating it for studies of human lipid synthesis.

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Cunnane, S. C., Wolever, T. M. S., Pencharz, P. B. & Grey, A. A. (1989). *Annual Meeting – Society of Magnetic Resonance in Medicine*, Amsterdam.

Cholesterol in the British household diet. By CECILIA MCGRATH, *University of Ulster at Coleraine BT52 7SA* and JANET LEWIS and D. H. BUSS, *Ministry of Agriculture, Fisheries and Food, London SW1P 3JR*

Between 1970 and 1975 the British household diet provided 405 mg cholesterol/d (Spring *et al.* 1978). The main contributors were eggs (34·3%), meat and meat products (24·5%), milk (13·5%) and butter (12·5%). Since that time, egg consumption has almost halved from 4·32 to 2·20 eggs per person per week, retail meat has become leaner, milk consumption has declined and one-third is now skimmed or semi-skimmed, and butter consumption has decreased from 154 g to 46 g per person per week (Ministry of Agriculture, Fisheries and Food, 1977, 1991).

Although the Department of Health and Social Security (1984) made no specific recommendations about dietary cholesterol, we wished to determine the extent to which these changes have reduced cholesterol intakes. We used the dietary records of the 7205 households participating in the National Food Survey during 1990 (Ministry of Agriculture, Fisheries and Food, 1991) and the most recent UK food composition tables (Holland *et al.* 1988, 1989). The Table shows that the national average intake has declined from 405 mg to 259 mg per person/d, with intakes slightly higher in Wales and Scotland. It also shows the contributions from selected foods.

	Britain				England	Wales	Scotland
	1970–75		1990		1990	1990	1990
	mg/d	% of total	mg/d	% of total		mg/d	
Eggs	139	34·3	62	23·9	61	60	74
Meat, total	99	24·5	86	33·2	86	90	86
Carcass meat and poultry	51	12·6	43	16·6	44	45	38
Liver and other offal	14	3·5	6	2·3	7	6	7
Meat products	34	8·4	37	14·3	35	39	42
Fish	11	2·8	11	4·2	12	12	10
Liquid milk	55	13·5	30	11·6	30	34	31
Cheese	12	2·8	15	5·8	15	12	15
Butter	51	12·5	15	5·8	14	17	19
Margarine	5	1·3	12	4·6	12	13	12
Other fats	5	1·3	6	2·3	6	7	5
Cakes and pastries	17	4·2	12	4·6	12	11	12
All other foods	11	2·7	10	3·9	10	9	11
Totals	405		259		258	264	275

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Dose-response of baked beans for hypercholesterolaemic pigs fed a Western-type diet. By NEUZA M. B. COSTA and A. G. LOW, *AFRC Institute for Grassland and Environmental Research, Shinfield, Reading RG2 9AQ* and ANN F. WALKER, *Department of Food Science and Technology, University of Reading, Reading RG6 2AP*

Plasma cholesterol levels of growing pigs may be raised by feeding a diet high in saturated fat (typical of a Western human diet), supplemented with 1% cholesterol. The same diet, when substituted with baked beans at a level of 30% on a dry weight basis leads to lower plasma cholesterol levels (Shutler *et al.* 1988). However, the effect of lower levels have not been studied.

To study the dose-response of baked beans, twenty-four Large White × Landrace pigs of 28–35 kg body-weight were made hypercholesterolaemic by feeding a semi-purified diet supplemented with 1% cholesterol for 2 weeks. After that they were divided into six homogenous blocks in accordance with their responsiveness to dietary cholesterol. One animal from each block was placed in one of the four groups and received their respective diets containing 0, 10, 20 or 30% baked beans for 4 weeks. Each diet provided about 12% of energy as protein, 40% as fat and 48% as carbohydrate, with a P:S ratio of 0·3. The diets were given twice daily at a level of 3·0% of body-weight on a dry matter basis. Fasting blood samples were taken fortnightly by venepuncture and analysed for plasma cholesterol and lipoproteins.

Results at the end of the study (see Table) show that consumption of baked beans reduced plasma cholesterol in proportion to the amounts of beans fed. Only at 30% bean substitution was a statistically-significant difference compared with the control achieved. The wide range of responsiveness to dietary cholesterol among the animals may have accounted for this. Indeed, the presence of hyper- and hypo-responders to dietary cholesterol has been noted before in pigs (Shutler *et al.* 1988) and in humans (Katan *et al.* 1986).

% Baked beans . . .	0		10		20		30	
	mmol/l	SE	mmol/l	SE	mmol/l	SE	mmol/l	SE
Total plasma cholesterol	5·98	0·67	5·66	0·86	4·77	0·68	3·85	0·31*
LDL-cholesterol	4·34	0·60	3·64	0·77	3·22	0·64	2·26	0·27*
HDL-cholesterol	1·32	0·07	1·77	0·14	1·33	0·07	1·30	0·07
VLDL-cholesterol	0·32	0·06	0·25	0·04	0·22	0·09	0·29	0·04
HDL-cholesterol:total cholesterol ratio	0·24	0·03	0·35	0·05	0·31	0·05	0·35	0·02

* Significantly different from the control (0% baked beans) by Student's *t* test ($P<0·05$).

The level of 30% baked bean substitution also significantly lowered LDL-cholesterol compared with the control. The group fed on 10% baked beans showed higher levels of HDL-cholesterol before and after the bean diet, therefore, this increase does not seem to be related to the bean intake. Although not significant, HDL-cholesterol:total cholesterol of pigs fed on baked beans was higher than the control.

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Does a low-fat diet impair growth in pre-school children? By J. A. PAYNE[1], T. R. KIRK[2] and N. R. BELTON[1], [1]*Department of Child Life and Health, University of Edinburgh EH9 1UW* and [2]*Queen Margaret College, Edinburgh EH12 8TS*

There is controversy about whether recommendations to decrease the intake of fat in the UK diet to reduce heart disease should be applied to children under the age of 5 years. The prevailing view is one of caution due to concern that low fat intakes may impair growth and development (Department of Health and Social Security, 1988). However, there has been very little data on nutritional intake and growth parameters in this age group with which to make an informed judgement.

In the present study, energy and nutrient intakes were assessed in 153 children aged 2–5 years from Edinburgh using the 7 d weighed inventory method. Height, weight, mid-arm and mid-calf circumferences, and triceps and subscapular skinfold thicknesses were also measured and compared to current UK standards (Tanner *et al.* 1966; Tanner & Whitehouse 1975). Growth velocity data were obtained for a subset of fifty-four children from a 1 year follow-up study. Children with fat intakes below 30% energy from fat (group mean 27·7%, LF) were compared to those with fat intakes greater than 40% energy from fat (group mean 42·7%, HF). The Table shows that there are no significant differences in energy intakes or growth parameters between the groups, as assessed by independent *t* tests.

Comparison of energy intake and growth parameters

Group . . .	LF		HF		
Number . . .	20		23		
Male:female ratio . . .	11:9		10:13		
	Mean	SD	Mean	SD	
Energy (MJ (kcal))	4·91 (1174)	0·73 (174)	4·98 (1192)	1·12 (269)	NS
Age (months)	40	10	39	10	NS
Height (cm)	98·6	6·7	97·3	7·8	NS
Height percentile	65	30	68	22	NS
Weight (kg)	15·8	2·4	15·5	2·7	NS
Weight percentile	60	31	60	30	NS
Sub-scapular skinfold (mm)	5·8	1·5	5·7	1·3	NS
Triceps skinfold (mm)	9·7	1·9	9·3	1·9	NS

For the subset of children for whom growth velocity data were available there was no significant correlation with % energy from fat.

In conclusion, this study found no evidence to suggest that the growth of Edinburgh children taking a low-fat diet was compromised.

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Is milk a suitable supplementary food for the nutritional support of West African children? By K. ERINOSO, S. HOARE, S. SPENCER, P. G. LUNN and L. T. WEAVER, *MRC Dunn Nutrition Unit, Keneba, The Gambia and Cambridge, England*

Milk represents a major source of protein, energy and calcium in early life, and in some parts of the world it makes a significant contribution to diet during childhood. However, lactose intolerance, common in many parts of Africa, reduces the suitability of milk as a supplementary food. To define the age-related prevalence of lactose maldigestion (LM) 218 children aged 12–72 months from the rural Gambian villages of Keneba, Kanton Kunda and Manduar were studied; of these 99% were ethnic Mandinka.

After an overnight fast, each child ingested an aqueous solution of lactose (2 g/kg body-weight) followed by the collection of end-expiratory breath samples half-hourly for 3 h obtained using a mask or 'blow-out'. Breath hydrogen was measured using an electrochemical analyser (GMI, Scotland). A rise in breath hydrogen concentration of >20 ppm above fasting baseline was taken as an index of lactose maldigestion.

Age (months) . . .	13–24	25–36	37–48	49–60	61–72	All (13–72)
Total *n*	34	42	46	42	54	218
LM *n*	7	32	35	32	43	149
LM %	21	76	76	76	80	68

There was a significant rise in the percentage of lactose maldigesters from 21% to 76% between the 2nd and 3rd–5th years of life ($P<0{\cdot}001$) (see Table). Only eight children had diarrhoea in the 2 weeks before the test. Clinical signs of lactose intolerance (diarrhoea, abdominal discomfort or flatus) followed in only seven children (3%), four of whom had diarrhoea before the test.

There was no significant difference in the mean time of introduction of supplementary diet, or cessation of breast feeding between the lactose digesters and maldigesters aged 12–36 months. However, there was a greater proportion of normal lactose digesters than maldigesters in the group of infants still receiving breast milk (85% *v.* 15%), and greater proportion of maldigesters in the fully weaned group (63% *v,* 37%) ($P<0{\cdot}001$).

Throughout the age range there was no significant relationship between weight-for-age (WFA) or weight-for-height (WFH) and the ability to digest lactose: the majority of both digesters and maldigesters at all ages lay in the 80–90% WFA, and 90–100% WFH ranges (NCHS standards). There was no significant relationship between the results and history of antibiotic treatment during the 2 weeks preceding the test.

This study shows that the ability to digest lactose is lost in the majority of children during the second year of life, coincident with the cessation of breast feeding. The lack of association between lactose digestion and anthropometry suggests that in this rural West African community the inability to digest lactose is unlikely to be a major factor contributing to childhood growth failure. However, the high prevalence of lactose maldigestion in children >2 years suggests that milk may be an inappropriate supplementary food for this age group.

Iron intake during weaning and the protein–energy nutritional and iron status of Asian toddlers in Sheffield. By M. B. DUGGAN, *Department of Paediatrics, University of Sheffield*

Full information from a study of the 4 d weighed energy intakes, protein–energy and biochemical iron status is available on ninety-six healthy Asian children aged 4–40 months, and living in Sheffield.

Sixteen of these children were found to have Fe-deficiency anaemia, fulfilling all the following criteria for this diagnosis; HB <11 g/l, serum ferritin <10 μg/l, and erythrocyte protoporphyrin >80 mmol/mol haem. All were aged >12 months. A control group of twenty-four children, in whom all of the above-mentioned markers for Fe-deficiency were negative, and who were all >12 months of age, was identified.

The following variables were compared by Student's *t* test for unpaired data (all analyses with 38 df) viz; weight, crown–heel length and weight-for-length SD scores, (SDS; in which weight and length data are expressed in terms of their SD from the means of an age-appropriate reference population) total and standardized energy intake, dietary energy (E) density, % of total dietary energy coming from milk, and dietary Fe intake mg/d. The following significant differences were observed.

	Fe-deficiency anaemia		Normal Fe status		
Variable	Mean	SE	Mean	SE	*t* value
Weight SDS	0·14	0·30	–0·9	0·21	2·83
Crown–heel length SDS	0·67	0·27	–0·47	0·22	3·27
Diet E density (kJ/g (kcal/g))	3·423 (0·819)	0·171 (0·041)	4·063 (0·972)	0·238 (0·057)	–2·84
% Diet E from milk	59·3	4·73	42·0	4·45	2·71

Sheffield Asian parents rely heavily on commercial baby foods and also avoid pork and meats which have not been slaughtered in the Islamic tradition. This results in avoidance of many baby foods containing haem-iron. Nevertheless, the weight- and length-for-age data suggest that the children are well grown. The apparently banal observation that nutritional anaemia can coexist with relatively good protein–energy nutritional status is of importance because of the reliance placed by community child health workers on weight-for-age as an indicator of nutritional status. Screening for iron deficiency is probably justified in this vulnerable group.

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Tissue protein turnover in normal rats after a single dose of rat growth hormone. By J. A. MARTINEZ and A. S. DEL BARRIO, *Department of Nutrition and Food Science, University of País Vasco, Vitoria, Spain* and J. LARRALDÉ, *Department of Physiology and Nutrition, University of Navarra, Pamplona, Spain*

Somatotropin (GH) has multiple actions on growth, body composition and function through direct or indirect effects on nutrient homeorhesis (Buttery & Dawson, 1990). In this context, GH has been reported to increase protein deposition in a wide variety of species; however, virtually all studies conducted to date in laboratory animals have been performed in GH-sensitive animals or by using in vivo conditions (Pell & Bates, 1990). The aim of this communication was to evaluate the short-term effects of GH on in vivo tissue protein turnover in intact female rats.

Overnight-fasted female intact rats weighing about 50–55 g received a single subcutaneous dose (60 μg) of rat GH (rGH) 40 min before protein synthesis rate (Ks) was assessed in liver and gastrocnemius muscle by using the phenylalanine large-dose method as validated for intraperitoneal injection (Martínez, 1987). Concentrations of immunoreactive somatomedin-C (IGF-1) were measured in unextracted serum, using a non-equilibrium technique. Nucleic acids content and cathepsin A activity in liver and gastrocnemius muscle were also measured.

	Control (*n* 6)		GH-treated (*n* 6)		Statistical significance
	Mean	SD	Mean	SD	
Plasma IGF-1 (U/l)	0·91	0·15	0·92	0·05	NS
Liver:					
Weight (g)	1·66	0·04	1·67	0·08	NS
RNA/DNA (mg/mg)	2·06	0·51	2·18	0·31	NS
Cathepsin A (U/g)	128·6	10·3	123·7	7·8	NS
Ks (%/d)	52·1	12·5	54·2	20·6	NS
Gastrocnemius:					
Weight (g)	0·48	0·04	0·48	0·03	NS
RNA/DNA (mg/mg)	1·37	0·38	1·46	0·25	NS
Cathepsin A (U/g)	55·9	1·7	58·5	2·3	$P<0·05$
Ks (%/d)	14·4	2·4	18·0	3·5	$P<0·05$

NS, not significant (Student's *t* test).

The homeorhetic responses to GH in the partitioning of nutrients towards specific processes has been attributed either to a direct interaction with its receptors, or indirectly by systemic or local stimulation of IGF production (MacRae & Lobley, 1991). Other possible factors involved in the mechanism of action are the modulation of GH circulating concentration by GH or IGF's binding proteins, changes in the activity of other hormones, or changes in GH or IGF-1 receptors populations and cell signalling. In this context, short-term effects are associated with protein-sparing functions (Pell & Bates, 1990). Thus, our findings suggest that the acute treatment induces rapid changes in both muscle protein synthesis and degradation apparently not mediated by circulating levels of IGF-1; however, no changes were observed in liver protein turnover.

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Effect of salbutamol on skeletal muscle functional capacity in man. By LUCIE MARTINEAU[1], MICHAEL A. HORAN[3], RODERICK A. LITTLE[2] and NANCY J. ROTHWELL[1], [1]*Department of Physiological Sciences,* [2]*North Western Injury Research Centre, University of Manchester, Manchester M13 9PT* and [3]*Department of Geriatric Medicine, Hope Hospital, Salford M6 8HD*

Numerous reports have shown that a variety of β_2-agonists (e.g. clenbuterol, cimaterol) rapidly reduce body fat content and increase lean tissue mass in small mammals (e.g. Emery *et al.* 1984). No published information is available for humans, possibly due to the absence of clinically-approved β_2-agonists comparable to clenbuterol or cimaterol. Salbutamol, a β_2-agonist used clinically, has not been reported to induce physiological or functional changes in muscle of experimental animals. However, we have recently demonstrated that infusion of salbutamol in rats induces the same anabolic effects as clenbuterol (unpublished data). The present study investigated the effects of a sustained-release preparation of salbutamol on skeletal muscle functional capacity in humans.

Twelve healthy, male volunteers (17–46 years; 62–83 kg) ingested either salbutamol (Volmax, 8 mg; Glaxo UK) or a placebo (ascorbic acid) twice daily for 14 d. Measurements were performed immediately prior to initiation of the treatment, and after 1 and 2 weeks. The strength of the non-dominant quadriceps increased after 7 d in all subjects taking salbutamol (mean (SEM)) (9 (2%); $P<0{\cdot}05$), and remained elevated after 14 d (13 (4%); $P<0{\cdot}05$). This increase was half that observed after 14 d for the dominant quadriceps (24 (6%); $P<0{\cdot}05$). While the strength of the non-dominant hamstrings remained constant in the salbutamol-treated group, the strength of the dominant hamstrings increased after 7 d (18 (5%); $P<0{\cdot}05$), but returned to baseline values 1 week later. The grip strength of both hands was also increased for only 7 d in these subjects (9 (2%); $P<0{\cdot}05$). The maximal static inspiratory mouth pressure increased after 14 d on salbutamol (8 (2%); $P<0{\cdot}05$); the expiratory mouth pressure remained constant. No significant changes in muscle strength were observed for the control group during the trial. Furthermore, there were no significant changes in body-weight, skinfold thickness (measured at five sites), or arm and leg circumferences in either group.

These data suggest that short-term administration of a slow-release preparation of salbutamol increases muscle strength in man. However, the magnitude and duration of this effect vary between muscle groups.

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Energy intake and expenditure in body-builders. By R. M. QUEVEDO[1], M. COX[1], W. A. COWARD[3], D. JONES[2], P. PACY[1], I. SMEATON[1], D. THORPE[1] and D. J. MILLWARD[1], [1]*Nutrition Research Unit, London School of Hygiene and Tropical Medicine, St Pancras Hospital, 4 St Pancras Way, London NW1 0PE*, [2]*Rayne Institute, University College Hospital, University Street, London WC1* and [3]*MRC Dunn Nutrition Unit, Downhams Lane, Milton Road, Cambridge CB4 1XJ*

In adults, energy requirements are determined by rates of energy expenditure, which in turn are a function of basal metabolism, thermogenic processes and physical activity. Whilst the prediction of basal metabolism (RMR) can be made with reasonable confidence on the basis of established prediction equations, energy expenditure above basal is more difficult to predict without detailed information of patterns and extent of physical activity. It is also the case that there is increasing concern that the measurement of energy intakes, assumed to equal rates of energy expenditure in adults in balance, may underestimate actual values. We report here studies designed to investigate the relationship between measured energy intake, prediction equations for energy expenditure and actual energy expenditure in a cohort of body-builders.

Measurements were made in ten adult body-builders (seven male and three female). Mean body-weights 74 (SD 12) kg, mean age 30 (SD 6) years. Resting metabolic rate (RMR) was measured by indirect calorimetry by means of a ventilated hood, and total energy expenditure was measured by the doubly-labelled water technique. Body composition was determined by densitometry (underwater weighing), by bioimpedance, and from body water estimated from the ^{18}O space. Food energy intake was determined by a 7 d weighed-intake protocol. All subjects were judged to be highly compliant. The body composition results will be reported separately.

The ratio of measured RMR to that predicted by the Schofield equations (Schofield *et al.* 1985) was 1·04 (SD 0·12). The food energy intake was 1·69 times the RMR (SD 0·23) and the protein:energy ratio was 20·7 (SD 4·8). The total energy expenditure was calculated from the CO_2 production rate using a conversion factor based on the estimated RQ from the composition of the food (i.e. the food Q) the mean value being 0·87 (SD 0·02).

Total energy expenditure was 192 (SD 30) kJ/kg, equivalent to a physical activity level (PAL, i.e. total energy expenditure/RMR) of 1·95 (SD 0·25), higher than that calculated from the activity diary (1·66 (SD 0·08)), with no correlation between individual values. The ratio of energy expenditure over energy intake was 1·11 (SD 0·15, range 0·95–1·34). Since, with one exception, all subjects maintained weight throughout the period of measurement, these results would imply that energy intakes were underestimated.

An examination of the relationship between the energy expenditure above basal metabolism and body-weight revealed no significant relationship.

In conclusion, then, for this group of body-builders total energy requirements are, on average, 192 kJ/kg, a value implying a PAL of 1·95.

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Resting metabolic rate and body composition of normal and athletic adults. By R. M. QUEVEDO[1], M. COX[1], D. J. MILLWARD[1], R. HESP[2], D. HALLIDAY[3] and P. PACY[1], [1]*Nutrition Research Unit, London School of Hygiene and Tropical Medicine, St Pancras Hospital, 4 St Pancras Way, London NW1 0PE*, [2]*Department of Medical Physics, Clinical Research Centre, Northwick Park, Harrow* and [3]*Nutrition Research Group, Clinical Research Centre, Northwick Park, Harrow*

We report here studies designed to determine the influence of body composition on the resting metabolic rate (RMR). We have measured RMR and body composition (fat and fat-free mass (FFM)) in male and female body-builders, elite light- and heavy-weight female rowers, two groups of non-athletic males (differing in age) and one group of younger females. RMR was measured by the ventilated-hood technique, whilst body composition was measured by a combination of underwater weighing, bioimpedance, body water from the ^{18}O space, total body potassium and skin-fold thickness.

For the data set as a whole, RMR was more significantly related to FFM than to body-weight (r 0·776 *v.* 0·713), although for men the difference was smaller (r 0·744 *v.* 0·722) than in women (r 0·856 *v.* 0·722). Multiple regression of RMR on fat and lean tissue with zero intercept (according to the model of Garby *et al.* 1988) indicated values for the regression coefficients for the entire group, men and women respectively, of 113 (SE 4·3), 101 (SE 6·0) and 130 (SE 4·4) kJ/kg per d for FFM and 37 (SE 19·6), 64 (SE 27·8) and −4·5 (SE 19·0) kJ/kg per d fat.

		Age (years)		RMR (kJ/kg)		Fat (% body-wt)		FFM (kg)		RMR/FFM (kJ/kg)	
	n	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Male controls (a)	11	27·2	6·9	97	9·6	16·7	5·7	57·8	8·9	116	12·1
Male controls (b)	4	47·5	5·1	101	7·5	24·9	8·1	48·6	1·6	135	6·7
Male body-builders	17	28·3	4·9	95	12·1	15·5	4·7	68·8	7·1	112	14·2
Female controls	9	24·4	4·2	97	13·4	24·1	3·9	41·4	3·7	128	13·4
Female body-builders	13	27·2	5·1	99	7·5	19·0	4·4	50·6	4·9	123	11·3
Light-weight rowers	11	27·1	3·5	117	9·2	11·6	5·3	53·0	3·5	132	6·3
Heavy-weight rowers	11	26·6	3·8	108	7·5	19·0	3·5	58·4	3·6	134	9·6
All men	32	30·3	8·6	96	10·9	17·1	6·1	62·5	10·3	117	14·6
All women	42	26·5	4·2	106	11·7	18·2	6·0	51·2	7·0	129	10·9
All subjects	74	28·1	6·7	102	12·5	17·7	6·0	56·0	10·2	124	13·8

RMR, resting metabolic rate; body-wt, body-weight; FFM, fat-free mass.

These results confirm the influence of body composition on RMR but also indicate potential confounding effects. Sex is important; one possibility being the composition of FFM, with more skeletal muscle in FFM in men. A changing composition of FFM may also explain an influence of size on the RMR (i.e. an increasing proportion of muscle mass with size) since in men the RMR/FFM was inversely related to FFM (r −0·576). However, there was no influence of size in women (r 0·04). This might reflect the second confounding influence, i.e. any effect of habitual physical activity on the metabolic rate. This would tend to confound the size influence since the athletes had the larger FFM in each sex. Clearly, any further resolution of the influence of physical activity on the RMR requires careful matching for FFM size.

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