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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Three Hundred and Ninety-eighth Meeting of the Nutrition Society was held in the Barnes Lecture Theatre, Royal Society of Medicine, London, on Tuesday, 15 May 1984, when the following papers were read:

Seasonal changes in nutritional status of urban Gambian children. By A. M. TOMKINS^{1,2}, R. HAYES² and D. DUNN², ¹Medical Research Council Laboratories, Fajara, nr Banjul, The Gambia and ²London School of Hygiene and Tropical Medicine, London WC1E 7HT

In previous studies of the epidemiology of protein-energy malnutrition in The Gambia, the increase in numbers of malnourished children in a rural community during the rains (June–September) has been attributed to shortages of food (the hungry season), difficulties in child care because of intense farming activity by women and changes in patterns of morbidity (Rowland *et al.* 1977). There is no information on the nutritional status of urban communities.

Bakau Old Town, on the Atlantic coast, was selected because of its stable population among whom agricultural activity is rare and food supplies in the market are usually plentiful throughout the year. A series of anthropometric measurements were made on children age 6–36 months to cover the different seasons. Results are shown in the table.

Proportion of children with less than 2 Z scores for anthropometric measurement (compared with N.C.H.S. standard; World Health Organization, 1983)

Anthropometric measurement	Season	Age (months)				
		6–11	12–17	18–23	24–29	30–36
Wt/age	May 1981	6	18	15	17	10
	Sept 1981	14	33	28	26	17
	Feb 1982	10	16	15	17	11
	Sept 1982	19	12	31	28	19
Height/age	May 1981	8	15	24	29	30
	Sept 1981	8	21	25	37	28
	Feb 1982	4	16	27	27	39
	Sept 1982	11	10	24	42	33
Wt/height	May 1981	2	9	7	7	1
	Sept 1981	6	16	11	8	3
	Feb 1982	0	7	2	8	0
	Sept 1982	5	11	16	7	7

The proportion who were underweight was greatest during the second year of life and was nearly always increased when measured at the end of the rains. There was a greater proportion of short children in the older age groups but there was no evidence of any seasonal effect on height. The proportion of thin children was greatest during the second year of life; the proportion frequently being greatest when measured after the rains.

These proportions of malnourished children are lower than in Keneba, a deprived rural area. However, they do show a marked seasonal pattern of malnutrition which could not be attributed to agricultural activity or local food shortage. This suggests that other factors, such as infection, may play an important role in the weight faltering that occurs among Gambian children even in a more privileged community.

Rowland, M. G. M., Cole, T. & Whitehead, R. G. (1977). *British Journal of Nutrition* 37, 441–450.
World Health Organization (1983). *In Measuring Changes in Nutritional Status*. WHO Monograph. Geneva: WHO.

The effect of acute malaria infection on nitrogen metabolism in young children. By A. M. TOMKINS^{1,2}, P. J. GARLICK², E. FERN² and J. C. WATERLOW², ¹*Medical Research Council Laboratories, Fajara, nr Banjul, The Gambia*, and *London School of Hygiene and Tropical Medicine, London WC1E 7HT*

Growth faltering and weight loss have been described during several systemic infections in children accompanied by a catabolic loss of body nitrogen. In a previous investigation of a group of well-nourished children who were studied during the acute stage and convalescent phase of measles, we showed that rates of breakdown and synthesis of whole body protein were raised during acute infection (Tomkins *et al.* 1983). Urinary excretion of N, creatinine and 3-methylhistidine was also increased. During these studies the children received a constant, formula diet providing 70 mg N and 418 kJ (100 kcal)/kg body-weight per d. It was not possible to assess whether these changes in N metabolism would have been prevented by the provision of extra nutrients.

In the present study, we investigated five well-nourished children (aged 4 months to 6½ years) during the acute and early convalescent phase (4–6 d later) of acute infection with *Plasmodium falciparum* malaria. The children received a formula diet providing 200 mg N and 502 kJ (120 kcal)/kg body-weight per d during both phases of the illness. Rates of whole-body protein synthesis and breakdown were estimated by measurement of the excretion of ¹⁵N in urinary ammonia after an oral dose of [¹⁵N] glycine, as previously described (Garlick *et al.* 1980).

Most children excreted greater quantities of urinary N (g N/kg body-weight per 9 h) during the acute phase (mean 0.138 (SE 0.038)) than during early convalescence (mean 0.097 (SE 0.07)). Similarly, most children excreted more urinary creatinine (μmol/kg body-weight per 9 h) during the acute phase (mean 344 (SE 38)) than during early convalescence (mean 305 (SE 65)). The mean values (g protein/kg body-weight per 9 h) for rates of protein breakdown (4.87 (SE 1.97)) and synthesis (4.75 (SE 2.18)) were higher in the acute phase than early convalescence (2.69 (SE 0.71), 3.20 (SE 0.68) for breakdown and synthesis respectively).

These results show a similar pattern to our study of children with measles in that rates of protein breakdown and, to a lesser extent, synthesis, are elevated during systemic infection. However, the rates of protein synthesis were higher and quantities of urinary N were lower than in our previous study.

Although the two studies are not directly comparable it is possible that the greater protein intake in the present study, by stimulating synthesis of protein, prevented the sometimes catastrophic loss of body N in children with severe infection. The implication is that even in severe systemic infection the 'obligatory' N loss may not be obligatory if sufficient nutrients are given.

Garlick, P. J., Clugston, G. A. & Waterlow, J. C. (1980). *American Journal of Physiology* **238** (*Endocrinology and Metabolism* 1), E235–E244.

Tomkins, A. M., Garlick, P. J., Schofield, W. M. & Waterlow, J. C. (1983). *Clinical Science* **65**, 313–324.

Vitamin status of elderly residents in Part III accommodation. By K. O. CHUNG-A-ON¹, D. E. THOMAS², S. F. TIDMARSH², D. M. SHAW² and J. W. T. DICKERSON¹, ¹*Department of Biochemistry, University of Surrey, Guildford, Surrey GU2 5XH* and ²*Biochemical Psychiatry Laboratory, Welsh National School of Medicine, Whitchurch Hospital, Whitchurch, Cardiff CF4 7XB*

There are a number of reports of sub-clinical nutritional deficiency amongst old people living at home and in institutions (Vir & Love, 1979). In contrast, there are few reports on the nutritional status of residents in Part III accommodation although low status with respect to folic (Read *et al.* 1965) and ascorbic acids (Andrews *et al.* 1969) has been described.

In our study, fifteen residents (mean age 85.1 years) were selected from four old people's homes. The home menus were reviewed and it was decided that a weighed dietary measurement on three consecutive days would give a reasonable estimate of intake. This restricted period also made it possible to obtain similar information from thirty-five community controls (mean age 74.3 years). A fasting blood sample was obtained on the day following the dietary measurements. Plasma and leucocyte ascorbic acid and serum and erythrocyte folate were determined by standard techniques. Thiamin, riboflavin and pyridoxine status was determined by measurement of the activity coefficients of erythrocyte transketolase (*EC* 2.2.1.1), glutathione reductase (*EC* 1.6.4.2) and aspartate aminotransferase (*EC* 2.6.1.1) respectively.

The thiamin pyrophosphate values showed that 50% of the subjects in each group had evidence of thiamin deficiency. More residents than controls had evidence of ascorbic acid and folate deficiency. Only one control had evidence of riboflavin deficiency and none of pyridoxine deficiency.

Energy intakes tended to be lower in the residents with a greater proportion of values below the recommended daily allowance (RDA) (Department of Health and Social Security, 1979). A greater number of residents than controls had low intakes of ascorbic acid, thiamin and iron. All the subjects received less than the RDA of vitamin D for the housebound elderly. The actual intakes of ascorbic acid are likely to have been lower than those calculated due to cooking losses.

Many of those living in Part III accommodation were there because they were not able to look after themselves and were likely to be in a poor nutritional state on entry into the home. Although on superficial examination the menu appeared to provide an adequate diet, the amounts eaten by the residents suggested the need for serious reviews with respect to some nutrients.

It is assumed that the residents supplement their diet with food bought with their pocket money. In the fifteen residents investigated, this was not the case, except for the occasional biscuit or sweet which increased energy intake only. Demand for Part III accommodation is increasing and it would seem that there is a real need for a review of the diet if the nutritional requirements of the residents are to be met.

Andrews, V., Letcher, M. & Brook, M. (1969). *British Medical Journal* **ii**, 416-418.

Department of Health and Social Security (1979). *Recommended Intakes of Nutrients for the UK*, pp. 6-7. London: H.M. Stationery Office.

Read, A. F., Gouch, K. R., Pardoe, J. L. & Nicholas, A. (1965). *British Medical Journal* **ii**, 843-848.

Vir, P. & Love, A. M. G. (1979). *American Journal of Clinical Nutrition* **32**, 1934-1947.

Tryptophan and related variables in Part III accommodation residents. By S. F. TIDMARSH¹, K. O. CHUNG-A-ON², D. E. THOMAS¹, D. M. SHAW¹ and J. W. T. DICKERSON², ¹*Biochemical Psychiatry Laboratory, Welsh National School of Medicine, Whitchurch Hospital, Whitchurch, Cardiff CF4 7XB* and ²*Department of Biochemistry, University of Surrey, Guildford, Surrey GU2 5XH*

Lehmann *et al.* (1981) suggested that a proportion of old people do not absorb tryptophan normally and that this may be associated with dementia. Of the fifteen residents investigated in Part III accommodation, seven of them had signs of memory loss as indicated by the Hare scale (Hare, 1978). A fasting blood sample was obtained for the determination of tryptophan, albumin, non-esterified fatty acids (NEFA) and insulin. When the residents were split into two groups, those with and those without signs of memory loss, there was no significant differences between the variables investigated.

In a previous study (D. M. Shaw and S. F. Tidmarsh, unpublished results) of patients with senile dementia, fasting plasma tryptophan levels were lower ($P < 0.001$) in the patients than in controls. In the present study, fasting levels of tryptophan in the residents were similarly lower than those of the healthy elderly ($P < 0.02$), although the difference was smaller. There was no correlation between plasma tryptophan and dietary tryptophan in patients with dementia, although there was a significant correlation in the controls. The residents also showed no significant correlation for dietary and plasma tryptophan, yet tryptophan intakes were similar between the groups, suggesting that other factors such as increased requirement or renal losses could be responsible for the lower plasma concentration.

There were no significant correlations between NEFA and free and total tryptophan, and for albumin with free and total tryptophan in the residents as was found in patients with senile dementia. However, the controls had a significant negative correlation between NEFA and free tryptophan, in accord with the report of Curzon & Knott (1974).

Insulin concentration in the plasma of residents was similar to those of patients and controls investigated previously, and showed no significant differences. It would therefore seem that although the group did not exhibit differences between those with and those without memory loss, the group exhibits the trends seen previously in patients with senile dementia.

Curzon, G. & Knott, P. J. (1974). *British Journal of Pharmacology* 50, 197-204.

Hare, M. (1978). *British Medical Journal* ii, 226-227.

Lehmann, J., Persson, S., Wallinder, J. & Wallin, L. (1981). *Acta Psychiatrica Scandinavica* 64(2), 123-131.

Effect of methylxanthines on the volume and composition of milk from Wistar rats. By ALEXANDRA D. HART and R. F. GRIMBLE, *Nutrition Department, Southampton University, Southampton SO9 5NH*

Methylxanthines commonly occur in the human diet. The main ones are caffeine, theobromine and theophylline due to the consumption of coffee, tea, chocolate products and cola drinks. The present study examined the effects of methylxanthines on the volume and composition of milk from Wistar rats.

Standard laboratory chow was given and either caffeine, theobromine, theophylline or a mixture of all three, were administered via drinking water during pregnancy and lactation. A control group received plain water. The dams received the drugs until the 14th day of lactation. Three days after birth, litter sizes were equalized to eight. Pup growth was measured as an index of lactational performance. The methylxanthines were given in the proportions occurring in tea. The mean daily doses for caffeine, theobromine and theophylline during lactation were; 60, 2 and 1 mg per kg body-weight. Body-weight changes, food and fluid intakes were measured throughout lactation. Milk volume was measured between the 12th and 13th day of lactation by a modification of the method of Rath & Thenen (1979). Pups were removed 16 h prior to milking under diethyl ether anaesthesia on the 14th day of lactation.

	Control (n 12)		Caffeine (n 12)		Theophylline (n 11)		Theobromine (n 11)		Mixture (n 11)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Dam weight change (g/10 d lactation)	7		-1		13		4		12	
Pup wt (g) at day 13	21.5	0.69	24.5**	0.74	22.1	0.65	23.6*	0.57	21.6	1.17
Food intake (g/dam per d)	35.1	1.38	40.0	1.56	33.8	0.47	35.8	0.79	37.0	1.65
Fluid intake (ml/dam per d)	46.3	1.61	61.4**	2.95	50.0	3.07	44.5	2.23	55.0**	2.06
Milk composition (g/l):										
Protein	80.0	3.3	93.0	5.1	88.0	6.6	88.0	6.0	84.0	5.3
Amino acid N	0.59	0.06	0.58	0.04	0.62	0.06	0.57	0.07	0.80	0.12
Lactose	16.1	1.27	20.8*	1.75	23.0*	2.73	24.7**	2.22	15.7	1.36
Water	755	12	733	9	774	14	733	12	723	12
Milk intake (ml/pup per d)	5.7	0.5	6.7	0.5	5.6	0.3	6.5	0.3	6.0	0.5

Significant differences from controls: * $P < 0.05$, ** $P < 0.01$.

While the methylxanthines had no effect on litter size or pup weight at 3 d, caffeine and theobromine stimulated milk volume and led to an increase in pup growth. Part of the increased growth was due to more concentrated milks. Not all milk constituents were affected to the same extent. All three methylxanthines increased lactose concentration, theobromine producing the largest effect. There was no significant increase in total protein, although the caffeine group had the highest mean value.

When the methylxanthine mixture was given, interactions prevented the effects seen in the separate drug treatments with the exception of a stimulation of fluid intake.

Rath, E. A. & Thenen, S. N. (1979). *Journal of Nutrition* 109, 840-847.

The influence of dietary pectin on the uterus in mice and rats. By ELERI JONES and R. E. HUGHES, *Department of Applied Biology, UWIST, Cathays Park, Cardiff*

Recent interest in dietary fibre has resulted in a widespread acceptance of its role as an important dietary component (National Advisory Committee on Nutrition Education, 1983). Pectin is one of the most effective of the purified dietary fibres tested in humans for their ability to lower plasma cholesterol (Kay & Truswell, 1980). We have studied, in four separate experiments, the influence of pectin on the development of the uterus in mice and rats.

Mice (age 5 weeks) and rats (age 4 weeks) were used. Control groups received the following synthetic diet (g/kg): maize starch, 570; sucrose, 60; glucose, 60; casein, 130; gluten, 60; maize oil, 60; salt mixture, 57; vitamin mixture, 3. The test groups received the control diet to which pectin (Pectin USP; H.P. Bulmer Ltd, Hereford) had been added to the required concentration (usually 160 g/kg).

The experiment continued for 5–7 weeks. There were no significant differences between the food intakes of the control and test groups. In both mice and rats, in all experiments, the weight of the uterus (both absolute and relative) was significantly reduced in the pectin groups ($P < 0.001$); the weights of the testes, adrenals, liver and kidneys were unchanged. Relevant results from one of the experiments are given in the table (5-week test with eight animals in each group).

Dietary pectin (g/kg) . . .	0		160	
	Mean	SE	Mean	SE
Testes (g)	2.94	0.8	3.08	1.0
Testes (% body-wt)	0.91	0.3	1.0	0.3
Uterus (g)	0.556	0.037	0.259***	0.027
Uterus (% body-wt)	0.263	0.015	0.135***	0.012
Plasma cholesterol (mg/l)	600	34	397***	46

Significantly different from control: *** $P < 0.001$.

Plasma cholesterol was significantly reduced in the pectin groups.

Cholesterol is a biochemical precursor of oestrogenic hormones. A biochemical link between fibre and oestrogen production is theoretically quite plausible. It has yet to be determined whether other types of dietary fibre similarly influence uterine development. These results, if applicable to humans, could be of considerable significance as a dietary pectin content of 160 g/kg (dry weight) approaches that used to lower blood cholesterol in humans (Kay & Truswell, 1980).

Kay, R. Mc. & Truswell, S. (1980). In *Medical Aspects of Dietary Fibre*, pp 67–74 [G. A. Spiller and R. Mc. Kay, editors]. New York and London: Plenum Press.
National Advisory Committee on Nutrition Education (1983). *Lancet* ii, 835.

Menstrual cycle hormonal changes and energy expenditure. By JOANNA T. BISDEE, *MRC Dunn Clinical Nutrition Centre, Addenbrookes Hospital, Cambridge CB2 1QU* and W. P. T. JAMES, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

The well recognized changes in body temperature during the menstrual cycle may depend in part on changes in heat production but published evidence on basal metabolic rate is limited and conflicting. Whether specific sex hormones account for altered rates of total energy expenditure (EE) in humans is also unclear. This study aimed to monitor both hormonal and EE changes in young menstruating women who were maintained on a constant diet to exclude the effects of altered food intake.

Nine normal weight non-smoking women, aged 19–32 years, were studied first at home for over 2 months to establish their usual basal body temperature and food intake, and then during a 6 week residential study in the Dunn Centre where they received a constant diet. They underwent repeated 36 h whole-body calorimetry. Measurements of daily salivary and urinary hormones established free progesterone levels, preovulatory luteinizing hormone surge, and changes in the ratio, oestrone-3-glucuronide:pregnanediol-3 α -glucuronide. All subjects showed an increase in sleeping metabolic rate (SMR) from preovulatory to premenstrual stages of the cycle with mean differences of 340 (SD 80) kJ/24 h (6% of preovulatory levels). Energy expenditures for early and late follicular and luteal stages were 137.5, 135.5, 141.6, 143.3 (SMR) and 256.7, 254.7, 259.6, 260.9 (24 h) kJ fat-free mass. Analysis of variance showed the change in SMR to be significant ($P < 0.001$). Twenty-four-hour measurements showed greater variability. Maximum EE coincided with peak urinary pregnanediol-3 α -glucuronide and salivary progesterone levels but there was no clear relationship between the increase in progesterone and the extent of SMR or EE changes.

The early increase in body temperature in the luteal phase did not coincide with the later rise in SMR. This suggests that vasomotor changes are also important in determining the increase in body temperature in the luteal phase of the menstrual cycle.

A study on energetic efficiency during pregnancy in mice. By D. RICHARD and P. TRAYHURN, *MRC Dunn Nutrition Laboratory, Milton Road, Cambridge CB4 1XJ*

Rats and mice increase their energy intake during pregnancy and this is presumed to be an important factor in meeting the energy costs of reproduction. Evidence for an increase in energetic efficiency has recently been reported in pregnant rats fed *ad lib.* (Naismith & Brookes, 1983) but there is little information on the overall energetics of pregnant animals restricted to a 'normal' energy intake. In the present study we have investigated the energetic efficiency both of pregnant mice pair-fed to the *ad lib.* energy intake of unmated mice, and of pregnant animals given unrestricted access to food.

Mice of the 'Aston' variety, aged 80–90 d and weighing approximately 29 g, were divided into three groups of similar mean body-weight. Two groups were mated and the third served as unmated controls. The start of pregnancy was dated from the appearance of vaginal plugs. One group of pregnant animals was allowed to feed unrestrictedly, while the other was pair-fed to the *ad lib.* intake of the unmated controls. Food intake was measured throughout the study and faeces were collected. After day 19 of pregnancy the energy content of all three groups of mice was determined with an adiabatic bomb calorimeter (mothers and fetuses plus placentas were treated separately), together with the energy content of the food and faeces. The initial energy content of the experimental groups was estimated by reference to a base-line group of mice of similar age and weight range.

	Controls (n 13)		Pregnant pair-fed* (n 8)		Pregnant <i>ad lib.</i> * (n 7)	
	Mean	SE	Mean	SE	Mean	SE
Body-wt gain (g)	1.8	0.6	14.2	1.9	21.3	0.9
Digestible energy intake (kJ)	1257	42	1244	22	1459	30
Energy gain (kJ)	34	19	8	18	134	16
Gross efficiency (%)	2.5	1.5	0.5	1.4	9.2	1.1

*Includes fetuses and placentas.

The table shows that although the weight gain of the pair-fed pregnant animals was considerable, it was less than that of the pregnant *ad lib.* group. There was a substantial difference in energy gain and gross efficiency between the two pregnant groups, both indices being almost zero in the pair-fed mice. Only the *ad lib.* pregnant animals showed increases in energy gain and gross efficiency compared with the controls. Interestingly, only half of the extra energy intake of the pregnant mice fed *ad lib.* was deposited. The difference in energy gain between the *ad lib.* and pair-fed pregnant groups was entirely accounted for by differences in maternal energy stores; there was no difference in the number of fetuses or their energy density.

It is concluded from the present study that there is no increase in gross energetic efficiency in pregnant mice restricted to the normal energy intake of non-pregnant animals.

D.R. is in receipt of a Fellowship from the Medical Research Council of Canada.

Naismith, D. J. & Brookes, R. H. (1983). *Proceedings of the Nutrition Society* 42, 79A.

Changes in milk fatty acid and soluble citrate concentrations caused by short-term feeding of oil to dairy cows. By W. BANKS, J. L. CLAPPERTON, D. D. MUIR and ANNE K. GIRDLER, *Hannah Research Institute, Ayr KA6 5HL*

The heat stability of milk protein is related to the soluble citrate content of the milk (Morrisey *et al.* 1981) which, in turn, is related to the short-chain fatty acid content of the milk (Faulkner & Clapperton, 1981). The short-chain fatty acid content may be modified by adding oils or fats to the diet of the cow. Citrate content varies widely during lactation and, to try to avoid these changes, short-term experiments lasting 3 weeks in all were carried out with fat being given in the second week.

In each of eight experiments there were groups of four cows. In weeks 1 and 3 of each experiment the animals were offered a diet of hay, sugar-beet pulp and concentrates and, in week 2, 500 g/d of either soya-bean oil or beef tallow were substituted isoenergetically for part of the concentrates. The milk of all the cows in each group was collected in bulk at the end of each weekly period, the total fat content and the fatty acid composition were determined on the whole milk and the citrate content on the skimmed milk.

Week	Soya-bean oil				Tallow			
	FAT (g/l)	SCFA (g/l)	CIT (mmol/l)	CT MAX (min)	FAT (g/l)	SCFA (g/l)	CIT (mmol/l)	CT MAX (min)
Early lactation								
1	40.5	8.96	7.29	27.6	44.0	9.05	8.77	13.7
2	39.1	5.84	9.14	27.9	41.9	6.59	8.97	15.6
3	39.5	9.30	6.95	29.9	42.7	10.78	7.81	18.6
Late lactation								
1	41.6	10.63	7.39	13.6	44.9	12.37	8.88	13.4
2	41.3	8.40	9.33	13.4	51.5	11.22	10.47	12.0
3	39.7	10.23	7.56	14.5	46.6	12.87	8.36	13.8

FAT, milk fat; SCFA, short-chain fatty acids; CIT, soluble citrate; CT MAX, maximum coagulation time.

The results are shown in the table. There were no significant changes in the total concentration of fat in the milk but there were large changes in both short-chain fatty acid and citrate concentrations and these latter correlated well. It is suggested that such experiments can be used to investigate changes in minor milk constituents. Despite these changes, there were no significant changes in the heat stability of the milk protein of concentrated milk as measured by the coagulation time.

Faulkner, A. & Clapperton, J. L. (1981). *Comparative Biochemistry and Physiology* **68A**, 291-293.

Morrisey, P. A., Murphy, M. F., Hearn, C. M. & Fox, P. F. (1981). *Journal of Food Science and Technology* **5**, 117-127.

A comparative study on the influence of high-fat diets on thermogenesis in brown adipose tissue of cold-acclimated rodents. By S. W. MERCER and P. TRAYHURN, *MRC Dunn Nutrition Laboratory, Milton Road, Cambridge CB4 1XJ*

Reports in the literature suggest that high-fat diets may enhance cold tolerance (LeBlanc, 1957; Kuroshima *et al.* 1977). In view of the importance now attributed to brown adipose tissue (BAT) in non-shivering thermogenesis in small mammals, the effect of feeding high-fat diets on thermogenesis in the tissue has been investigated using three species of laboratory rodent. The species studied were rats, mice and a hibernator, the golden hamster.

Adult male rats (Dunn hooded strain), mice (C57B1 10ScSn strain) and golden hamsters (MB strain) were each caged individually and acclimated at 4° for 21 d, during which time they were given either a stock low-fat (34 g/kg) diet or a high-fat diet containing 200 g maize oil/kg. BAT, protein content, cytochrome oxidase (*EC* 1.9.3.1) activity and mitochondrial purine nucleotide (GDP) binding were measured as described previously (Trayhurn, *et al.* 1982).

High-fat feeding had little effect on the weight, protein content or cytochrome oxidase activity of BAT in any of the three species. However, as shown in the table, there was a marked increase in mitochondrial GDP binding on the high-fat diet in both rats and mice; the hamsters failed to show this increase. The augmentation of BAT thermogenesis in cold-acclimated rats and mice on the high-fat diet implied by the GDP-binding studies was subsequently confirmed by measurements of mitochondrial respiration and swelling. Further studies on cold-acclimated mice revealed a similar response to a diet high in saturated and monosaturated fatty acids (200 g beef tallow/kg).

Mitochondrial GDP binding (pmol/mg mitochondrial protein)

	Stock diet		Maize-oil diet	
	Mean	SE	Mean	SE
Mice	622	34	866 ^{***}	39
Rats	911	35	1279 ^{***}	78
Hamsters	1093	67	1010	73

n 5-13; ^{***}*P* < 0.001 compared with stock diet.

It is concluded that the maximum activity of the mitochondrial proton conductance pathway in BAT can be increased by dietary fat in rats and mice, though not in hamsters. The effect is independent of energy intake, and not an overfeeding response, since the digestible energy intake was not significantly different on the high-fat and low-fat diets. We suggest that the interspecific difference in the response to dietary lipid may relate to differences in the ATP requirement for lipogenesis, hamsters having much lower rates of fatty acid synthesis in BAT when given a high-carbohydrate/low-fat diet than rats and mice (Trayhurn, 1981).

S.W.M. holds a Research Studentship funded by Unilever plc.

Kuroshima, A., Katsuhiko, D. & Yahata, T. (1977). *Canadian Journal of Physiology and Pharmacology* **55**, 943-950.

LeBlanc, J. C. (1957). *Canadian Journal of Biochemistry and Physiology* **35**, 25-30.

Trayhurn, P. (1981). *Biochimica et Biophysica Acta* **664**, 549-560.

Trayhurn, P., Douglas, J. B. & McGuckin, M. M. (1982). *Nature* **298**, 59-60.

Energy balance and thermogenesis in young male and female rats given a cafeteria diet. By N. J. ROTHWELL and M. J. STOCK, *Department of Physiology, St George's Hospital Medical School, Tooting, London SW17 0RE*

Young male rats exhibit diet-induced thermogenesis (DIT) when energy intake is increased by feeding a palatable cafeteria diet, and this helps to offset the development of obesity (Rothwell & Stock, 1982). In the present study, we investigated the effects of feeding a cafeteria diet on energy balance and brown adipose tissue (BAT) activity in young (37-d-old) female rats (initial weight 95 g) and compared these to the effects in male rats (115 g) of the same age.

Compared to their respective stock-fed controls, cafeteria feeding stimulated metabolizable energy intake by 405 kJ/kg body-weight ($W^{0.75}$) per d (41%) in male and 435 kJ/ $W^{0.75}$ per d (47%) in female rats over the 14 d experiment. This hyperphagia caused only slight and non-significant increases in body-weight in both groups but significant ($P < 0.001$) increases in body energy gain (male 130, female 147 kJ/ $W^{0.75}$ per d, equivalent to 47 and 67% of respective control values). Energy expenditure, calculated from energy intake and body energy gain, was significantly ($P < 0.001$) elevated by 255 kJ/ $W^{0.75}$ per d (36%) and 290 kJ/ $W^{0.75}$ per d (41%) in male and female cafeteria-fed rats respectively. Only 49 and 56 kJ/ $W^{0.75}$ per d (i.e. 19%) of the extra heat production could be accounted for in terms of increased maintenance and the extra cost of energy deposition in male and female cafeteria-fed rats. The residual excess heat production was equivalent to approximately 50% of maintenance heat production and indicates a similar capacity for DIT in both sexes.

Other indices of thermogenic capacity were also increased by cafeteria feeding. Injection of noradrenaline stimulated resting oxygen consumption in all rats, but the effect was much greater in the cafeteria-fed groups (% increase: male control 62 (SE 4), cafeteria 85 (SE 3), $P < 0.001$; female control 45 (SE 5), cafeteria 85 (SE 3), $P < 0.001$). Cafeteria feeding significantly ($P < 0.001$) increased interscapular BAT mass by 349 mg (154%) and 237 mg (126%) and mitochondrial protein by 1.17 mg (70%) and 0.58 mg (32%) in male and female cafeteria-fed rats respectively. The activity of the mitochondrial proton conductance pathway, assessed from purine nucleotide (GDP) binding, was significantly enhanced in male cafeteria-fed rats (86 (SE 10) pmol GDP/mg protein) compared with controls (49 (SE 6), $P < 0.01$), but was not significantly altered by diet in the females (control 69 (SE 7); cafeteria 93 (SE 8), NS). However, total GDP-binding capacity (specific binding \times mitochondrial protein) was significantly elevated by cafeteria feeding in both male (166 pmol/mg protein (202%) increase, $P < 0.001$) and female (100 pmol/mg protein (81%) increase, $P < 0.01$) cafeteria-fed rats. In terms of BAT function, these biochemical indices suggest that female rats are somewhat less responsive to cafeteria feeding than males.

Rothwell, N. J. & Stock, M. J. (1982). *British Journal of Nutrition* 47, 461-471.

Changes in mitochondrial uncoupling protein and GDP-binding in brown adipose tissue of cafeteria-fed rats. By M. ASHWELL¹, N. J. ROTHWELL², D. STIRLING¹, M. J. STOCK² and P. D. WINTER², ¹MRC Dunn Nutrition Unit, Milton Road, Cambridge CB4 1XJ and ²Department of Physiology, St George's Hospital Medical School, Tooting, London SW17 0RE

Both diet-induced and non-shivering thermogenesis (DIT and NST) are associated with increases in brown adipose tissue (BAT) heat production. BAT mitochondria exhibit uncoupled oxidative phosphorylation and high proton conductance due to the presence of a 32 000 MW uncoupling protein (UCP). Purine nucleotides (e.g. GDP) bind to this protein, and GDP-binding capacity and the concentration of UCP increase in rodents exhibiting NST (see Ashwell *et al.* (1983) and Rothwell & Stock (1984) for review). We and others have demonstrated increases in BAT mitochondrial GDP-binding capacity in cafeteria-fed rats exhibiting DIT. However, Himms-Hagen *et al.* (1981) reported increases in GDP-binding without increases in UCP (determined from SDS-polyacrylamide gels) in cafeteria-fed rats. In the present study, we report increases in the amount of UCP detected by radioimmunoassay using a specific antiserum against the protein (Lean *et al.* 1983).

Young (80 g) male rats were given a control pelleted diet or a cafeteria diet for 15–16 d. The mass, mitochondrial GDP-binding and concentration of UCP were assessed in interscapular BAT.

Diet	BAT mass (mg)		Mitochondrial GDP-binding (pmol/mg protein)		UCP (µg/mg protein)	
	Mean	SEM	Mean	SEM	Mean	SEM
	Control (n 7)	250	13	64	11	20
Cafeteria (n 6)	445 ^{***}	19	121 [*]	21	34 ^{***}	3

* $P < 0.05$, *** $P < 0.0001$.

The results presented in the table show marked hypertrophy of BAT in cafeteria-fed rats and significant increases in specific GDP-binding (89%) and UCP (70%). These results confirm those from previous studies on GDP-binding in cafeteria-fed rats exhibiting DIT and show that increases in binding capacity are accompanied by proportional changes in UCP. The failure to detect changes in UCP using SDS-gel electrophoresis may be due to the poor sensitivity of this method. The current study provides further confirmation of the similarities between DIT and NST.

Ashwell, M., Jennings, G., Richard, D., Stirling, D. M. & Trayhurn, P. (1983). *FEBS Letters* **161**, 108–112.

Himms-Hagen, J., Triandafillou, J. & Gwilliam, C. (1981). *American Journal of Physiology* **241**, E116–E120.

Lean, M. E. J., Branch, W. J., James, W. P. T., Jennings, G. & Ashwell, M. (1983). *Bioscience Reports* **3**, 61–71.

Rothwell, N. J. & Stock, M. J. (1984). In *Recent Advances in Physiology*, vol. 10, pp. 349–384 [P. F. Baker, editor]. Edinburgh: Churchill Livingstone.

Carbohydrate-induced thermogenesis in man. By JOANNA T. BISDEE, *MRC Dunn Clinical Nutrition Centre, Addenbrookes Hospital, Cambridge CB2 1QE* and W. P. T. JAMES, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

Whole-body calorimetry permits analysis of the thermogenic response to both substituting carbohydrate (CHO) for fat in a diet and the effects of overfeeding CHO. Eight males aged 20–28 years were given a controlled diet with a series of faecal markers for 6 weeks in the Dunn Centre, and total urine and faecal output were collected throughout the study. Diets were changed every 2 weeks, and two 36 h calorimetry runs with controlled activity were made in each period. Twenty-four hour energy expenditure (EE), sleeping metabolic rate (SMR) and basal metabolic rate (BMR) were assessed separately in relation to the measured metabolizable energy intake. The first two diets were isoenergetic, designed to maintain body-weight, and contained 30 and 45% of energy as fat (protein constant). After 4 weeks, energy intake was raised by 50% with equal parts starch and sucrose. Changing to the high-CHO isoenergetic diet led to a -2.73 (SD 4.96)% change in BMR, -0.69 (SD 2.68)% change in SMR, and a 0.24 (SD 3.60)% change in EE; none of these effects were significant. On CHO overfeeding, EE increased but with little evidence of a progressive rise after the first week. EE rose by 9.7 (SD 2.4)%, SMR by 13.1 (SD 3.1)% and BMR by 10.3 (SD 6.4)%. Expressed as % extra energy given, dietary thermogenesis was 18.2 (SD 4.0)% but varied from 13.8 to 26.1 %. This response from a 5.5 – 7 MJ/d increase in energy intake was similar to that found by Schutz *et al.* (1982) (dietary thermogenesis of 27%) but our absolute increases with the smaller food increments were less impressive.

Earlier studies with lean men given similar energy increases but derived from fat (Dalloso & James, 1984), led to EE increases of 9–11% of the incremental energy intakes. Thus absolute increases in EE are greater on CHO- than fat-overfeeding, but while the energy cost of CHO to fat conversion could account for this effect, the thermogenic response to fat exceeds the biochemical cost of storage. Thus there seems little need to invoke regulatory dietary thermogenesis in explaining the metabolic response to selective increases in CHO intake.

Dalloso, H. M. & W. P. T. James (1984). *British Journal of Nutrition* **52**, 49–64.

Schutz, Y., Acheson, K., Bessard, T. & Jequier, E. (1982). *Clinical Nutrition* **1**, Supplement, 75 (abstract F100).

Erythrocyte (Na⁺-K⁺)-ATPase (EC 3.6.1.3) in normal man: relationship with indices of body composition, energy expenditure and racial origin. By MOUSA NUMAN AHMAD and ANTHONY RICHARD LEEDS, *Department of Nutrition, Queen Elizabeth College, University of London, London W8 7AH*

The membrane-bound (Na⁺-K⁺)-ATPase (sodium- and potassium-ion-activated adenosine triphosphatase; ATP phosphohydrolase, EC 3.6.1.3) is the enzymic expression of the ubiquitous 'sodium pump'. It is estimated that 5-50% of basal cellular thermogenesis is used to drive it. Recently, the pump has received a lot of attention regarding its possible relationship to obesity, but the available evidence is still controversial (Beutler *et al.* 1983).

To investigate the status of the erythrocyte Na pump, (Na⁺-K⁺)-ATPase activity was measured in erythrocytes from thirty-one normal healthy individuals (fourteen men and seventeen women), between 22 and 54 years of age. The (Na⁺-K⁺)-ATPase activity was estimated directly by ATP hydrolysis by purified membranes and indirectly by ⁸⁶Rb-uptake into intact cells. A linear relationship ($r+0.917$, $P<0.001$) was found between the two techniques. The relationships between (Na⁺-K⁺)-ATPase activity and various physical and certain biochemical characteristics of the individuals in the study are presented in the table.

Variable	ATP hydrolysis			⁸⁶ Rb-uptake		
	n	Correlation coefficient	P	n	Correlation coefficient	P
Body mass index (kg/m ²)	31	-0.483	<0.01	20	-0.447	<0.05
Body-wt as % ideal	31	-0.509	<0.01	20	-0.458	<0.05
Metabolic body size (kg ^{0.75})	31	-0.468	<0.01	20	-0.469	<0.05
Blood pressure (mm Hg)	31	-0.580	<0.001	20	-0.687	<0.001
Fasting blood glucose (mmol/l)	24	-0.393	<0.05	18	-0.548	<0.02
Total serum thyroxine (nmol/l)	24	+0.655	<0.001	18	+0.632	<0.01
Erythrocyte [Na ⁺] (mmol/l)	31	-0.797	<0.001	20	-0.898	<0.001
Erythrocyte [Na ⁺]:[K ⁺]	31	-0.850	<0.001	20	-0.890	<0.001

In six individuals, fasting (Na⁺-K⁺)-ATPase activity responded significantly ($P<0.001$) to a meal. The enzyme activity also showed highly significant correlations with the measured basal metabolic rate per kg body-weight ($r+0.951$, $P<0.001$) and sedentary metabolic rate per kg body-weight ($r+0.828$, $P<0.001$) in eight individuals. In the case of twenty-six closely matched individuals (thirteen whites and thirteen non-whites), the (Na⁺-K⁺)-ATPase activity showed a marked difference ($P<0.001$) between the two racial groups. Age did not show a significant correlation with the Na pump activity whereas marked sex variation was observed.

The results confirm the hypotheses that relationships exist between the Na pump activity and body-weight indices, nutrition, thyroid hormone, blood glucose and blood pressure and provide the first report demonstrating in man the associations of the Na pump activity with basal energy expenditure, energy intake and racial origin. The results suggest that the human Na pump should not be studied in isolation but in a whole-body context. Therefore, a greater understanding of the status of the Na pump in obesity could be achieved by careful matching between patients and controls.

Beutler, E., Kuhl, W. & Sacks, P. (1983). *New England Journal of Medicine* 309, 756-760.

Obesity in children—a single state or a set of conditions? By M. GRIFFITHS, E. HOINVILLE, J. P. W. RIVERS and P. T. FOX, *Department of Human Nutrition, London School of Hygiene and Tropical Medicine, London WC1E 7HT*

Anthropometric measurements usually provide two types of definition of obesity, one using various indices of weight and height to measure heaviness and the other based on fatness, using measurements such as skinfold thickness. This presents a problem in diagnosing obesity in children.

In a population of children such as the one described by Tanner & Whitehouse (1975) the triceps skinfold measurement increases, decreases and then rises again between the ages of 1 and 11 years. At about 16 years it increases a third time. A similar pattern can be seen in other skinfold measurements. However, indices of weight/height do not fluctuate in this way so a variation in the relation of fatness and heaviness in growing children is to be expected.

The present study investigated this relationship in boys over the age range 1–8 years with special reference to 4-year-old boys. The data analysed were drawn from the National Pre-School Child Growth Survey (Fox *et al.* 1981).

In the study population the frequency distributions of the triceps skinfolds showed progressively greater positive showing with age; that of weight/height did not. It was possible to detect at any age sub-populations who were heavy, who were fat and who were both heavy and fat. With increasing age the heavy tended also to be fat. However, a second population existed at all ages who were fat but not heavy. The table shows these groups of 4-year-old boys using the 90th percentile of triceps skinfold thickness as the measure of fatness and above the 90th percentile for weight/height to measure heaviness.

Number of 4-year-old boys above and below the 90th percentile for triceps skinfold thickness and weight/height

Wt/height	Triceps skinfold thickness		Total
	Below 90th percentile	Above 90th percentile	
Below 90th percentile	813	54	867
Above 90th percentile	62	16	78
Total	875	70	945

These results have implications for the diagnosis of obesity. For example, if heaviness is used as a predictor of fatness, there are 79% false positives and 6% false negatives. Perhaps more importantly, the results suggest that there may be no single state identifiable as obesity in children, rather that we are identifying different sets of conditions which may relate to the individual's pattern of energy storage in growth. This possibility and its implications will be discussed.

- Fox, P. T., Elston, M. D. & Waterlow, J. C. (1981). In *Sub-committee on Nutritional Surveillance (Committee on Medical Aspects of Food Policy), Second Report*. DHSS report on health and social subjects, no. 21, pp. 64–82. London: H.M. Stationery Office.
- Tanner, J. M. & Whitehouse, R. H. (1975). *Archives of Diseases in Childhood* 50, 142–145.

Acceptability of sucrose in the diabetic diet. By M. E. J. LEAN, *MRC Dunn Clinical Nutrition Centre, Addenbrookes Hospital, Cambridge CB2 1QE* and B. R. TENNISON and D. R. R. WILLIAMS, *School of Clinical Medicine, University of Cambridge, Cambridge* (Introduced by P. TRAYHURN)

Recent dietary recommendations for diabetics encourage increased intakes of complex carbohydrates and dietary fibre: sucrose is still regarded an anathema (British Diabetic Association, 1982). Dietary compliance is often poor and inflexible, restrictive advice is a contributory factor (Thomas, 1981). This study was designed to examine the acute effect on blood glucose of different amounts of sucrose and cereal fibre taken by C-peptide negative, insulin-dependent diabetics. Breakfast was chosen since it is usually after this meal that it is most difficult to control blood glucose. Twelve normal-weight subjects on twice-daily rapid- and intermediate-acting insulins and a high-carbohydrate (55% energy) diet, received four breakfasts on consecutive days in a Latin square design. Total dietary energy for each subject and percentages of energy derived from carbohydrates (55%) and fats (32%) were kept constant. Carbohydrate sources were (1) wholemeal bread, (2) white bread, (3) marmalade made with sucrose, (4) marmalade (22% total energy) on wholemeal bread.

Using two-way analysis of variance to allow for differences between subjects, no significant differences ($P < 0.05$) were found between the meals in peak elevation of blood glucose, nor in the area under the incremental blood glucose curve from 0 to 120 min. These results were not dependent on the fasting blood glucose of the individual.

Carbohydrate source . . .	1		2		3		4	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Peak elevation of blood glucose above fasting levels (mmol/l)	4.4	0.7	3.0	0.7	5.1	0.7	4.7	0.7
Upper 99% confidence limits	6.2		4.7		6.9		6.5	
Area under incremental blood glucose curve from 0 to 120 min (mmol/l per h)	5.1	1.3	2.3	1.3	4.6	1.3	5.4	1.3
Upper 99% confidence limits	8.4		5.6		7.9		8.6	

The results of the present study demonstrate that sucrose taken as marmalade does not produce a significant elevation of postprandial blood glucose in insulin-dependent diabetics. The lack of difference between wholemeal and white bread supports earlier studies (Simpson *et al.* 1979).

Occasional sucrose consumption is not incompatible with a high-carbohydrate, high-fibre diet. Indeed, consumption of fruit is encouraged and fruit provides most of its available energy as free sugars which have a glycaemic index close to that of sucrose (Jenkins *et al.* 1981). A more relaxed approach towards sugar-eating by diabetics might improve dietary compliance.

British Diabetic Association (1982). *Human Nutrition: Applied Nutrition* 36A, 378-394.

Jenkins, D. J. A., Wolever, T. M. S., Taylor, R. H., Barker, H., Fielden, H., Baldwin, J. M., Bowling, A. C., Newman, H. C., Jenkins, A. L. & Goff, D. V. (1981). *American Journal of Clinical Nutrition* 34, 362-366.

Simpson, R. W., Mann, J. I., Eaton, J., Carter, R. D. & Hockaday, T. D. R. (1979). *British Medical Journal* ii, 523-525.

Thomas, B. (1981). *Human Nutrition and Diabetes*, pp. 57-66 [M. R. Turner and B. J. Thomas, editors]. London: John Libbey & Co.

Epidemiological assessment of sodium sources in the diet by the use of the lithium-marker technique. By CLAUDIA P. SANCHEZ-CASTILLO, *MRC Dunn Clinical Nutrition Centre, Old Addenbrooke's Hospital, Cambridge CB2 1QU*, S. WARRENDER and T. WHITEHEAD, *Wolfson Research Laboratories, Queen Elizabeth Medical Centre, Birmingham* and W. P. T. JAMES, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

A lithium-labelled salt has been developed and tested for use in tracking the proportion of sodium in the diet derived from the tagged source and full metabolic studies have validated the approach (Sanchez-Castillo & James, 1983, 1984). The technique has therefore been applied to fifty-one randomly-sampled adult men and women from a single practice in Cambridgeshire, and thirty-two spouses all studied in May and June 1983. Subjects collected 12-d consecutive 24 h urine samples, cooking salt (CS) and table salt (TS) being provided throughout but tagged with Li from days 3–9 inclusive. Individual salt cellars were weighed, six blood pressures were taken at intervals by trained nurses with random zero sphygmomanometers, and a single observer (S-C) undertook anthropometry. Urinary electrolytes and Li were measured and the amounts of total discretionary salt added were calculated from the known Li content of the salt and the Li urinary output above baseline excretion. From the weekly weight of TS lost from individual containers, CS intake was estimated.

Results were calculated for the 7 d when Li-labelled salt was used, allowance being made for the Li excreted beyond the 7-d period. The Na excretion of adults from the same household proved to be similar so single members of each household (n 54) were analysed initially. Chloride excretion averaged 147.1 (SEM 6.5) mmol/d, the non-chloride form of Na providing an additional output of 4.5 (SEM 1.0) mmol/d. Based simply on chloride excretion and assuming that the TS used was completely ingested, TS provided 6.5 (SEM 0.9) % of salt intake, CS 5.1 (SEM 0.8) % and non-discretionary sources, i.e. from food and drink, 88.4 (SEM 1.2) %. If CS ingested is calculated by difference from Li-based NaCl intakes minus weighed TS used, then 29 (SEM 2) % of the CS used in cooking was ingested in food, the rest being discarded in cooking water. Thus a very large proportion of Na intake in this community is derived from non-discretionary sources.

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Sanchez-Castillo, C. P. & James, W. P. T. (1983). *Proceedings of the Nutrition Society* **42**, 168A.

Sanchez-Castillo, C. P. & James, W. P. T. (1984). *Proceedings of the Nutrition Society* **43**, 154A.

Estimating dietary sources of sodium with lithium-tagged salt. By CLAUDIA P. SANCHEZ-CASTILLO, *MRC Dunn Clinical Nutrition Centre, Old Addenbroke's Hospital, Cambridge CB2 1QE* and W. P. T. JAMES, *Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB*

A technique has been developed for fusing lithium carbonate with sodium chloride to produce a salt which when added to cooking water penetrates vegetables with Li and sodium in the molar ratios present in the water. Li recoveries from chronic oral doses of Li_2CO_3 amounted to 95% (Sanchez-Castillo & James, 1983). A metabolic study was therefore undertaken with five male volunteers for 41 d with complete collection of faeces, urine and sweat for defined intervals during which either the cooking salt was tagged with Li or Li-labelled table salt was provided *ad lib*. This permitted the validation of Li as a tracer for Na absorption and excretion by the three routes. Li and Na uptake by vegetables was also assessed.

Recoveries of Na and Li under full balance conditions were (mean and SD) 96 (4) % Na, 96 (2.9) % Li for Li-labelled table salt and 99 (4.4) % Na, 99 (4.4) % Li for Li-labelled cooking salt. The excretion of Na by urine, faeces and sweat amounted to 95.1 (SD 1.3), 1.5 (SD 1.5) and 2.1 (SD 0.5) % of the total excreted respectively. Li from cooking salt was excreted as follows: 97.1 (SD 0.9) % via urine, 1.1 (SD 0.5) % in faeces and 1.6 (SD 0.8) % in sweat. Similar recoveries were found in the Li-tagged table salt periods. Recoveries and excretion routes of Li were again unaffected by absolute Na intake or by the penetration of Li into cooked foods.

The cooking experiments confirmed the proportional entry of the two cations into a variety of foods. The use of the Li-tagged salt with urine monitoring is therefore valid for use in epidemiological studies.

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Sanchez-Castillo, C. P. & James, W. P. T. (1983). *Proceedings of the Nutrition Society* 42, 168A.

The nutritive value of school and hospital meals. By H. HERFST, *University of Wageningen, The Netherlands*, M. NELSON, *MRC Environmental Epidemiology Unit, Southampton General Hospital, Southampton SO9 4XY* and M. WOOLAWAY, *Wessex Regional Health Authority, Winchester*

Nutritionists often suggest that catering in schools and hospitals should exemplify good eating habits. The dietary goals proposed by the National Advisory Committee on Nutrition Education (NACNE) (1983) represent 'a consensus of views primarily derived from Government reports and from other major bodies of experts' and are useful standards of 'good eating habits'. Do meals served to schoolchildren and hospital patients reach these standards?

Eight Southampton schools provided menus for all school meals served during six 1-week periods in 1982–83. The meals were served to 4176 primary, 4601 junior and a similar but unspecified number of secondary schoolchildren. In-patients at the Queen Alexandra Hospital in Portsmouth completed menu cards each day to indicate their food choices for the three meals served. Seven thousand records of food choices were available from men, women and children in hospital during seven 1-week periods in 1982–83. With information from the schools and hospital about average portion sizes, the nutrient content of the meals was calculated using food composition tables (Paul & Southgate, 1978). All calculations took into account preparation and cooking losses but not serving or plate waste.

	Schools (per meal)			Hospital wards (per day)				NACNE goals
	First	Middle	Secondary	Children	Men	Women	Male geriatric	
Energy (MJ)	2.19	2.68	—	6.95	7.10	6.68	8.52	—
Percentage energy from:								
Protein	14	13	11	16	16	16	16	11
Fat	42	43	37	45	45	44	45	31
Carbohydrate	44	44	52	39	39	40	39	58
Dietary fibre (g)	5.3	7.5	—	13.7	14.6	13.9	18.3	—
(g/MJ)	2.4	2.8	2.2	2.0	2.1	2.1	2.1	3.0

The table shows total energy per person per meal (in schools) or per day (in hospital), the percentage of energy derived from protein, fat and carbohydrate, and amounts of dietary fibre per meal or per day and per MJ, together with the NACNE recommendations for a low-fat, high-fibre diet. The percentage of energy derived from fat was in every instance greater than the NACNE goal and, with the exception of secondary schools, greater than the average percentage of fat in the local diet (M. Nelson and C. Power, unpublished results). Dietary fibre intakes per MJ were considerably below the NACNE goal. In this study, meals provided by schools and hospitals do not match the NACNE recommendations for a healthy diet.

National Advisory Committee on Nutrition Education (1983). *Proposals for Nutritional Guidelines for Health Education in Britain*. London: Health Education Council.
 Paul, A. A. & Southgate, D. A. T. (1978). *McCance and Widdowson's The Composition of Foods*, 4th ed. London: H.M. Stationery Office.