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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

The Four Hundred and Twenty-eighth Meeting of the Nutrition Society was held in the Morris Lecture Theatre, Robin Brook Centre, St Bartholomew's Hospital, West Smithfield, London on Tuesday and Wednesday, 22/23 *July* 1986, *when the following papers were read:*

https://doi.org/10.1079/PNS19870019 Published online by Cambridge University Press

Vitamin A and synthetic retinoids induce gastrointestinal proliferation and gastric hyperkeratosis; similarities in the action of retinoids and epidermal growth factor. By J. TURTON, S. KEEFE, R. M. HICKS and J. GWYNNE, *School of Pathology, Middlesex Hospital Medical School, London W1P 7LD*

Vitamin A and synthetic retinoids stimulate bone resorption (Turton *et al.* 1985) and are cytoprotective in the gastrointestinal (gi) tract (Turton *et al.* 1986); one retinoid, 13-*cis*-N-ethylretinamide, is carcinogenic for the mouse liver (Hicks & Turton, 1986). Epidermal growth factor (EGF) also stimulates bone resorption, is cytoprotective, and may promote tumourigenesis (Rose *et al.* 1976). We have therefore investigated whether retinoids induce gi proliferation, as reported for EGF (Goodlad *et al.* 1986).

Female BALB/c mice (seventy and seventy-one at 3- and 8-weeks-old respectively) were fed for 5 weeks on a control diet or one containing vitamin A acetate (VAA), 13-*cis*-retinoic acid (13CRA) or N-(4-hydroxyphenyl)retinamide (4HPR) at levels producing hypervitaminosis A: each retinoid induced body and skeletal muscle weight loss, and femoral thinning. Stomach surface area and weight, small intestine length and weight, and caecum and colon weight were recorded and tissues taken for histology. All retinoids produced a significant increase in the relative weight but not the length of the small intestine. There were no consistent effects on caecum or colon weight. In all mice, stomach surface area was increased by each retinoid, the largest increase being in VAA-fed animals (35·3% in 3- and 20·9% in 8-week-old mice, $P<0.01$). Relative weights of the non-glandular and glandular stomach areas showed that only the weight of the non-glandular region was increased; this was significant with all retinoids in 8-week-old mice ($P<0.001$). Histological examination showed that each retinoid produced hyperkeratosis of the non-glandular stomach, with 13CRA and VAA producing pronounced effects. Considering the anti-keratinizing activity of retinoids in other organ systems, this observation was unexpected.

Weights of parts of the gi tract reflect cell population sizes and therefore increases in weight and surface area indicated that VAA, 13CRA and 4HPR induced gi proliferation. This response, together with bone resorption, cytoprotection and tumour promotion suggest similarities in action between retinoids and EGF. The retinoid-induced gastric hyperkeratosis seen in these experiments provides a further parallel, for EGF is reported to promote keratin formation (Carpenter & Cohen, 1979). As retinoids increase the number of cellular EGF receptor sites, the results presented here suggest that retinoid effects may be mediated by potentiating the action of EGF on susceptible tissues.

Carpenter, G. & Cohen, S. (1979). *Annual Review of Biochemistry* **48**, 193–216.
Goodlad, R. A., Wilson, T. J. G., Lenton, W., Gregory, H., McCullagh, K. G. & Wright, N. (1986). *Gut* (In the Press).
Hicks, R. M. & Turton, J. (1986). *Biochemical Society Transactions* (In the Press).
Rose, S. P., Stahn, R., Passovoy, D. S. & Herschman, H. (1976). *Experientia* **32**, 913–915.
Turton, J., Hicks, R. M., Gwynne, J., Hunt, R. & Hawkey, C. (1985). In *Retinoids, Differentiation and Disease*, pp. 220–251 [M. B. Sporn, chairman], Ciba Foundation Symposium no. 113. Pitman: London.
Turton, J., Rode, J., Hicks, R. M. & Gwynne, J. (1986). *Journal of Pathology* **148**, 87A.

Regulation of muscle protein metabolism in man as measured by the forearm model. By K. N. CHENG, P. J. PACY, F. DWORZAK, G. C. FORD, S. HUNTER and D. HALLIDAY, *Nutrition Research Group, Clinical Research Centre, Harrow HA1 3UJ*

Stable isotopes are increasingly used to evaluate protein metabolism. However, a major problem has been the inability to measure protein breakdown directly. Whole body protein breakdown is derived from flux in the fasted state in the stochastic model. Muscle protein breakdown has been estimated from 3-methylhistidine urinary excretion but its validity is in question, especially in disease. We have recently developed a model to study protein turnover in humans based on metabolite balance across the forearm (Cheng *et al.* 1985). The aim of the present study was to compare results in fed and fasted states in healthy volunteers using this model.

Simultaneous deep venous and 'arterialized' blood samples were collected before and at 10-min intervals 2·5 h after a primed, continuous infusion of L-[1-^{13}C,^{15}N]leucine. Double-labelled leucine enables deamination and reamination rates and their ratio, transamination, to be measured. Concentrations and isotopic enrichment of plasma leucine, α-ketoisocaproate (KIC) and carbon dioxide were measured by established techniques (Ford *et al.* 1985); forearm blood flow was measured by strain-gauge plethysmography. Forearm muscle metabolism was studied during continuous feeding (protein intake 0·095 g/kg per h) and after a 12–14 h overnight fast. A summary of results (nmol/litre tissue per min) is shown in the Table.

	Protein synthesis		Protein breakdown		Net protein balance		Trans-amination ratio		KIC oxidation	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Fasted (*n* 12)	700	60	980	80	−290	50	1·06	0·006	56	9
Fed (*n* 6)	1270*	110	870	100	+390*	90	1·18*	0·01	430*	40

*$P<0.001$ by unpaired *t* test.

During feeding, each subject was in positive protein balance, while protein balance was negative during fasting. This difference appeared to be principally due to decreased protein synthesis. Transamination was lower during fasting and the values recorded in this study are similar to those previously obtained from whole body studies (Matthews *et al.* 1981). These results are sufficiently encouraging to suggest a role for this model in future studies on muscle protein metabolism.

Cheng, K. N., Dworzak, F., Ford, G. C., Rennie, M. J. & Halliday, D. (1985). *European Journal of Clinical Investigation* **15**, 349–354.

Ford, G. C., Cheng, K. N. & Halliday, D. (1985). *Biochemical Mass Spectrometry* **12**, 432–436.

Matthews, D. E., Bier, D. M., Rennie, M. J., Edwards, R. H. T., Halliday, D., Millward, D. J. & Clugston, G. A. (1981). *Science* **214**, 1129–1131.

The effect of aerobic exercise on subsequent 24 h resting metabolic rate in normal male subjects. By P. J. PACY, J. D. WEBSTER, G. ISAACS, S. HUNTER and J. S. GARROW, *Nutrition Research Group, Clinical Research Centre, Harrow HA1 3UJ*

It has been suggested that the true energy cost of physical exercise may be twice the energy expended during the exercise period, since resting metabolic rate is increased for many hours after the exercise has finished (Hermansen *et al.* 1984). However, measurement of energy expenditure by indirect calorimetry for 100 min after a single exercise period (Freedman-Akabas *et al.* 1985), or for 340 min during which four periods of exercise were undertaken (Pacy *et al.* 1985), have failed to show a significant increase in metabolic rate after exercise. Since direct calorimetry can be used to measure energy expenditure with a reproducibility of 1–2% over a period of 24 h, we have used a direct calorimeter (Webster *et al.* 1986) to measure total 24 h loss in three normal male subjects with and without exercise.

The subjects were on a weight-maintaining diet, and were measured in the calorimeter on 2 d without exercise and 2 d in which they exercised on a bicycle ergometer at approximately 30% maximum oxygen uptake for 1 h, rested for 2 h, and then again exercised for 1 h. The sequence of exercise and control days for each subject was rotated according to a Latin square design. The total heat loss (MJ/24 h) for each subject on each occasion is shown in the Table; the energy cost incurred during the exercise periods has been subtracted from the total heat loss on the 'exercise' days, so the figures compare 24 h resting metabolism on days with and without exercise.

Total heat loss (MJ/24 h) in three normal male subjects with exercise (Ex) or resting (Rest). The immediate energy cost of the exercise has been subtracted from the total on Ex days

Subject	Ex 1	Ex 2	Rest	Rest
A	6·49	7·67	7·06	6·99
B	5·75	5·54	6·31	5·28
C	9·81	8·55	7·33	8·51

The mean difference in energy expenditure between exercise and rest days was 0·4 MJ (96 kcal): this difference is not statistically significant on analysis of variance. These results confirm that the level of exercise which can be achieved by normal subjects has no measurable effect on resting metabolism over 24 h.

Freedman-Akabas, S., Colt, E., Kissileff, H. R. & Pi-Sunyer, F. X. (1985). *American Journal of Clinical Nutrition* **41**, 545–549.

Hermansen, L., Grandmontagne, M., Moehlum, S. & Ingnes, I. (1984). *Medicine and Sports Science* **17**, 119–129.

Pacy, P. J., Barton, N., Webster, J. D. & Garrow, J. S. (1985). *American Journal of Clinical Nutrition* **42**, 764–768.

Webster, J. D., Welsh, G., Pacy, P. J. & Garrow, J. S. (1986). *British Journal of Nutrition* **55**, 1–6.

Influence of a mackerel diet on plasma lipoproteins and platelet function.

By T. A. B. SANDERS, L. LOVAT, M. MISTRY, KARINA UPTON, *King's College London (KQC), Campden Hill Road, London W8 7AH* and D. R. SULLIVAN, *Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, Ducane Road, London W12 0HS*

It has been proposed that the consumption of oily fish may decrease the risk of coronary heart disease by favourably altering plasma lipids, platelet function and thrombotic tendency (Dyerberg *et al.* 1978). Von Lossonczy *et al.* (1978) showed a marked reduction in plasma triglyceride but a tendency for low-density lipoprotein (LDL) and high-density lipoprotein (HDL) concentrations to increase with 200 g mackerel/d. We report the effects on plasma lipoproteins and platelet function of a diet containing 200 g mackerel/d taken for 2 weeks in ten healthy young men. The results are expressed as mean values with their standard errors.

	Control		Week 1		Week 2	
	Mean	SE	Mean	SE	Mean	SE
Plasma cholesterol (mmol/l)	4·03	0·289	4·08	0·328	4·16	0·264
Plasma HDL cholesterol (mmol/l)	1·21	0·065	1·34	0·116	1·42*	0·121
Plasma triglycerides (mmol/l)	1·15	0·097	0·55**	0·075	0·61**	0·054
LDL apoprotein B (mg/l)	980	45			1090**	41
Platelet 20:5ω3 (wt %)	0·7	0·15	3·0**	0·17	3·1**	0·22
Platelet 20:4ω6 (wt %)	21·5	0·53	18·8**	0·50	17·6**	0·69
Platelet thromboxane B_2 ($ng/10^8$ platelets)	59	3·8	41**	3·4	41**	3·5
Platelet β-TG ($ng/10^6$ platelets)	81	3·8	62**	4·2	60**	4·1
Platelet diameter (μm)	2·56	0·03	2·64**	0·03	2·70**	0·03

Significantly different from control: *$P<0.05$, **$P<0.01$.

The mackerel diet increased HDL cholesterol and reduced plasma triglyceride concentrations. We believe the observed increase in LDL apoprotein B can be explained by a decrease in very-low-density lipoprotein particle size which would hasten its conversion into LDL. There was rapid incorporation of eicosapentaenoic acid (20:5ω3) into the platelet lipids, mainly at the expense of arachidonic acid (20:4ω6), and this was accompanied by a reduction in the capacity to produce thromboxane B_2. Platelet aggregation induced by 1 μg collagen/ml was decreased after 1 week of the diet but returned to baseline values after 2 weeks. Platelet size and intraplatelet β-thromboglobulin (β-TG) were increased and decreased respectively by the fish diet. These changes may reflect changes in platelet synthesis or catabolism.

Dyerberg, J., Bang, H. O., Stofferson, E., Moncada, V. & Vane, J. R. (1978). *Lancet* **i**, 117–119.

Von Lossonczy, T. O., Ruiter, A., Bronsgeest-Schoute, H. C., van Gent, C. M. & Hermus, R. J. J. (1978). *American Journal of Clinical Nutrition* **31**, 1340–1346.

The percentage contribution of foods to the nutrient composition of diets of schoolchildren and adults. By C. A. HURREN and L. STOCKLEY, *AFRC Institute of Food Research, Colney Lane, Norwich NR4 7UA*

Since the changes in the types of school meals provided, following the 1980 Education Act, there has been concern expressed about the choice of foods now available to children, and the effect on their nutritional status.

Schoolchildren (*n* 22), aged 9–11 years, kept 3-d weighed intake records. Mean daily nutrient intakes of these children were compared with daily intakes of seventeen adults aged 21–48 years, who had previously kept a 7-d weighed intake record. Nutrient intake was calculated using food tables (Paul & Southgate, 1978) together with additional data (Wiles *et al.* 1980; MRC Dunn Nutrition Unit, unpublished data). A computer program was written to rank the sources of energy, fat and fibre intake for both the children and the adults as a percentage of total intake. The results are shown in the Table.

% of total energy intake			
Children		Adults	
Bread	11·8	Bread	13·2
Chocolate biscuits	5·8	Milk	7·0
Milk	5·8	Sugar	4·1
Crisps	5·7	Margarine	3·3
Low-fibre breakfast cereal	4·4	Cheese	3·0
Chocolate	4·0	Chocolate biscuits	2·9
Margarine	3·7	Plain biscuits	2·9

% of total fat intake			
Children		Adults	
Margarine	9·9	Milk	9·7
Crisps	9·4	Margarine	8·8
Milk	7·5	Butter	7·5
Chocolate biscuits	7·5	Cheese	6·7
Beef	4·9	Beef	4·1
Chocolate	4·6	Sausages	3·7
Cheese	4·1	Chocolate biscuits	3·6

% of total fibre intake			
Children		Adults	
Wholemeal bread	18·6	Wholemeal bread	24·9
Crisps	11·7	Muesli	4·1
White bread	6·7	Brown bread	4·0
Weetabix	6·5	Weetabix	3·6
Apples	5·4	Baked beans	3·2
Baked beans	4·7	White bread	2·9
Chocolate biscuits	3·3	Potatoes	2·9

The mean daily intake of dietary fibre by the children was 17·9 g and 36·8% of energy was provided by fat. This compares with 25·9 g of dietary fibre and 37·7% of energy from fat for the adults.

For the children, crisps and chocolate biscuits were a major contributor of energy, fat and fibre. Together they provide 11·5% energy, 16·9% fat and 15% dietary fibre. These foods were eaten almost exclusively as part of their packed lunch at school. Any decrease in their intake of high-fat foods in their main meals was offset by their large consumption of snack foods at mid-day.

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

Wiles, S. J., Nettleton, P. A., Black, A. E. & Paul, A. A. (1980). *Journal of Human Nutrition* **34**, 189–223.

https://doi.org/10.1079/PNS19870019 Published online by Cambridge University Press

Nutrient and food intakes in Caucasian and Gujarati men in North London. By L. STOCKLEY and A. J. BROADHURST, *AFRC Institute of Food Research, Colney Lane, Norwich NR4 7UA,* and S. KOTECHA, *MRC Epidemiology Unit, Northwick Park Hospital, Middlesex HA1 3UJ*

In 1984–5 a study was carried out in North London to investigate the relation between habitual fat intake and various indices. In the course of this study, dietary records were collected from two different ethnic groups.

Men aged between 45 and 54 years were identified on the age/sex register of a health centre in Wembley. Twenty of Caucasian and twenty of Gujarati origin were selected by random sampling. They were asked to keep a weighed food diary, with precise weighing of recipe dishes, for five consecutive days (Wednesday–Sunday). Nutrient intakes were calculated using values from Paul & Southgate (1978) and other sources. Two records from the Gujarati group were incomplete, and were excluded from the analysis. Five of the eighteen in this group were non-insulin dependent diabetics, and fourteen were vegetarians.

The intake of thirty-nine nutrients was calculated, and mean daily intakes of some of these are listed in the Table. Diabetics were excluded from the analysis. The mean body-weights of the Caucasians and Gujaratis were 81 and 69 kg respectively.

	Caucasian		Gujarati excluding diabetics	
	Mean	SD	Mean	SD
Energy (kJ)	11223	1506	9972	3280
(kcal)	2677	360	2376	784
Protein (g)	94·0	13	70·7**	29
Fat (g)	115·7	28	101·9	32
Sugars (g)	133·9	46	83·6**	29
Dietary fibre (g)	23·1	8	26·3	11
Calcium (mg)	1109	305	936	460
Iron (mg)	15·1	5	15·4	6
Vitamin B_{12} (μg)	6·4	3	1·7***	0·9
Polyunsaturated:saturated fats	0·26	—	0·46	—

Significantly different from the Caucasian group: **$P<0.01$, ***$P<0.001$.

The major sources of all of the nutrients were identified. For example, bread, fat spread, milk, sugar, cheese and potatoes accounted for 38% of the energy in the Caucasian diet. Chapatis, milk, ghee, rice and savoury snacks accounted for a similar percentage in the Gujaratis. White sugar added to foods or drinks in the home formed 25–30% of the total sugar intake in both groups. However, the absolute intake in the Caucasians was higher, with 37% of this coming from drinks, preserves, desserts and confectionery. The Na intake in this group was also higher, and the main contributor was bread (29%).

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

Diet and ischaemic heart disease in the community: the Caerphilly study.

By ANN M. FEHILY, J. W. G. YARNELL and BARBARA BUTLAND, *MRC Epidemiology Unit, 4 Richmond Road, Cardiff CF2 3AS*

In a South Wales Community Study (Anon., 1986) 2512 middle-aged men were screened for evidence of ischaemic heart disease (IHD) using the London School of Hygiene Chest Pain Questionnaire (Rose & Blackburn, 1968) and a 12-lead electrocardiograph (ECG). Nutrient intakes were calculated from the questionnaire (*n* 2423) (Yarnell *et al.* 1983) and 7-d weighed intake records (*n* 665) (Fehily *et al.* 1984).

Those who had symptoms of IHD (15%) may have altered their diet after the onset of the disease. Those who had ECG evidence of ischaemia but no symptoms were at increased risk of IHD (Rose *et al.* 1976) but were less likely to have altered their diet. Therefore nutrient intakes of those with ECG ischaemia only were compared with those of men in whom there was no evidence of IHD. Differences between the two groups were very small, those with ECG ischaemia tending to have lower intakes of energy (2–5%), alcohol (3–18%) and of several other nutrients. Total fat intake was 2–7% lower, starch 2% lower, sugars 3% lower, protein 2% lower, dietary fibre 2–6% lower, calcium 5% lower and magnesium 4% lower. When standardized for energy intake, these differences became even smaller. Allowing for the effects of differences between the groups in the distributions of age and body mass index did not alter these conclusions.

Anon. (1986). *The Caerphilly Collaborative Heart Disease Studies. Project Description and Manual of Operations.* ISBN 09508951 13.

Fehily, A. M., Phillips, K. M. & Sweetnam, P. M. (1984). *Human Nutrition: Applied Nutrition* **38A**, 270–276.

Rose, G., Baxter, P. J., Reidd, D. D. & McCartney, P. (1976). *British Heart Journal* **40**, 636–643.

Rose, G. & Blackburn, H. (1968). *Cardiovascular Survey Methods. World Health Organization Monograph Series* no. 56. Geneva: WHO.

Yarnell, J. W. G., Fehily, A. M., Milbank, J. E., Sweetnam, P. M. & Walker, C. L. (1983). *Human Nutrition: Applied Nutrition* **37A**, 103–112.

Six year compliance with dietary advice in cardiac patients. By V. REID and R. MULCAHY, *Cardiac Department and Department of Preventive Cardiology, St Vincent's Hospital, Dublin 4, Irish Republic*

There is continuing debate about the practicality of implementing healthy eating guidelines, especially with regard to the acceptability of a diet with a lower fat content (Marr & Morris, 1982). We have already reported that cardiac patients can change their diets in accordance with advice over a 1 year period (Reid *et al.* 1984). Our present interest is whether these improvements in dietary intake can be maintained in the long-term.

Thirty-eight post-myocardial infarction patients were included in a long-term diet study. Twenty-three (60%) of these were available for interview at 6 year follow-up. Fifteen patients were not interviewed: five had died, one was living overseas, one had an illness preventing interview and eight did not keep their appointments.

We have observed no significant changes in nutrient intake between the 1st and 6th year of follow-up in these patients. Serum cholesterol levels had reduced to 5·4 mmol/l but body-weight had returned to initial levels (mean 77 kg).

Seventeen of the twenty-three patients in our study were deemed to be good compliers and six were poor compliers at the 1st year follow-up. Good compliers tended to maintain the 1st year nutrient intakes at 6 years, although body-weight had increased from 72 to 74 kg. Poor compliers continued to have difficulty in achieving the recommended nutrient intakes. They had a high alcohol intake (12% of energy) masking a high proportion of energy from fat. Weight had increased from 81 to 85 kg but serum cholesterol levels had reduced from 5·9 to 5·6 mmol/l.

Mean daily intakes at 6 year follow-up

	Recommended	Total group	Good compliers	Poor compliers
Fat (% of energy)	30–35	38	37	39
Cholesterol (mg/d)	<300	282	273	308
Polyunsaturated:saturated fat	>0·75	0·75	0·91	0·28
Fibre (g/d)	20–30	22	24	18

Patient compliance largely determines changes in dietary habits. Eleven of the seventeen good compliers continued to improve their nutrient intakes over the 6 years, achieving most of the recommended objectives.

Marr, J. & Morris, J. C. (1982). *Lancet* **i**, 217–218.
Reid, V., Graham, I., Hickey, N. & Mulcahy, R. (1984). *Human Nutrition: Applied Nutrition* **38A**, 279–287.

Effect of dietary saturated and polyunsaturated fats on faecal excretion of cholesterol and its catabolites in rabbits. By N. JADIDI, J. D. EDWARDS-WEBB, R. W. OWEN and M. I. GURR, *Department of Food Quality and Human Nutrition, AFRC Institute of Food Research, Reading Laboratory, Shinfield, Reading RG2 9AT*

Diets rich in polyunsaturated fatty acids lower serum cholesterol concentrations compared with diets rich in saturated fatty acids. One of the suggested mechanisms of this cholesterol-lowering effect of polyunsaturated fatty acids has been an increased catabolism of cholesterol.

In the present study two groups of nine rabbits were given diets containing 200 g butter/kg or 200 g polyunsaturated margarine/kg for a period of 8 weeks. The fat content of each diet provided 50% of the total energy. The cholesterol content of the margarine was adjusted to that of the butter (2 g/kg). Food intakes were measured throughout the experiment. For 4 d before the animals were killed, faeces were collected and pooled over 24-h periods. The faeces were kept for analysis of neutral and acidic sterols by gas liquid chromatography–mass spectroscopy.

At the end of the 8 week period the plasma cholesterol concentrations of the margarine group were significantly lower than that of the butter group (696 (SEM 71·4) *v.* 1403 (SEM 169·2) mg/l; $P<0.01$).

Faecal sterol balance

	Butter		Margarine		Statistical significance (Student's *t* test)
	Mean	SEM	Mean	SEM	
Excretion of total sterols* (mg/d)	60·0	5·83	86·9	10·96	NS
Excretion of synthesized total sterols† (mg/d)	28·1	4·89	46·5	9·73	NS
Total synthesized sterol excretion† (mg/d) / Total plasma cholesterol pool size‡ (mg)	19·8	4·31	57·6	11·63	$P<0.01$

NS, not significant.

*Total sterols = neutral sterols + acidic sterols.

†Mean total sterol excretion – mean dietary cholesterol intake.

‡Calculated from body-weight, packed cell volume and plasma cholesterol concentration of each animal.

Although the daily excretion of synthesized sterols in the margarine group was higher than that in the butter group, it failed to be statistically significant due to large individual variation. However, when expressed as a ratio of their total plasma cholesterol pool size the differences were highly significant ($P<0.01$). These results suggest that dietary polyunsaturated fats, compared with saturated fats, do increase excretion of total sterols and that this increase over a period of time could be the major factor contributing to the plasma cholesterol-lowering effect of polyunsaturated fatty acids.

Response of rat caecal metabolism to varying proportions of white and wholemeal bread. By FIONA B. KEY and J. C. MATHERS, *Department of Agricultural Biochemistry and Nutrition, The University, Newcastle upon Tyne NE1 7RU*

Epidemiological studies have suggested that dietary fibre (DF), especially that from cereals, may protect against colorectal cancer (Bingham *et al.* 1979) but the mechanism(s) of any protection is unknown. We have investigated the response in caecal metabolism of rats given graded levels of DF as white and wholemeal bread.

Four groups of six male Wistar rats (initial weight 230 g) were housed individually in metabolism cages and offered diets in which freeze-dried bread provided 90% of the air-dry matter, together with casein, vitamins, minerals, sucrose and maize oil. The diets consisted of white bread (W), 2:1 white bread:wholemeal bread (WB), 2:1 wholemeal bread:white bread (BW) and wholemeal bread (B). After 21 d, the rats were killed and the caeca removed. The pH of caecal contents were measured immediately and samples of caecal contents were taken for volatile fatty acid (VFA) determinations.

	Diet				SE of	Significance of dietary effects		
	W	WB	BW	B	Mean	Lin	Quad	Dev
pH	6·1	6·2	6·1	6·1	0·10	NS	NS	NS
Total VFA (mmol/kg)	87	83	85	80	5·3	NS	NS	NS
Proportions of individual VFA (mmol/mol):								
Acetic acid	659	602	554	526	19·6	***	NS	NS
Propionic acid	161	171	152	166	6·5	NS	NS	*
Isobutyric acid	6	8	7	10	0·9	*	NS	NS
Butyric acid	153	196	267	274	20·5	***	NS	NS
Isovaleric acid	9	11	9	12	1·0	NS	NS	NS
Valeric acid	12	12	11	12	0·5	NS	NS	NS

Lin, Quad, Dev, linear, quadratic and deviations from linear and quadratic effects of diet respectively.

NS, not significant; $^{*}P<0.05$, $^{***}P<0.001$.

Increasing the proportion of wholemeal bread in the diet had no effect on pH or concentration of total VFA in caecal contents but resulted in a significant linear decrease in acetic acid with a concomitant linear increase in butyric acid. Isobutyric acid was also increased. The 79% increase in butyric acid is especially interesting given the apparent antineoplastic activity of this compound (Prasad, 1980).

F.B.K. holds an AFRC Food Research Studentship.

Bingham, S., Williams, D. R. R., Cole, T. J. & James, W. P. T. (1979). *British Journal of Cancer* **40**, 456–463.

Prasad, K. N. (1980). *Life Sciences* **27**, 1357–1358.

The effect of a dietary supplement of marine oil in healthy volunteers on platelet lipids, platelet function and platelet plasma membrane fluidity. By CAROLINE BOLTON-SMITH and M. J. GIBNEY, *Department of Nutrition, University of Southampton, Southampton SO9 3TU*

Dietary fish oils, rich in n-3 polyunsaturated fatty acids (PUFA), have been extensively studied for their ability to alter platelet function in man and in experimental animals. These oils lead to substantial changes in the fatty acid composition of platelet phospholipids: reduced arachidonic acid (20:4n-6) and increased eicosapentaenoic acid (20:5n-3), docosapentaenoic acid (22:5n-3) and docosahexaenoic acid (22:6n-3). Reduced levels of 20:4n-6 and increased levels of 20:5n-3 will lead to reduced thromboxane A_2 production by platelets and possibly to the production of trienoic thromboxane. However, the simultaneous enrichment with 22:5n-3 and 22:6n-3 could also alter membrane fluidity and change the sensitivity of platelets to aggregating agents.

Eight healthy male volunteers (22–39 years) participated in the study. Each volunteer supplemented their normal diet with 15 g encapsulated fish oil (Maxepa; Seven Seas Health Care, Hull)/d. Blood samples (200 ml) were taken before, during and after a period of 6 weeks of supplementation, with 12-week intervals between each sample (ethical stipulation). A weighed dietary assessment was carried out before and during the supplementary period. Platelets were analysed for their component phospholipid fatty acids, for membrane cholesterol and phospholipids, for membrane fluidity using fluorescence polarization with 1,6-diphenyl-1,3,5-hexatriene (DPH), for ADP- and collagen-induced aggregation and for collagen-induced thromboxane B_2 (TxB_2) production.

There were no significant changes in energy intake or in the percentage energy from fat during the study. Total n-3 PUFA intake (wt%) rose significantly ($P<0.05$) with fish-oil supplementation (mean and SEM: 2·5 (0·5), 6·0 (0·2), 2·1 (0·4)) while total n-6 PUFA intake remained constant (22·6 (3·3), 21·1 (2·5), 14·4 (5·1)).

20:5n-3 increased from trace values to 1·2 (SEM 0·2)% in phosphatidyl choline (PC) and to 3·7 (SEM 1·1)% in phosphatidyl ethanolamine (PE), while 20:4n-6 was significantly reduced ($P<0.05$) from 17·3 (SEM 1.5)% to 7·9 (SEM 1·9)% in PC and from 45·2 (SEM 6·3)% to 30·3 (SEM 5·4)% in PE during the supplementary period. The levels of 22:5n-3 and 22:6n-3 (mean and SEM) increased significantly ($P<0.05$) in PE during supplementation (1·7 (0·1) to 2·8 (0·6)% and 0·6 (0·2) to 3·4 (0·7)% respectively) and were not found in any other phospholipid fractions. Platelet cholesterol:phospholipid ratio (mean and SEM) remained unaltered during the three periods (0·32 (0·05), 0·27 (0·03), 0·34 (0·02)) as did platelet TxB_2 production (11·9 (1·2), 9·8 (0·9), 10·9 (0·6) ng/10^8 cells). Collagen-induced platelet aggregation did not alter during the study while ADP-induced aggregation at 5 and 10 μM (not with 2·5 μM) rose significantly during the supplementation period.

These results show that the expected changes in platelet fatty acids with fish-oil supplementation are not associated with changes in platelet function or platelet membrane fluidity.

The effect of dietary fish oil on immune-complex-induced atherosclerosis in rabbits. By CAROLINE BOLTON-SMITH[1], M. J. GIBNEY[1], P. J. GALLAGHER[2] and K. HILLIER[3], *Departments of [1]Nutrition, [2]Pathology and [3]Clinical Pharmacology, University of Southampton, Southampton SO9 3TU*

Platelet–vessel wall interactions play a central role in the development of atherosclerosis. Thromboxane A_2 (TxA_2) produced by platelets exerts a pro-thrombotic influence which is opposed by prostacyclin (PgI_2) produced by aortic endothelial cells. It is now well established that dietary marine oils, rich in eicosapentaenoic acid (20:5n-3) inhibit normal eicosanoid synthesis, including TxA_2 and PgI_2. The purpose of the present study was to determine whether a dietary fish oil (Maxepa; Seven Seas Health Care, Hull) would, in altering both TxA_2 and PgI_2 production, exert a net anti-atherogenic effect, using serum sickness to induce atherosclerosis in rabbits.

Twenty-four rabbits were weaned onto a semi-purified diet containing coconut oil (20 g/kg) for 14 d (period 1). The rabbits were then assigned (*n* 8) to one of three diets containing 20 g/kg of either maize oil, coconut oil or fish oil for a further 12 weeks (period 2). The levels of the oils were then increased to 60 g/kg for a further 34 weeks (period 3) and were supplemented with low levels of dietary cholesterol (2·5 g/kg) for the remaining 8 weeks (period 4). Each rabbit received 3×10 ml intravenous injections of horse serum at 14 d intervals during period 3, and a further 1×20 ml injection of horse serum during period 4. At 56 weeks the rabbits were killed.

Oil . . .	Coconut		Maize		Fish	
	Mean	SEM	Mean	SEM	Mean	SEM
Serum cholesterol in period 4 (mmol/l)	18·9*	2·2	16·4*	1·3	12·5	2·0
Aorta:						
Phospholipid 20:4n-6 (wt%)	14·7*	2·1	15·2*	2·0	7·6	0·4
Phospholipid 20:5n-3 (wt%)	trace		trace		5·1	—
Arch: 6-keto $PgF_{1\text{-}\alpha}$ (ng/kg per 30 min)	155	15	250*	65	85	5
Distal: 6-keto $PgF_{1\text{-}\alpha}$ (ng/kg per 30 min)	295*	75	275	45	125	20
Total atherosclerosis (% lesioned)	46·1	20·4	60·8*	15·7	19·6	16·0
Platelet:						
Phospholipid 20:4n-6 (wt%)	12·1*	0·6	10·4*	0·7	8·1	0·5
Phospholipid 20:5n-3 (wt%)	trace		trace		11·0	1·4
TxB_2 production (ng/10^8 cells)	9·8	1·4	12·1	1·6	5·2	1·3

20:4n-6, arachidonic acid.
Significantly different from fish-oil treatment: *$P<0.05$.

Serum sickness was confirmed in all rabbits by renal histology. Dietary fish oil led to a significant decrease in the ratio of 20:4 to 20:5 in both platelet and aortic phospholipids and consequently to TxB_2 and 6-keto-$PgF_{1\text{-}\alpha}$ production. Neither circulating total immune complexes nor anti-horse serum IgG were influenced by dietary treatment. The reduced atherosclerosis in fish-oil-fed rabbits is likely due to reduced TxB_2 synthesis although other factors may be involved.

The Kilkenny Health Project: patterns of food intake in individuals consuming low-, moderate- and high-fat diets. By M. J. GIBNEY, *Department of Clinical Medicine, Trinity College Medical School, St James' Hospital, Dublin* 8, *Irish Republic,* MARY MALONEY, *Dublin Institute of Technology, Kevin Street, Dublin* 8, *Irish Republic,* EMER SHELLY, *Kilkenny Health Project, Parnell Street, Kilkenny, Irish Republic*

The Kilkenny Health Project is a 10-year programme of health education for the reduction in risk factors for coronary heart disease. Nine hundred individuals (aged 35–44 years) were randomly selected to participate in a base-line study of risk factors and health beliefs. Of these, thirty men and thirty women were randomly chosen for a dietary survey using the 7 d weighed dietary intake method. The survey was carried out during July–August 1985.

Mean (and SE) daily energy intakes were 12·5 (2·6) MJ (males) and 8·4 (2·2) MJ (females). Dietary fibre intakes were 24·1 (5·9) (males) and 20·4 (8·8) (females) g/d. The respective distribution of energy (%) between protein, carbohydrate, fat and alcohol was 14·7 (2·7), 43·3 (6·9), 36·0 (7·1) and 5·9 (7·7) for men and 15·6 (3·4), 43·8 (5·7), 39·1 (4·8) and 1·2 (2·3) for women. The main sources of fat in the diet were (%): butter 18·7, fresh meat 17, milk 12·4, biscuits and cakes 11·2, margarine 9·2, processed meat 5·8, eggs 5·0 and bread 4·2. When alcoholic beverages were included, fourteen men and seven women had diets with less than 35% energy from fat (low), eight men and thirteen women had diets with fat energy between 35 and 40% (moderate) and eight men and ten women had fat energy >40% (high). When alcoholic beverages were excluded from the calculations, the numbers with low-, moderate- and high-fat diets were respectively 6♂, 5♀; 14♂, 14♀; 10 and 11♀. The mean (and SE) energy (MJ) intakes for these groupings were: 9·7 (3·1), 9·9 (2·8) and 9·8 (2·7), of which fat supplied 33·0, 38·0 and 49·8% respectively and sugar provided 19·1, 18·1 and 12·6% respectively. The mean daily fibre (g) intakes for the low-, moderate- and high-fat groups were 24 (9), 22 (8) and 21 (6) respectively.

When fat energy was classified into low, moderate and high, with alcohol excluded, the respective intakes of foods were as follows (g/d): bread 220 (100), 179 (92) and 152 (61); potatoes 263 (102), 217 (91) and 190 (91); table sugar 58 (49), 49 (44) and 14 (20). This decreasing trend contrasted with an increasing trend in the intake (g/d) of fresh meat 105 (66), 125 (46) and 138 (88); biscuits and cakes 46 (33), 52 (42) and 64 (42); fruit 56 (50), 62 (54) and 85 (74); cheese 5 (7), 9 (11) and 10 (12). Thus, the low-fat eaters were characterized by a higher intake of bread, potatoes and sugar and a lower intake of fresh meat, fruit, vegetables other than potatoes, cheese, biscuits and cakes, and sweets and chocolates. The intakes of milk and spreadable fats did not show any comparable consumption trend. For men, saturated fatty acids (SFA) provided 14·6 (4·3)% of total energy while for women the comparable value was 15·4 (3·6)%. Seventeen men and thirteen women consumed less that 15% of energy from SFA.

These results indicate that dietary goals of 35% energy from fat, 15% energy from SFA and dietary fibre intakes of 25 g/d are feasible. They further show that low-fat diets tend to be high-sugar diets.

Fetal growth in low birth weight Nigerian babies. By M. B. DUGGAN, *Department of Paediatrics, Ahmadu Bello University Hospital, Zaria, Nigeria*

Body size was studied in relation to gestational age in 217 low birth weight (<2·5 kg) babies born in hospital in Zaria, Nigeria. Weight, crown–heel length and head circumference were measured, and gestational age assessed (Farr *et al.* 1966). Measurements on singletons and twins were compared with appropriate reference standards (Usher & McLean, 1969; Wilson, 1974) both graphically and in terms of their standard deviation score related to the reference mean (Waterlow *et al.* 1977).

Ninety-two (42%) babies were male, and 81 (37%) were twins. The antenatal history was known for 129 mothers of whom twenty-one had suffered from pre-eclamptic toxaemia, and ten from anaemia.

With advancing gestational age an increasing negative deviation from reference values for weight, length and head circumference was observed. The significance of these trends was confirmed statistically, by comparison of means and by regression of the standard deviation scores for weight, length and head circumference upon the gestational age. The nutritional status, estimated as the weight:length ratio, at different gestational ages was compared graphically with reference values, and with data from other developing countries.

As a result of combined underweight and stunting, the mean weight:length ratio of Nigerian low birth weight babies remained close to the reference mean values until the last weeks (>36 weeks) of gestation. Furthermore, although the prevalence of significant underweight and stunting increased significantly with advancing gestational age, the prevalence of significant wasting (weight for length less than 90% of the reference value) was similar at all gestational ages.

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Energy expenditure in vaccination, infection and controlled hyperthermia. By R. XIMENES, M. COX and A. M. TOMKINS, *Department of Nutrition, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT,* and K. COLLINS, *Geriatric Research Unit, St Pancras Hospital, London NW1*

Infection is frequently complicated by weight loss as a result of a negative energy balance (Beisel, 1977). The purpose of the present study was to measure the energy expenditure (RME) in well-nourished adults in three febrile states.

The RME was calculated from measurements of oxygen and carbon dioxide collected by continuous suction from an acrylic hood placed over the head and shoulders. There were two groups of subjects. (1) Volunteers were measured before and after a vaccination (monovalent typhoid vaccine, Wellcome Laboratories). Those who developed a pyrexia were subsequently warmed using an air-conditioned heated bed as described by Fox (1969). The temperature of the circulating air was adjusted so that the time course of the rise in body temperature was the same as that experienced during the vaccination. (2) Patients with a variety of infections were measured at the height of the pyrexial phase and measurements were repeated at least 5 d after return of normal temperature.

	Vaccine study				Heated bed study			
Volunteers, reactive	RME at normal temperature (basal)	Increased temperature (°)	RME at increased temperature (°)	% increase in RME	RME at normal temperature (basal)	Increased temperature (°)	RME at increased temperature (°)	% increase in RME
1	1484	38·3	1802	43·4	1484	38·4	1802	21·0
2	1636	37·4	1772	8·3	1630	37·3	1715	5·2
3	1429	37·4	1644	15·1	1412	37·4	1488	5·4
4	1533	38·1	1822	37·3	1533	38·0	1822	18·8
5	1775	37·6	2121	20·0	—	—	—	—

	Infection study			
Patients				
A	1726	40·0	2176	26·0
B	1246	39·3	1601	28·0
C	1696	39·9	2002	18·0
D	1364	37·6	1387	5·8
E	2005	38·7	2437	21·5
F	1527	39·0	1880	23·1
G	1883	37·9	2056	9·2

There was a linear association between RME and body temperature in each of the experimental groups but the value of the regression coefficient differed considerably (0·028 in the vaccinated group, 0·058 in those with controlled hyperthermia and 0·086 in the patients with systemic infection), indicating a greater RME per unit temperature rise in the vaccinated volunteers than the infected patients. We suggest that RME may be considerably greater during the development of pyrexia than during established infection.

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The composition of macrobiotic diets. By ALISON HINDS and PATRICIA A. JUDD, *Department of Food and Nutritional Sciences, King's College (KQC), Campden Hill Road, London W8 7AH*

The 'macrobiotic' diet originally comprised a regimen of seven diets containing increasing proportions of grains (Kushi, 1977). The strictest diet consisting solely of brown rice is clearly inadequate and has led to widespread condemnation of the macrobiotic diet. However, less is known about the adequacy of more moderate macrobiotic diets. Macrobiotic dietary rules discourage the consumption of mammalian and avian meat and fat, sweeteners including sugar, hot spices and any foods artificially flavoured or chemically preserved, and restrict fluid intakes. However, salted foods, such as miso, are used. We have assessed the nutrient intakes of ten subjects (five male, five female) following typical macrobiotic diets, as advocated by the Community Health Foundation (London), using a 7 d weighed food record. Nutrient intakes were calculated by computer and were supplemented by analysis of commonly used macrobiotic foods (Table 1).

Table 1. *Nutrient composition (/kg food)*

Food	Energy (kJ)	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	Fe (mg)	Ca (mg)	Zn (mg)	Na (mg)	K (mg)
Tofu	5270	1260	121	52	1	14	5000	20	740	70
Tempeh	5940	1420	171	39	90	87	1100	80	660	520
Sourdough bread	12760	3050	85	7·5	311	15	190	56	1750	820
Seitan	10460	2500	220	3·4	5	38	180	10	1510	350
Wholemeal zenryu fu	16440	3930	308	17	14	30	290	9	10	930
Umemboshi paste	1210	290	11	2	4	25	42	7	27370	1050
Amazake	7490	1790	35	7	131	3	680	30	120	150
Tofu-burgers	10500	2510	169	91	15	52	450	140	1070	1580
Rice cakes	9410	2250	42	4	682	15	220	70	120	100

The diets contained approximately 500 g cereals/kg and 200–300 g seasonal vegetables/kg. Beans, fermented bean-products and seaweeds formed a significant part of the diet and small amounts of fish and other seafoods were consumed. Energy and protein intakes were adequate, fat intakes tended to be lower (16–36% energy) and fibre intakes were greater (18–45 g/d) than that in the general population. Both iron and calcium daily intakes were high (18–44 mg and 710–1480 mg respectively). Vitamin B_{12} intakes were less than 1 μg/d in nine out of the ten subjects but this excludes any contribution from fermented foods. It is not known whether these foods provide sufficient vitamin B_{12} in the diet. Consequently, those who follow a macrobiotic diet may risk deficiency.

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Defective thermogenic response to mild-cold exposure in type II diabetes and Cushing's disease. By M. E. J. LEAN and P. R. MURGATROYD, *Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge*

In animal models of congenital obesity and diabetes, the development of insulin resistance is associated with impaired thermogenic responsiveness of brown adipose tissue to cold exposure (Mercer & Trayhurn, 1983). The thermogenic defect appears to be partly mediated through elevated circulating corticosteroid concentrations (Holt *et al.* 1983) and has been related aetiologically to impairment of glucose tolerance (Cawthorne *et al.* 1984).

Whole-body indirect calorimetry has been used to study the thermogenic responses to mild-cold of six normal weight control women and eleven overweight women with simple obesity (*n* 4), type II diabetes (*n* 5) or Cushing's disease (*n* 2). Treatment for diabetes was diet plus intermediate-acting insulin; metyrapone therapy for Cushing's disease was witheld for 24 h before study. Subjects entered the calorimeter after a 2·5 MJ meal at 19.00 hours and slept under a single sheet. Core temperature was monitored using an aural thermister probe. Overnight ambient temperatures of 28° and 22° were studied in random order using the sleeping period 04.00 to 07.00 hours for analysis, with comparisons by Wilcoxon's and sign tests.

Sleeping energy expenditures (watts) at 28° (thermoneutral) were: controls 66 (SEM 8), simple obesity 77 (SEM 9), diabetics 78 (SEM 21), Cushing's 61 and 66. The responses of the groups to exposure at 22° are shown with reference to 28°: all controls had increases in energy expenditure, whereas all the diabetic and Cushing's syndrome patients had decreases. The difference from controls was highly significant for the diabetics considered alone (−3·5 (SEM 1·5)%, $P<0.001$). After weight loss following bilateral adrenalectomy, sleeping energy at 22° was increased by 1·4% and 5% in the women with Cushing's disease, the response to mild-cold being normalized in one case. As in Blaza & Garrow (1983), mixed responses were observed in simple obesity.

			Body mass index		Change in core temperature (°)		Change in energy expenditure (% change)	
	n	Age	Mean	SEM	Mean	SEM	Mean	SEM
Controls	6	32	23·1	1·2	−0·3	0·1	+3·8*	1·3
Diabetic/Cushing's overweight	7	50	33·6	2·2	−0·6	0·2	−4·1*	1·2
Simple obesity	4	41	35·8	1·4	−0·4	0·2	−2·0	2·1

*$P<0.05$ for paired analyses within groups.

This thermogenic defect may be important in the aetiology of weight gain in certain subgroups of obesity. Further studies are required to establish the mechanism.

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Abdominal adipose tissue distribution in diabetes, Cushing's syndrome and phaeochromocytoma: a computed tomography index for brown adipose tissue activity? By M. E. J. LEAN, *MRC Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge* and A. K. DIXON, *University Department of Radiology, Addenbrookes Hospital, Hills Road, Cambridge*

Increased abdominal fat deposition, leading to increased waist:hip ratio, is now an established risk-factor for the development of a number of metabolic problems including diabetes and hyperlipidaemia. The mechanism is unclear but the proportion of fat in internal as opposed to subcutaneous sites seems important (Ashwell *et al.* 1985), internal adipose tissue normally being the more metabolically active (e.g. Ostman *et al.* 1979). It is postulated here that variations in lipid accumulation may reflect functional differences which relate to the origins of human internal fat as brown adipose tissue (Heaton, 1972).

A computed X-ray tomography method (Ashwell *et al.* 1985) has been used to study the abdominal adipose tissue distribution in conditions which, from analogy with animal studies, might be expected to show altered lipid accumulation in brown adipose tissue. In control subjects the proportion of internal fat at the umbilical level showed significant correlation ($P<0.001$) with age in both men (mean 38 (SEM 3)% of total fat, n 41, r 0·67) and women (mean 22 (SEM 1)%, n 48, r 0·47), but none with body mass index (r 0·15 and 0·17). Compared with expected results (χ^2), or with age-matched controls (Wilcoxon's test), there was a significantly greater proportion of intra-abdominal lipid in overweight women with type II (maturity onset) diabetes (35 (SEM 3)%, n 13, $P<0.01$) and Cushing's syndrome (40 (SEM 5)%, n 6, $P<0.05$), and a significantly lower proportion in patients of both sexes with the noradrenaline-secreting tumour, phaeochromocytoma (men 27 (SEM 2)%, women 14 (SEM 1)%, n 11, $P<0.01$).

These alterations in abdominal fat distribution might have been predicted if human intra-abdominal fat were considered to behave in the same way as animal brown adipose tissue, as opposed to the subcutaneous white adipose tissue. A proportionally greater increase in the lipid content of brown adipose tissue is associated with decreased thermogenic activity in weight gain from diabetes (Goodbody & Trayhurn, 1981) and hyperadrenocorticism (Galpin *et al.* 1983). Decreased lipid content of brown adipose tissue, in relation to body-weight change, accompanies increased thermogenic activity with phaeochromocytoma in animals (Ricquier *et al.* 1983).

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Long-term effects of fructose consumption on adipose tissue cellularity and glycaemic status in non-insulin dependent diabetic rats. By S. P. DEBOLT[1], N. CARSWELL[2], O. E. MICHAELIS IV[2], and O. L. TULP[1], [1]*Department of Nutrition and Food Sciences, Drexel University, Philadelphia, PA, and* [2]*Beltsville Human Nutrition Research Center, USDA, Beltsville, MD, USA.*

Since dietary sucrose and fructose have been identified as risk factors in the pathogenesis of obesity, type II diabetes (NIDDM) and other diseases (Reiser, 1985), excess consumption of these carbohydrates may have important implications for public health. The interaction of genetic predisposition for obesity+NIDDM and of dietary fructose (F) intake on the expression of these stigmata as they occur in the SHR/N-*cp* rat, a recently developed strain which bears these traits (Michaelis *et al.* 1985) was investigated.

Animals of both lean and corpulent (corp) phenotypes were fed on diets containing (g/kg) 540 carbohydrate as cooked maize starch (MS) or as equal parts MS and F (MSF), 200 protein, and 160 fat, plus vitamins, minerals and non-nutritive fibre from weaning until 9 months of age.

	Glycaemic status (% of lean MS)						Adipocyte cellularity/three sites					
	Final body-wt (g)		Response to glucose	Fasting plasma insulin	Hb A_{1c} (% total Hb)		Depot wt (g)		No. of cells ($\times 10^6$)		Cell size (nl)	
	Mean	SE			Mean	SE	Mean	SE	Mean	SE	Mean	SE
Lean MS	455	8	100	100	20	4	17·5	0·4	43	3	0·36	0·07
Lean MSF	471	7	119	125	22	4	20·2	0·3	45	3	0·37	0·06
Corp MS	820	32	174	1325	24	1	137·9	2·3	94	7	0·57	0·10
Corp MSF	865	36	211	1675	19	5	143·4	2·9	86	6	0·58	0·15

After 12 weeks on the diet, fasting blood glucose concentrations were similar in lean and corp rats fed on either diet, but fasting insulin and triglyceride concentrations and the glucose and insulin responses following an oral glucose tolerance test (2·5 g glucose/kg body-weight) were greater in corp than in lean animals ($P<0·05$), and were greatest in the MSF-fed obese+NIDDM rats, consistent with further deterioration of peripheral insulin sensitivity and glycaemic status. Final body-weights, adipocyte size, number and fat-pad weight of epididymal, retroperitoneal and dorsal fat depots were greater in corp than in lean rats ($P<0·05$). Lipoprotein lipase (*EC* 3.1.1.34) activity per g adipose tissue and final haemoglobin (Hb) A_{1c} values were unaffected by dietary carbohydrate type in either ($P>0·05$). These results indicate that partial substitution of F for MS was neither beneficial nor ameliorative in the expression of diabetic stigmata in an animal model highly predisposed to obesity+NIDDM, and suggest that caution should be used in recommending significant fructose consumption in NIDDM.

Supported by Drexel University and BHNRC–USDA institutional resources.

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Relations between dietary intake and urinary excretion for sodium and potassium. By J. C. MATHERS, J. MCNEILL and SARAH M. WEIR, *Department of Agricultural Biochemistry and Nutrition, The University, Newcastle upon Tyne NE1 7RU*

Measurement of urinary excretion appears to be a promising method of estimating dietary intake of nutrients such as sodium, potassium and nitrogen. We have examined the relations between dietary intake and urinary output for Na and K for normal adults who were consuming their usual diet and again when Na intake was deliberately reduced.

Eleven apparently healthy subjects (six female, five male), aged 20–39 years, made daily collections of their urine for two non-sequential 7 d periods. In Period 1, subjects ate their usual diets, whilst in Period 2 they attempted to reduce sodium chloride intake by avoiding salt-rich foods and by reducing intake of those foods which made major contributions to Na intake in Period 1. Wholemeal bread made without added salt was provided in Period 2. Food intake was assessed by the weighed inventory method and intakes of Na and K were calculated using the Microdiet program (Bassham & Fletcher, 1985). Two subjects only took discretionary salt.

Mean K intakes in Periods 1 and 2 were identical (3·7 and 3·7 (SE 0·18) g/d respectively) but Na intake was significantly ($P<0·001$) reduced from 2·9 in Period 1 to 1·0 (SE 0·14) g/d in Period 2. Using data for individual days, linear relations of the form urinary Na (or K) output (g/d)=A×Na (or K) intake (g/d)+B were calculated and the regression coefficients obtained are given in the Table.

	Na			K		
	Period 1	Period 2	Both periods	Period 1	Period 2	Both periods
A	0·85	0·85	0·85	0·86	0·93	0·90
SE	0·038	0·027	0·018	0·040	0·030	0·027
B	0·12	0·14	0·14	0·13	0·22	0·15
SE	0·048	0·017	0·026	0·047	0·042	0·035
Correlation coefficient	0·93	0·96	0·97	0·93	0·96	0·94
n	77	76	153	77	76	153

There was a close relation between dietary intake and urinary output for Na and K in both periods. For Na, there was no significant ($P>0·05$) difference in this relation between periods but for K, urinary output for a given intake tended to be higher in Period 2 than in Period 1 and parallel curve analysis (Ross, 1980) demonstrated significant ($P<0·001$) displacement in this relation between periods for K.

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Sodium and blood pressure in the hypertensive type II diabetic: randomized blind, controlled and crossover studies of moderate Na restriction and Na supplementation. By P. M. DODSON, M. BEEVERS, R. HALLWORTH, M. J. WEBBERLEY, R. F. FLETCHER and K. G. TAYLOR, *Department of Diabetes and Endocrinology, Dudley Road Hospital, Birmingham B18 7QH*

Reports have suggested that exchangeable body sodium is increased by approximately 10% in diabetic subjects and that this abnormality can be reversed, with a reduction in blood pressure, by diuretic agents. We have therefore performed a randomized blind controlled study to assess the hypotensive response to moderate Na restriction for 3 months in hypertensive type II diabetic subjects (Na restricted, *n* 17, mean age 61·9 (SE 7·5) years, percentage ideal body-weight 126·7 (SE 20·2)) and in a matched control group. Following this, a randomized double-blind crossover of slow Na supplementation (80 mmol/d) *v.* placebo for 1 month was completed in nine Na-restricted patients.

After 3 months, the Na-restricted group demonstrated significant reductions in systolic blood pressure (supine 179·7 (SE 18·2) to 160·5 (SE 5·6) mmHg, $P<0·001$, erect 182·3 (SE 20·1) to 160·9 (SE 15·8) mmHg, $P<0·001$) and daily urinary Na excretion (198·7 (SE 65·9) to 136·8 (SE 38) mmol/d, $P<0·001$), but no changes occurred in weight, diabetic control or diastolic pressure. No significant changes occurred in the control group (systolic blood pressure (mmHg): supine 173·8 to 167·5; erect 175·9 to 166·5). Following this period, supine systolic blood pressure significantly increased on slow Na supplementation compared with placebo (placebo, 161·8 (SE 17·7); slow Na, 171 (SE 17·1) mm Hg; $P<0·005$).

We conclude that moderate Na restriction may have a significant hypotensive effect and that the dietary recommendation of moderate reduction of salt intake would seem appropriate for hypertensive type II diabetic patients.

The effects of haemo-dialysis on salt and sweet taste. By R. SHEPHERD and C. A. FARLEIGH, *AFRC Institute of Food Research, Norwich NR4 7UA*, and C. ATKINSON, *Leeds Polytechnic, Leeds LS1 3HE* and J. S. PRYOR, *Norfolk and Norwich Hospital, Norwich NR1 3SR*

Changes have been reported in the sensitivity and preference for salt taste when patients undergo dialysis (Shepherd *et al.* 1985, 1986). In the present study, fifteen patients took part, along with fourteen matched controls. Fifty-eight people donating blood took part in a parallel study, in order to determine whether taste changes might simply be a function of a reduction in blood volume during dialysis. The patients and controls were tested on six occasions. They tasted six samples of soup varying in salt concentration both before and after dialysis, on each of three occasions; they tasted six samples of apple purée varying in sucrose concentration on three separate occasions. The blood donors took part in one session each, tasting either soup (*n* 28) or apple (*n* 30) before and after giving blood. The samples were rated on a seven-category intensity scale, giving a measure of sensitivity, and on a 100 mm relative-to-ideal rating scale, which gives a measure of the individual's most preferred (or ideal) concentration.

For the dialysis patients, ratings on both scales were higher after than before dialysis for the salt in soup (see Table), but not for the sucrose in apple. The ratings were plotted against log concentration, and regression lines fitted. The slopes of the salt functions were higher after dialysis (not significantly for the intensity ratings) and the ideal concentration was lower following dialysis. Neither effect was found for the sucrose. No effects were found for either the control subjects or the blood donors, and neither of these groups differed significantly overall from the patients.

	Patients			Controls			Blood donors		
	Pre-dialysis	Post-dialysis	SED	Pre-interval	Post-interval	SED	Pre-blood donation	Post-blood donation	SED
Relative-to-ideal ratings:									
Average rating	−5·2	−0·3*	1·8	−4·1	−2·0	1·5	−0·9	1·6	1·6
Slope of function	28·7	32·9*	1·6	33·9	33·9	1·8	28·2	28·8	1·8
Log_e (ideal)	1·20	0·98*	0·10	1·11	1·06	0·04	1·01	1·01	0·07
'Ideal' (g Na/kg)	3·3	2·7	–	3·1	2·9	–	2·7	2·7	–
Intensity ratings:									
Average rating	2·74	2·99*	0·09	2·28	2·39	0·11	2·53	2·64	0·11
Slope of function	1·38	1·61†	0·11	1·85	1·88	0·13	1·83	1·89	0·15

Values significantly different from pre-dialysis values: *$P<0.05$, †$P<0.1$

These results confirm previous findings of increased sensitivity and decreased preference for salt on dialysis. This effect did not generalize to sweet taste. The effect does not appear to be related simply to changes in blood volume, but presumably to more complex changes in blood biochemistry on dialysis.

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The effects of glucose feeding during starvation on rat jejunal secretion.

By A. YOUNG and R. J. LEVIN, *Department of Physiology, University of Sheffield S10 2TN*

A recognized feature of famine is severe diarrhoea. We have shown that a 72 h fast greatly increases rat jejunal secretion in vivo and in vitro in response to secretagogue challenge (Levin & Young, 1985, 1986). However, the effects of specific dietary constituents upon intestinal secretion have not been investigated.

Male rats (230–250 g) were starved for 72 h. One group was allowed to drink water *ad lib.* while another had isotonic glucose solution instead. Electrogenic secretion was measured in vitro using standard techniques (Baldwin & Levin, 1985) following the addition of carbachol (C) and bethanecol (B) (both 1 mM) to the serosal fluid. Fluid secretion in vivo was determined gravimetrically following C stimulation (55 μg/kg intraperitoneally). All surgery was carried out under pentobarbitone anaesthesia.

The basal short-circuit currents (ISc) and basal fluid secretion were unaltered in both the 72-h fasted rats and the glucose-fed rats compared with fed controls. However, C and B significantly elevated the ΔISc (ΔISc = peak ISc – basal ISc) in the 72-h fasted jejuna (B = +137%, n 10, $P<0.001$; C = +210%, n 6, $P<0.001$). The smaller elevation that occurred with glucose feeding (B = +39%, n 11, $P>0.05$; C = +28%, n 6, $P>0.05$) was not significant. The statistics were conducted using unpaired student's t test.

Although basal fluid secretion was unaltered between the groups, C stimulation caused an elevation in total fluid secretion in 72-h fasted rats over the fed control level (+48%, n 6, $P<0.001$). In the glucose-fed rats this elevation did not occur (+3%, n 6, $P>0.05$). The glucose ingested during the fast represented an intake of 150 kJ/d compared with a normal food intake of 289 kJ/d. Thus glucose ingestion significantly ameliorates the hypersecretion of fasted rat jejuna in response to cholinomimetics.

The authors gratefully acknowledge financial support from the British Digestive Foundation.

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Basal and pentagastrin-induced gastric acid and milk-clotting enzyme secretion in pigs from birth to 4 weeks of age. By P. D. CRANWELL, *School of Agriculture, La Trobe University, Victoria* 3083, *Australia,* and B. FOLTMANN, *Institute of Biochemical Genetics, University of Copenhagen, Denmark* and M. J. NEWPORT and G. L. HOWARTH, *AFRC Institute of Food Research, Shinfield, Reading RG2 9AT*

The aim of the present investigation was to study the development of the gastric capacity of the young pig to secrete acid and milk-clotting enzymes (M-CE) in response to the secretagogue, pentagastrin. The experiments were performed on thirty-two Large White × Landrace pigs, 0–27 d old, 1·2–6·4 kg body-weight from ten litters. Six pigs (3–28 h old) were not suckled by the sow, the remaining twenty-six pigs were reared entirely by the sow. Surgical preparation and gastric perfusion studies were done as described by Cranwell (1985). Following the basal period, pentagastrin was infused intravenously at dose rates of 4 and 8 μg/kg per h during two 1·5–2·5 h periods. Total milk-clotting activity was measured at pH 5·3 by radial diffusion in bovine skim-milk containing agarose (Lawrence & Sanderson, 1969). Depending on the experimental conditions the milk-clotting activities of pig chymosin and pig pepsin may differ by a factor of 0·5–2 (Foltmann *et al.* 1981). Purified pig chymosin was not available for the present study and pig pepsin was used as the standard. Milk-clotting activity was expressed as clotting equivalents (CE), i.e. the amount of pig pepsin which produces the same diameter precipitation zone.

There was a significant correlation between stomach weight (Y; g) and body-weight (X; kg), $Y = 5·22$ (SE 0·26) $X + 1·11$ (SE 0·85), r^2 0·93, $P<0·001$. The mean ratio of stomach weight:body-weight was 5·75 (SE 0·17) g/kg. In the six unsuckled pigs the mean maximal acid outputs (MAO) per unit body-weight and per unit stomach weight were: 138·6 (SE 14·8) μmol/kg per h and 25·4 (SE 3·0) μmol/g per h respectively. In all pigs there was a significant correlation between MAO (Y; mmol/h) and stomach weight (X; g), $Y = 0·145$ (SE 0·015) $X - 1·373$ (SE 0·285), r^2 0·76, $P<0·001$.

Considerable quantities of activated M-CE were present in the gastric contents of both fasted and unsuckled pigs (range 0·2–14·0 mg CE). During the basal period the mean output of M-CE was 2·13 (SE 0·27) mg CE/h (range 0·4–6·3 mg CE/h). There was a significant correlation between maximal M-CE output (Y; mg CE/h) and stomach weight (X; g), $Y = 0·52$ (SE 0·06) $X - 1·76$ (SE 1·08), r^2 0·71, $P<0·001$. During the infusion of pentagastrin, MAO occurred at the same time as maximal M-CE output in twenty-seven pigs. For all pigs there was a significant correlation between maximal M-CE output (Y; mg CE/h) and MAO (X; mmol/h), $Y = 4·98$ (SE 0·61) $X + 2·62$ (SE 1·04), r^2 0·70, $P<0·001$. The constant in the regression equation, 2·62 mg CE/h, is similar to the mean basal M-CE output, 2·13 mg CE/h. Thus the amount of M-CE secreted above basal amounts during pentagastrin infusion is related to the amount of acid secreted by the stomach.

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Ontogeny of pig gastric proteases; observations on fetal, stillborn, unsuckled newborn, suckled and growing pigs. By B. FOLTMANN, *Institute of Biochemical Genetics, University of Copenhagen, Denmark* and P. D. CRANWELL, *School of Agriculture, La Trobe University, Victoria* 3083, *Australia* and M. J. NEWPORT and G. L. HOWARTH, *AFRC Institute of Food Research, Shinfield, Reading RG2 9AT*

Cranwell *et al.* (1987) studied the development of the gastric secretory capacity of milk-clotting enzymes in young pigs. In the present investigation the milk-clotting enzymes are characterized and their locations in the gastric mucosa determined. Observations were made on the gastric contents and mucosa of six fetal pigs (105 d gestation) and four stillborn pigs, together with perfusates and gastric mucosa from pigs used in the previous experiment (Cranwell *et al.* 1987) and the gastric mucosa from a grower pig (111 d old).

The gastric contents of fetal and stillborn pigs consisted of clear, gelatinous fluid and some bile-stained material; the amounts were: fetal pigs 4–15 g, stillborn pigs 24–29 g. The pH of the gastric contents ranged from 2·5 to 5·0 and its acidity was 1–10 and 1–8·5 μmol/ml for fetal pigs and stillborn pigs respectively.

The gastric mucosa of each stomach was divided into cardiac (C), fundic (F) and antral (A) regions. Mucosal tissues were extracted (1:4, w/v) and the zymogens activated as described by Foltmann *et al.* (1981). Total milk-clotting activity was determined as described in Cranwell *et al.* (1987). Individual proteases were separated by agar gel electrophoresis at pH 5·3 and detected by clotting of casein (Foltmann *et al.* 1985). Examples of the results are shown in the Table.

	Fetal pigs				Stillborn pigs				Sucking pigs (3 d)			Sucking pigs (20 d)			Grower pigs (111 d)		
Stomach region . . .	C	F	A	GC	C	F	A	GC	C	F	A	C	F	A	C	F	A
CE (mg/ml)	0·3	1·0	0·1	0·4	3·4	9·0	4·0	0·5	0·5	3·5	0·9	0·2	1·5	0·7	2·8	5·0	0·4
Pepsin (*EC* 3.4.23.1)	0	0	0	0	0	0	0	0	(+)*	(+)	(+)	(+)	+	+	3+	3+	+
Gastricsin (*EC* 3.4.23.3)	0	0	0	0	0	0	0	0	(+)	(+)	(+)	+	+	+	2+	3+	2+
Chymosin (*EC* 3.4.23.4)	+	3+	+	2+	3+	3+	3+	2+	(+)	3+	+	+	3+	+	0	0	0

CE, clotting equivalents (Cranwell *et al.* 1987) in mucosal extracts and gastric contents (GC)
*Qualitative rating of casein clot after 2 h at 25°; comparison with pepsin standards: (+), <10 μg/ml; +, 10–40 μg/ml; 2+, 40–300 μg/ml; 3+, >300 μg/ml.

The results are consistent with previous observations that pigs produce little or no pepsin but considerable amounts of chymosin during their first days of life (Foltmann *et al.* 1981). Furthermore it is noteworthy that the gastric contents of fetal and stillborn pigs had a high concentration of chymosin and a low pH.

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Sympathetic activity in brown adipose tissue during lactation in mice. By PAUL TRAYHURN and MONICA C. WUSTEMAN, *Dunn Nutrition Laboratory, Medical Research Council and University of Cambridge, Downham's Lane, Milton Road, Cambridge CB4 1XJ*

Recent studies have indicated that the thermogenic activity of brown adipose tissue (BAT) is suppressed during lactation in rats and mice (Trayhurn *et al.* 1982; Isler *et al.* 1984). The mechanisms underlying this reduction in thermogenesis have not, however, been established. The main stimulus for thermogenesis in BAT is noradrenaline secreted by the sympathetic nervous system. Several studies have now shown that the reduced energy expenditure on BAT thermogenesis in obese animals relates to a decrease in sympathetic activity (see Romsos, 1985). In the present study we have measured the turnover of noradrenaline in BAT of lactating mice, in order to determine whether the reduction in thermogenesis during lactation results from a decrease in sympathetic activity.

Female mice of the 'Aston' variety were mated at about 8 weeks of age. After mating they were housed singly in plastic cages in a room at 22°. Dams were used at 9–13 d postpartum (peak lactation) and divided into groups of similar mean litter size. Virgin mice, housed singly and of the same age as the lactating animals, were taken as controls. Noradrenaline turnover was measured using the α-methyl-*p*-tyrosine (α-MPT) method (Young & Landsberg, 1977). Each mouse was injected intraperitoneally with 250 μg α-MPT/kg body-weight. Interscapular BAT was removed from groups of mice at 0, 1, 2 and 4 h after injection with α-MPT. The noradrenaline content of the tissues was measured by high-pressure liquid chromatography.

	Control			Lactating		
	Mean	SE	*n*	Mean	SE	*n*
Interscapular BAT wt (mg)	66·3	1·5	35	100·5***	2·7	44
Noradrenaline content (ng/organ)	104·2	3·4	54	78·8***	2·8	44
Fractional turnover rates (%/h)	40·0	0·7	4	12·7***	2·0	3
Turnover rate (ng/organ per h)	36·6	3·5	4	11·0**	2·9	3

Significantly different from control values: $^{**}P<0·01$, $^{***}P<0·001$.

The Table shows that the amount of interscapular BAT was increased in the lactating animals, but the total content of noradrenaline was significantly reduced in the tissue in lactation. Both the fractional turnover rate of noradrenaline and the total turnover rate (ng/organ per h) in BAT were much lower in the lactating mice than in the virgin controls.

These results indicated that the activity of the sympathetic nervous system is markedly reduced in BAT during lactation in mice. The reduction in thermogenesis in the tissue in lactating animals (Trayhurn *et al.* 1982; Isler *et al.* 1984) is therefore likely to be caused primarily by a decrease in sympathetic activity, although the involvement of additional factors cannot be discounted.

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