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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

A Scientific Meeting was held at the Royal Society of Medicine, London, on Thursday, 27 January 1994, when the following papers were presented.

Resting metabolic rate in rural Nepali men and women. By V.R. TUFFREY and S.S. STRICKLAND <u>Centre for Human Nutrition, London School of Hygiene and Tropical Medicine,</u> Keppel Street, London WC1E 7HT

It has been noted that resting metabolic rates (RMR) are frequently lower in populations of wellnourished adults living in tropical compared with temperate regions (Henry & Rees, 1991). Within tropical populations, a mechanism of increased 'metabolic economy' has been postulated in the chronically (Soares & Shetty, 1991) and acutely undernourished (Ferro-Luzzi *et al.* 1990). Such effects may be artefacts if inappropriate methods are applied to correct for body size and composition.

Physiological and anthropometric measurements were made on adults of two ethnic groups (mongoloid (M) and non-mongoloid (NM)), living at approximately 2000 m. above sea level in Kaski District, Nepal, an area of great seasonal variation in climate and agricultural work-load. RMR was measured with an Oxylog modified for low flow rates (McNeill *et al.* 1987).

		RMR (MJ/d)		<u>Ht (m)</u>	Ht (m)		Wt (kg)		FFM (kg)		FM (kg)		()
	<u>N</u>	Mean	SD	Mean	SD	Mean	SD.	Mean	SD	Mean	SD	Mean	SD
Male M	22	6.34	0.94	1.602	0.048	53.7	4.8	46.1	3.5	7.9	2.6	46	9
Male NM	29	6.13	1.03	1.605	0.057	50.6	4.9	45.4	4.3	5.6	1.4	37	9
Female M	27	5.09	1.20	1.503	0.042	46.7	5.2	35.6	3.2	11.3	3.5	35	7
Fem. NM	26	5.26	1.06	1.495	0.058	44.3	7.0	35.0	4.5	9.9	3.8	32	8

Values of RMR agreed more closely with those predicted by equations derived from data from temperate (Schofield *et al.* 1985; mean difference +2 (SD 18) %), rather than tropical populations (Henry & Rees, 1991; mean difference +11 (SD 19) %). By two way analysis of variance, absolute RMR was significantly different by sex (P<0.001), but not by ethnic group. The difference between sexes still remained (P<0.05) after analysis of covariance had been used to remove the linear effect of body weight, but not after having adjusted for fat-free mass (FFM) in the same way. A stepwise multiple regression of RMR against body weight was used to test for 'adaptation' to low weight-forheight. After having removed the linear effects of FFM and height, there was a significant association between RMR and body weight in women (P<0.001) but not in men. However, in women, the association between RMR and weight was not significant after having removed the linear effects of fat mass (FM) and height. Age had no independent effect in either sex. In a sub-sample measured in three seasons, by repeat measures analysis of variance there were differences between seasons in body weight (N 25, P<0.001), FFM (N 20, P<0.05) and FM (N 20, P<0.01), but not in RMR.

In conclusion, no differences in RMR were found by ethnic group, or by sex after adjusting for body size and composition. No evidence was found for metabolic 'adaptation' to either acute or chronic energy deficiency. This mechanism cannot be ruled out, however, given the limited precision and accuracy of the field techniques.

This study was funded by the Overseas Development Administration, with further financial support for VRT from the Simon Population Trust.

Ferro-Luzzi, A., Scaccini, S., Taffese. S., Aberra, B. & Demekc, T. (1990). European Journal of Clinical Nutrition 44 (Suppl. 1), 7-18.

Henry, C.J.K. & Rees, D.G. (1991). European Journal of Clinical Nutrition 45, 177-185.

McNeill, G., Cox, M.D. & Rivers, J.P.W. (1987). <u>American Journal of Clinical Nutrition</u> 45, 1415-1419. Schofield, W.N., Schofield, C. & James, W.P.T. (1985). <u>Human Nutrition: Clinical Nutrition</u> 39C (Suppl. 1), 5-96. Soares, M.J. & Shetty, P.S. (1991). <u>European Journal of Clinical Nutrition</u> 45, 363-373. Energy intake in hospitalized elderly patients. By P. MARSHALL¹, M. HOGARTH², G. FROST¹, S. MARTIN¹, F. BARTLETT¹ C. BULPITT², C. NICHOLL², L. LOVAT². ¹Department of Nutrition and Dietetics and ²Department of Medicine (Geriatrics). Hammersmith Hospital, Du Cane Road, London W12 OHS

The Kings Fund report <u>A positive approach to nutrition as treatment</u> has focused attention on malnutrition in the hospital population. Many reports have cited the elderly as an 'at risk' group (Constans *et al.* 1992). Interest has focused on protein, vitamins and minerals with energy intake being reported as a secondary finding (Bunker & Clayton, 1989). Of 119 patients admitted to the acute care of the elderly ward and recruited for a trial of energy and vitamin supplementation, fifty-two had a 24 h weighed food intake as part of the study protocol. We report the energy intake in these patients. Patients with a body mass index (BMI) <15 or >25 kg/m² or with discharge planned within 1 week were excluded. Weight was measured on electronic scales to the nearest 100 g. Arm span was taken as a surrogate measure of height and measured from finger tip to the centre of the sternal notch (Kwok & Whitelaw, 1991). The 24 h weighed food intake was carried out within 2 weeks of admission and analysed using the Microdiet computer program. Energy requirements were estimated using a standard equation (COMA, 1991). The Table shows the results of this study expressed as mean values.

		and the second distance of the second distanc	
	Result	SEM	
Age (years)	82	0.6	
Sex (m/f)	M 21 F 31		
BMI (kg/m ²)	19.6	0.39	
Energy intake (MJ)	4.41	0.31	
Energy requirements (MJ)	6.93	0.09	

Energy intakes and requirements compared by Student's t test were significantly different (P<0.0001). The mean percentage energy deficit between requirements and intake in those with BMI >20 or <20 kg/m² (40 % and 35 % respectively) was not statistically significant.

The hospitalized elderly clearly are at risk from sub optimal energy intakes and the consequences these carry. Further research is needed to assess methods of increasing energy intake and the clinical impact this will have. Patients who are classified as normal weight on admission may be as much at risk of low energy intake as those who are underweight.

Support for this study was given by SmithKline Beecham.

Bunker, V.W. & Clayton, B.E. (1989). Age and Ageing 18, 422-429.

Constans, T., Bacq, Y., Brechot, J.F., Guilmot, J.L., Choutet, P. & Lamisse, F. (1992). Journal of the American Geriatrics Society 40, 263-268.

Kwok, T. & Whitelaw, M. (1991). Journal of the American Geriatrics Society 39, 492-496.

COMA (1991). Dietary Reference Values for Energy & Nutrients in the United Kingdom Annex 2.

A comparison of estimated <u>v</u> measured energy expenditure in the obese. By C. MACQUEEN and G. FROST, <u>Department of Nutrition and Dietetics</u>, <u>Hammersmith Hospital</u>, <u>Du Cane Road</u>, <u>London</u> W12 OHS

A number of reports have demonstrated that the obese have a higher energy expenditure than their lean counterparts (James, 1983). We have demonstrated that weight reduction diets prescribing 2.01 MJ less than the estimated requirements (derived from WHO, FAO and UNU data) produce significantly greater weight loss than those based on traditional diet history methods (Frost, 1989). However, the data that this formula is based on includes few obese subjects. The energy expenditure work for this group appears to be an extrapolation from lean subjects (Schofield, 1985). The present study compares the accuracy of energy expenditure estimated using a formula against that estimated by indirect calorimetry. Thirty-four volunteers were recruited from the Hammersmith Hospital General Dietetic Out-patients Clinic (age 19-67 years, nine male, twenty-five female) with a Body Mass Index > 29 kg/m² (29-65 kg/m²). Their energy expenditure was measured using a Deltatrac indirect calorimeter (S.W. Vickers Ltd, Kent, UK; calibrated using standard gas after each subject and alcohol burning once every month). Measurements lasted 40 min including a 5 min run-in period and were carried out in the morning after an overnight fast. Weight was measured to the nearest 0.5 kg (Seka Scales, West Germany) and height to the nearest centimetre using a standometer.

There was no significant difference between energy expenditure estimated by the formula (7.7 SD 1.8 MJ) or by indirect calorimetry (7.0 SD 1.46 MJ). The lower mean estimate of energy expenditure from indirect calorimetry may be explained by the subjects restricting their food intake before the measurement.



Calorimeter MJ/day

The use of a formula to estimate energy expenditure in the obese appears to be a valid clinical tool. The formula used:

Men:		Activity factors:
18-30 yrs:	$BMR = 0.063 \times wt + 2.896$	Light = $BMR \times 1.55$
31-60 yrs:	$BMR = 0.048 \times wt + 3.653$	Moderate = $BMR \times 1.79$
60 + yrs:	$BMR = 0.049 \times wt + 2.459$	Heavy $=$ BMR \times 2.10
Women:		
18-30 yrs:	$BMR = 0.062 \times wt + 2.036$	Light = BMR x 1.56
31-60 yrs:	$BMR = 0.034 \times wt + 3.538$	Moderate = BMR x 1.64
60 + yrs:	$BMR = 0.038 \times wt + 2.755$	Heavy $=$ BMR x 1.82

The calorimeter was purchased with capital funding from the Special Health Authority and Cow & Gate.

Frost, G. (1989). <u>Practical Diabetes</u> 6, 273-275. James, W. (1983). <u>Lancet</u> ii, 386-389. Lean, M.E.S. & James, W.P.T. (1986). <u>Lancet</u> i, 723-725. Schofield, W.N. (1985). <u>Human Nutrition: Clinical Nutrition</u> 39C (Suppl.1), 1-41.

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Meal composition and postprandial mood and alertness: the influence of the fat and energy content of meals ingested mid-morning and at lunch time. By A.S. WELLS and N.W. READ, Centre for Human Nutrition, University of Sheffield, Northern General Hospital, Herries Rd, Sheffield S5 7AU

Large lunches cause a greater postprandial decline in alertness than low energy snacks (Craig & Richardson, 1989), and similarly high-fat lunches cause a greater postprandial decline in alertness than isoenergetic low-fat meals (Wells et al. 1993). The aim of the present study was to investigate the effects of fat content, energy content, and time of ingestion on postprandial alertness and mood.

There were two groups of nine healthy male volunteers, and each subject was tested on two non-consecutive test days in a single blind cross-over design. Group A (21 - 28 years) ingested meals at 10.30 hours and group B (21 - 33 years) ingested meals at 12.30 hours. Before the meal, and at 30 min intervals for 3 hours after the meals, subjects completed the Profile of Mood States questionnaire (McNair et al. 1971), and a series of bipolar visual analogue scales. The meals were similar in appearance, taste and protein content, yet the low-fat - high-carbohydrate (CHO) meal (fat:CHO energy ratio 7:88) was 418 kJ higher in energy than the high-fat - low-CHO meal (fat:CHO energy ratio 55:40).

After both of the meals ingested at 10.30 hours subjects became significantly more dreamy, feeble and fatigued. These effects were greater (P < 0.05) after the high-fat - low-CHO meal than after the low-fat - high-CHO meal notwithstanding the greater energy content of the latter (Table). Subjects also felt less friendly after the low-fat - high-CHO meal than after the high-fat low-CHO meal, (P < 0.05; Table).

	Mean post prandial change								
Measure	<u>High-fat</u>	-low-CHO	Low-fat	Pt					
	Mean	SD	Mean	SD					
Dreamy (scale 0-100)	15.2	5.7	8.8	6.8	0.033				
Feeble (scale 0-100)	12.6	5.6	5.2ª	4.0	0.022				
Fatigued (scale 0-28)	4.0	2.1	2.2 ^b	2.0	0.001				
Friendly (scale 0-100)	-0.7	3.6	-8.1ª	3.7	0.002				

^a Mean rating was significantly different from that of the high-fat - low-CHO group; ^b mean rating

3 h after the meal was significantly different from that of the high-fat - low-CHO group (P < 0.05).

+ significance levels determined using multiple variance analysis of variance.

After both of the meals ingested at 12.30 hours subjects felt significantly more dreamy, feeble and fatigued. There were no significant differences in these measures between the high-fat low CHO and the low-fat - high-CHO meals consumed at lunch time. Subjects tended to feel less friendly after the low-fat - high-CHO meal, but this failed to reach statistical significance.

In conclusion, independently of energy content, the incorporation of fat into a morning meal increases post prandial lassitude. However, this effect does not occur when the meal is eaten at lunch time.

Craig, A. & Richardson, E. (1989). International Archives of Occupational and Environmental Health 61, 313 -319 McNair, D.M., Lorr, M. and Droppleman, L.F. (1971). Manual: Profile of Mood States. Educational and Industrial Testing Service, San Diego, California.

Wells, A.S., Read, N.W. & Craig, A. (1993). Proceedings of the Nutrition Society 52, 352A.

1993

Nutritional composition (by chemical analysis) of sweet home-prepared weaning foods for infants. 1. Energy and macronutrients. By B. J. STORDY, ALISON M. REDFERN and JANE B. MORGAN, <u>School of Biological Sciences. University of Surrey. Guildford.</u> <u>Surrey GU2 5XH</u>

We have previously reported on the nutritional content by chemical analysis of 108 savoury weaningfood samples (Morgan *et al* 1993). Here we report on the results of the energy and macronutrient analysis of the sweet samples.

A study of weaning of 1004 infants in England was conducted between June and October 1992. A sub-sample of infants was identified as receiving a home-prepared meal at least once daily. Each mother or carer collected a duplicate sample (approximately 150 g) of the home-prepared sweet foods The foods were analysed (by National Measurement Accreditation Service accredited methodology) for fat, protein (N x 6.25), total carbohydrate (CHO) and non-starch polysaccharides (NSP), at Campden Food and Drink Association (Chipping Campden, Gloucs). Total energy was calculated from protein, fat and CHO using the energy conversion factors used in food labelling (Holland *et al.* 1991). We present the results of our analyses of ninety-eight sweet foods classified according to the age of the infant and compared with the mean nutrient content of ready-to-use baby desserts and puddings from the range of two major manufacturers (Table). Of the meals, 43 % had an energy density less than that of human milk (290 kJ/100 ml).

Age (months) n Nutrient	4-6 27 Mean SD	7-9 45 Mean SD	10-12 24 Mean SD	Manufactured infant foods 63 Mean SD
Energy (MJ/kg) Fat (g/kg) Protein (g/kg) CHO (g/kg) NSP (g/kg)	2.9 0.9 8 12 14 14 143 41 11 4	3.5 1.5 18 18 21 15 151 61 11 6	$\begin{array}{cccc} 3.7 & 1.2 \\ 18 & 15 \\ 28 & 18 \\ 150 & 62 \\ 9 & 4 \end{array}$	2.9 0.6 10 10 11 9 143 30 7 7

These data reveal that the energy density of some home-prepared meals is surprisingly low. There was some indication that energy density and protein content increased in the meals for older infants compared with the younger ones. The NSP content of some of the meals for younger infants was high and mean values at all ages exceeded mean values for manufactured desserts. Later estimations of the 24 h records of total energy and nutrient intake of which the meal was a part, will reveal whether these findings reflect overall nutrient supply.

Holland, B., Welch, A. A., Unwin, I. D., Buss, D. H., Paul, A. A. & Southgate, D. A. T. (1991).

The Composition of Foods 5th ed. Cambridge : RSC/MAFF.

Morgan, J.B., Redfern, A.M. & Stordy B.J. (1993). Proceedings of the Nutrition Society. 52 384A.

Influence of dietary long-chain *n-3* fatty acids on susceptibility to oxidation of low-density lipoprotein in man. By LUCIE POLLARD, FRANCESCA OAKLEY, and T.A.B. SANDERS. Department of Nutrition and Dietetics, King's College, University of London, Campden Hill Road, London W8 7AH

It has been proposed that the oxidative modification of low-density lipoprotein (LDL) is involved in the process of atherogenesis (Steinberg, 1993). Polyunsaturated fatty acids, especially those of the n-3 series, are more prone to oxidation than monounsaturated or saturated fatty acids. Moreover, oil-rich fish, which are the major sources of long-chain n-3 fatty acids, are also poor sources of vitamin E. We have previously reported that long-chain n-3 fatty acids increase the requirement for vitamin E in rats (Pollard & Sanders, 1993). We report the influence of diets of varying fatty acid composition on susceptibility of LDL to copper-induced oxidation. Healthy male volunteers (n 11) each received three isoenergetic diets for 3-week periods in a metabolic unit. The diets differed only in their fatty acid composition, vitamin E intakes were maintained constant. The diets were designed to provide approximately 35% energy as fat: the control diet was designed to supply 18% energy as saturates and 4% energy as linoleic acid, the experimental diets were both designed to be low in saturates (10% energy) with 6% energy as oleic acid and either 2% energy as linoleic acid or 2% energy as eicosapentaenoic acid (20:5n-3) and docosahexaenoic acid (22:6n-3) provided as fish oil, which was incorporated into foods. Fasting blood samples were obtained at the end of each dietary period. LDL was prepared by ultracentrifugation in a SW40 rotor and LDL oxidation induced by copper was determined by monitoring of conjugated diene formation. The lag phase to the commencement of conjugated diene formation was taken as the measure of susceptibility to oxidation. Vitamin E concentrations, as α -tocopherol, in LDL and plasma were determined by HPLC and the fatty acids composition of LDL by GLC. The results were as follows:

	High Satu	irated	Low satur 2% ener fatty a	rated + gy n-6 icids	Low saturated + 2% energy n-3 fatty acids		
	Mean	SE	Mean	SE	Mean	SE	
LDL α -tocopherol (μ mol/l)	14.4	1.74	15.8	2.62	21.8	6.33	
Lag phase (min)	198	25.2	167	16.4	88*	12.3	

*P < 0.01 compared with other groups

Plasma α -tocopherol and LDL tocopherol concentrations were similar on all three diets. The fatty acid composition was altered by the *n*-6 and especially the *n*-3 fortified diet (data not shown). The lag phase was markedly reduced when the subjects received the *n*-3 fortified diet demonstrating increased susceptibility to oxidation. Despite the capacity of *n*-3 fatty acids to promote LDL oxidation *in vitro*, they inhibit the development of atherosclerosis in animals (Sanders,1993). We propose that there are homeostatic mechanisms that minimize the potential harmful effects caused by the increased susceptibility of *n*-3 fatty acids to oxidation. T.A.B.S. acknowledges a grant from the British Heart Foundation

Pollard,L. & Sanders, T.A.B. (1993). Proceeedings of the Nutrition Society 52, 319A Sanders,T.A.B. (1993). Proceedings of the Nutriton Society 52, 457-472. Steinberg,D. (1993). New England Journal of Medicine 328,1487. Increased factor VII coagulant activity following the consumption of a high-fat meal in healthy subjects. By TAMARA DE GRASSI¹, G.J. MILLER² and T.A.B. SANDERS¹. ¹Department of Nutrition and Dietetics, King's College, University of London, Campden Hill Road, London W8 7AH and ²MRC Epidemiology and Medical Care Unit, Wolfson Institute of Preventive Medicine, Medical College of St Bartholomew's Hospital, Charterhouse Square, London EC1M 6BO

Elevated levels of factor VII coagulant (VIIc) activity are associated with increased risk of fatal ischaemic heart disease (IHD) particularly in men over the age of 55 years (Meade et al., 1993). Many studies have also demonstrated statistically significant and positive associations between plasma triacylglycerol concentrations and VIIc levels. Subsequent studies of healthy adults observed over 24 hour periods showed that the peaks and troughs in plasma triacylglycerol levels were accompanied by similar alterations in VIIc activity, but with a lag of several hours (Miller et al., 1991). The results in younger subjects have been less consistent. We report the influence of isoenergetic test meals of varying fat content on the VIIc activity in four male and two female normotriacylglycerolaemic subjects aged 22-43 years. Each subject consumed test meals at each level of fat intake (15, 60, 90 and 120 g) in random order. Venous blood venous samples were obtained in the fasting state and at 3 h and 7 h after the test meal and plasma triacylglycerol concentrations and VIIc activity were determined. The results were as follows:

Fat load	1	5 g	6	0 g	9	10 g	120 g	
VIIc (% standard)	Mean SE		Mean	SE	Mean	SE	Меап	SE
Fasting	102	5.1	110	10.5	107	4.7	112	7.1
3 h	104	7.6	107	7.4	118	8.5	115	7.0
7 h	98"	7.9	98"	4.1	11 9 ⁶	8.8	121	5.3
Triacylglycerol (mmol/l)								
Fasting	0.8	0.15	0.8	0.16	0.8	0.17	0.8	0.13
3 h	1.2*	0.25	1.2*	0.22	2.0 ^b	0.42	1.8	0.18
7 h	1.2	0.32	0.8	0.18	1.3	0.29	1.2	0.25

Values in the same row not sharing the same superscript are significantly different: P < 0.01

Plasma triacylglycerol concentrations were significantly elevated by test meals providing 90 and 120 g fat compared with those providing 15 and 60 g fat at 3 h. Factor VIIc activity was significantly elevated 7 hours after the 90 and 120 g fat meals. This study demonstrates that VIIc activity can be increased in healthy subjects providing there is a substantial increase in plasma triacylglycerol concentration. The lack of a relationship between VIIc activity and fatal IHD in younger men may reflect their capacity to clear dietary lipids from the circulation more efficiently.

T.A.B.S. acknowledges a grant from the British Heart Foundation.

Meade, T.W., Ruddock, V., Stirling, Y., Chakrabarti, R. & Miller, G.J. (1993). Lancet 342,1076-1079. Miller, G.J., Martin, J.C., Mitropoulos, K.A., Reeves, B.E.A., Thompson, R.L., Meade, T.W., Cooper, J.A. & Cruickshank, J.K.(1991). Atherosclerosis 86:163-171. Gastrointestinal flora do not contribute to increased ethane production on feeding fish oils. By B. J. P. QUARTLEY, C. M. WILLIAMS, M.N CLIFFORD and R. WALKER, <u>School of Biological Sciences</u>, University of Surrey, Guildford GU2 5XH

There is a need to develop non-invasive methods for the measurement of oxidative stress. The measurement of alkanes in breath samples is one such method and has been widely used in animal studies (Lawrence & Cohen, 1982) and to a limited extent in humans (Van Gossum *et al.* 1992). It has however been reported that the gut flora can produce alkanes under certain circumstances (Gelmont *et al.* 1981). The aim of this study was to investigate their possible contribution when fish oils are given as an oxidant source.

Twelve conventional Hooded rats (9 weeks old) on standard rat chow had their basal ethane exhalation measured using a recirculation system and were randomly divided into two groups of six. A control group (CO) received a semi-synthetic diet containing 50 g maize oil/kg as lipid source. A fish-oil group (FO) received a similar diet with MaxEPA (19.5 % 20:5, 10.2 % 22:6, Sevenseas) as the lipid source. Animals were allowed free access to food which was replaced daily. After 1 week on CO diet and 1 week on FO diet animals had their ethane exhalation measured. Animals in FO group were maintained on diet for a further week and ethane was measured at the end of 2 weeks.

Matched germ-free Hooded rats were subject to the same dietary treatments, maize oil or fish oil. However since it was not possible to return animals to germ-free conditions after ethane measurement it was only possible to measure ethane once from each germ-free animal. Basal ethane measurements and measurements for 1 week (maize-oil and fish-oil diets) and 2 weeks (fish-oil diet only) were thus carried out on separate groups of germ-free animals. There were six animals in each group. Food intakes of germ-free and conventional animals and of maize-oil and fish-oil diets were similar. Diets were irradiated to maintain germ-free conditions; both conventional and germ-free animals received the same irradiated diet. Results are expressed as nmol ethane accumulated in the recirculation system per kg body weight after 1.5 h.

Diet		Mai	ze oil		Fish oil	
		CO basal	1 week CO	FO basal	l week FO	2 week FO
Conventional	Mean	2.25	2.51	2.68	22.68ab	26.68ab
animals	SD	0.25	0.78	0.48	3.84	9.66
Germ-free	Mean	2.22+	6.88c	2.22+	29.38ac	30.93ac
animals	SD	0.29	0.77	0.29	5.39	12.06

a P<0.001 compared with 1 week CO; b P<0.001 compared with conventional FO basal; c P<0.001 compared with germ-free basal.

⁺Germ-free FO and CO basal were the same group of animals.

There was no significant difference in ethane exhalation on standard irradiated chow between germ-free and conventional animals. Replacing chow with a semi-synthetic maize oil diet caused a significant increase in ethane exhalation in germ-free animals only. Both germ-free and conventional animals showed a significant increase in ethane exhalation after 1 week on FO diet when compared with basal and CO groups. This increase was maintained after 2 weeks. There was no significant difference in the increase in ethane exhalation between germ-free and conventional animals on FO diet.

These findings indicate that when fish oils are given in the diet the increase in ethane production scen is true *in vivo* production and not a product of gut flora. It should be noted that irradiation of the diet can be expected to destroy part of the vitamin E and possibly induce lipid peroxidation. However as all animals received irradiated diets these effects do not invalidate the comparisons.

This work was supported by funding from Nestec Ltd.

Gelmont, D., Stein, R.A. & Mead, J.F. (1981). <u>Biochemical and Biophysical Research Communications</u> 102, 932-936. Lawrence, G.D. & Cohen, G. (1982). <u>Analytical Biochemistry</u> 122, 283-290.

Van Gossum, A., De Cuyper J., Ooms, H., Cremer, M. & Jeejeebhoy, K. N. (1992). <u>Acta Gastro-Enterologica Belgica</u> 15, 245-248. The effect of postprandial lipaemia on platelet aggregation. By C. HALLETT, S.M.O. HOURANI and C.M. WILLIAMS, <u>School of Biological Sciences</u>, <u>University of Surrey</u>, <u>Guildford</u>, <u>Surrey</u>, <u>GU2</u> <u>5XH</u>

Postprandial lipaemia is the appearance of lipid particles in blood following the ingestion of a fatty meal and is monitored by increases in plasma and chylomicron triacylglycerol concentrations.

The main physiological function of platelets is haemostasis, and disturbances in their function may play an important part in the pathogenesis of coronary artery disease. Platelets are known to interact with lipoproteins (Aviram & Brook, 1983), but detailed studies of the effects of food ingestion and postprandial lipaemia on platelet aggregation have received insufficient attention.

In the present study, ten male subjects (18 - 23 years) were randomly as igned to two groups and given either a high-fat (80 g) or a low-fat (20 g) meal. (The meals were standardized for their carbohydrate and protein content.) Blood samples were taken in the fasted state and 5 h (300 min) postprandially. Platelet aggregation was studied in platelet-rich plasma (3 x 10^8 platelets/ml). Aggregation was measured in response to a range of thrombin (0.05 - 0.5 U/ml) and adenosine diphosphate (ADP; 0.3 - 30 µmol/l) concentrations. The pD₂ value (i.e. the negative log of the agonist concentration causing half the maximal response) was calculated for each concentration-response curve and these are shown in the Table. The plasma triacylglycerol (plasma TAG) and chylomicron triacylglycerol levels were also measured and the plasma values are shown below.

				pD	2		-					
	.		ADP		THROMBIN				Plasma TAG (mmol/l)			
	<u>0 min 300 min</u>			0 min300		min	<u>0 n</u>	0 min		min		
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Low-fat group (n 5)	5.67	0.15	5.74	0.18	0.74	0.14	0.70	0.11	1.13 ^a	0.20	1.44 ^b	0.20
High-fat group (n 5)	5.83	0.11	5.73	0.16	0.69	0.06	0.67	0.08	0.91 ^a	0.17	1.72 ^b	0.49

a, b Mean values were significantly different (P < 0.05), paired comparisons.

No significant differences were found between pD₂ values at baseline and at 300 min postprandially for either low- or high-fat meals using either ADP or thrombin as the agonist. There were no significant differences in pD₂ values in subjects following the high-fat compared with the low-fat meal. There was no correlation found between pD₂ values and chylomicron or plasma triacylglycerol levels. The 300 min chylomicron and plasma triacylglycerol values were significantly higher than baseline for both the low-fat (P < 0.05) and the high fat meal (P < 0.05). However there were no significant differences in 300 min triacylglycerol values between the high- and low-fat meals.

In conclusion, there is no effect of postprandial lipaemia on *in vitro* platelet aggregation in response to ADP or thrombin, and this lack of response is observed even following the consumption of a high-fat meal. Reported effects of postprandial lipaemia on a tendency to thrombosis (Aznar *et al*, 1987) cannot therefore be attributed to the effect of lipoprotein concentration on platelet aggregation.

Aviram, M. & Brook, J.G. (1983). <u>Atherosclerosis</u> 46, 259 - 268. Aznar, J., Santos, M.T. & Vallés, J. (1987). <u>Thrombosis Research</u> 48, 567 - 576. Meal fat content and postprandial lipaemia: apo B48 and retinyl palmitate as markers of chylomicron particles. By S. G. ISHERWOOD, S. SETHI, B. J. GOULD, R. J. HOWLAND and C M. WILLIAMS, <u>Nutritional Metabolism Research Group, School of Biological Sciences, University of Surrey, Guildford GU2 5XH</u>

Factors which increase the magnitude, or prolong the time-course, of postprandial lipaemia may be associated with an increased risk of coronary heart disease (Zilversmit, 1979). Retinyl palmitate (RP) has been used as a marker for chylomicron (CM) and chylomicron remnant (CMR) particles. However, due to the apparent transfer of RP to other lipoprotein classes during the late postprandial phase, it would be preferable to use apolipoprotein B48 (apo B48) as a marker for CM particles as this protein remains associated with the CMR until its uptake by receptors in the liver. The availability of a specific antibody to apo B48 (Peel *et al.* 1992) enables both these markers to be measured, and their responses compared, in experimental studies of postprandial lipaemia in man.

In the present study the effect on postprandial lipaemia of test meals of varying fat content was investigated. Ten matched, healthy, male subjects were recruited and randomly allocated to receive on separate occasions each of three test meals containing 20, 40 and 80 g fat. Blood samples were collected in the fasted state and hourly after eating the meal for an 8 h period. Retinyl palmitate (100mg; 170,000 IU) was taken with the meal. Triacylglycerol (TAG) and non-esterified fatty acid (NEFA) concentrations were measured in plasma and apo B48, RP and TAG concentrations were measured in a CM-enriched fraction isolated from plasma by ultracentrifugation. An aliquot of the same CM sample was used as an internal standard in the analysis of apo B48. The results are shown in the Table as total area under the time response curve (AUC).

	AUC									
	20 g	fat	40 g	fat	80 g fat					
	Mean	SEM	Mean	SEM	Mean	SEM				
CM-TAG (mmol/l)	165	26	246	35	300 ^a	31				
CM-RP (µg/ml)	369	80	705	173	1040 ^a	207				
CM-Apo B48 (% standard)	12901	3036	15120	4051	19553	5402				
Plasma-TAG (mmol/l)	493	32	545	40	610 ^a	28				
Plasma-NEFA (mmol/l)	177	13	188	21	218	14				

a, mean value was significantly different (P < 0.05) compared with the low-fat meal.

The CM-TAG, CM-RP and plasma-TAG AUC values were significantly higher after the high-fat compared with the low-fat meals. The CM-apo B48 AUC showed the same trend but this difference did not reach statistical significance. The values for plasma-NEFA AUC increased with increasing meal fat content but differences between meals did not show statistical significance. Comparison of the pattern of NEFA response showed a more marked rise in plasma-NEFA in the early postprandial period (30-360 min) in response to the high fat meal (P<0.04) compared with the low-fat meal.

In conclusion there is a progressive increase in TAG concentrations and CM particle number in response to meals of increasing fat content. Apparently similar responses were observed for retinyl palmitate and apo B48, the two markers for intestinally-derived lipoproteins.

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Zilversmit D. B. (1979). <u>Circulation</u> 60, 357-365.

The effect of prolonged exercise on selected brain monoamine concentrations in the rat. By W. WILSON¹, C.A. MARSDEN¹ and R.J. MAUGHAN². <u>¹Department of Physiology and Pharmacology</u>, <u>Nottingham University</u>, NG7 2UH and <u>²Department of Environmental and Occupational Medicine</u>, <u>Aberdeen University</u>, <u>Aberdeen AB9 2ZD</u>

Dietary manipulation, starvation and exercise alter the plasma amino acid profile and may therefore influence the uptake of plasma free tryptophan across the blood-brain barrier and synthesis of brain serotonin (Chauloff, 1989). Prolonged exercise of moderate intensity increases the plasma free fatty acid and free tryptophan concentration and may increase brain serotonin synthesis (Davis *et al* 1992).

This study investigated the effects of 60 min of moderate intensity exercise on serotonin and dopamine concentration in different brain regions of the rat. Male hooded Lister rats (280-327 g) were housed individually for the duration of the experiment. After a week of familiarization to the treadmill, the rats to be trained (T,n6) began a 3 week period of training during which the speed of the treadmill was increased to 20 m/min at an incline of 8° and time spent running to 60 min. The control rats (C, n6) were placed on the stationary treadmill for the same length of time and both groups underwent the same handling protocol. On the day of the experiment, the trained rats ran for 60 min at 20 m/min, were killed immediately and the brain regions dissected. The control group underwent the same protocol but did not run on the treadmill.

Exercise increased plasma free (or non-protein bound) tryptophan concentration (T, 10.5 (SE 1.5) μ mol/1: C,6.2 (SE 0.9) μ mol/1, p<0.05). The effect of exercise on serotonin (5-HT), its metabolite 5-hydroxyindoleacetic acid (5-HIAA), and dopamine (DA) concentrations (nmol/g) is shown in the following table.

Area	Condition	<u>5-H</u>	T	<u>5-HI</u>	<u>AA</u>	DA
		Mean	SE	Mean	SE	Mean SE
Frontal Cortex	С	2.39	0.27	2.36	0.25	n.m.
	Т	3.30	0.54	2.13	0.18	n.m.
Hippocampus	С	1.51	0.31	2.38	0.17	n.m.
	Т	2.19	0.31	2.08	0.12	n.m.
Hypothalamus	С	2.00	0.22	2.40	0.35	1.59 0.27
	Т	1.71	0.71	1.87	0.17	2.49* 0.31

*P<0.05 v. C; n.m., not measured

Moderate intensity exercise of 60 min duration increased brain dopamine concentration in the hypothalamus, and although not significant, there was a trend towards increased brain serotonin concentration in the hippocampus and in the frontal cortex. The results therefore suggest that prolonged exercise may increase the available supply of the neurotransmitter precursor, plasma free tryptophan and increase subsequent neurotransmitter synthesis.

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The influence of oral creatine supplementation on metabolism during sub-maximal incremental treadmill exercise. By A. L. GREEN¹, P. L. GREENHAFF¹, I. A. MACDONALD¹, D. BELL², D. HOLLIMAN² and M.A. STROUD², ¹Department of Physiology and Pharmacology, Medical School, Queen's Medical Centre, Nottingham NG7 2UH and ²Army Personnel Research Establishment, Farnborough, Hants GU14 6TD

It has recently been demonstrated that dietary creatine (CR) supplementation can increase muscle total CR content (Harris *et al.* 1992) and improve performance during maximal voluntary isokinetic knee extension (Greenhaff *et al.* 1993). The present study investigated the effect of dietary CR supplementation on metabolism during sub-maximal incremental exercise.

Eight healthy men ran (10 km/h) on a motor driven treadmill at pre-determined workloads equivalent to 50, 60, 65, 70, 75, and 80 % of maximal O₂ uptake ($\dot{V}O_2$ max). During the final minute at each workload, expired gas was collected for the determination of O₂ consumption ($\dot{V}O_2$) and a blood sample was obtained from a superficial forearm vein for subsequent determination of blood lactate concentration. One hour following the completion of this exercise, subjects performed five bouts of isometric contraction at 70 % of their maximal voluntary force, to the point of exhaustion. Each bout of contraction was separated by 3 min recovery. Two days later, subjects began consuming 5 g CR four times / d for 5 d, after which they returned to the laboratory and repeated the exercise tests. The lactate and $\dot{V}O_2$ responses to exercise were similar before and after CR ingestion (Table).

			Pre (CR		Post CR						
% VO _{2max}	<u>60</u>		<u>70</u>		<u>80</u>		<u>60</u>		<u>70</u>		<u>8(</u>	<u>)</u>
****	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
VO ₂ (1/min)	3.0	0.2	3.5	0.1	3.8	0.2	3.0	0.1	3.4	0.1	4.0	0.1
Blood lactate (mM)	1.4	0.2	1.8	0.2	4.2	0.6	1.3	0.2	2.2	0.2	4.4	0.4

Total isometric endurance time (sum of all five bouts) before CR was 252 (SE 25) s. After CR ingestion this increased significantly by 59 (SE 22) s (P<0.05). Thus, CR ingestion had no influence on cardio-respiratory function and blood lactate concentration during sub-maximal incremental treadmill exercise. However, CR ingestion did improve performance during short duration intense exercise, as previously reported (Greenhaff *et al.* 1993).

This study was approved by the Army Personnel Research Establishment Ethics Committee.

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