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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

A Scientific Meeting was held at the Clarendon Suites, Birmingham on Tuesday–Thursday, 6–8 December 1994, when the following papers were presented. The first six papers constituted an interdisciplinary meeting with BAPEN.

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Changes in body composition in response to chemotherapy in patients with acute myeloid leukaemia. By G.E.A BETTANY[1], A.C. NEWLAND[2] and J. POWELL-TUCK[1], *Department of Human Nutrition[1] and Department of Haematology[2], The London Hospital Medical College, London E1 1BB*

The body composition of seven patients (four male, three female, mean age 30.6 (SD 11.3) years) with acute myeloid leukaemia (AML) and a normal baseline Quetelet index, were studied using anthropometric measurements of the non-dominant arm and whole body dual energy X-ray absorptiometry (DEXA) together with measurements of resting energy expenditure (REE) by indirect calorimetry at the beginning and end of a mean admission periods for chemotherapy of 31.5 d (range 28-36 d). REE values were within 5% of those predicted by Schofield (1985). The chemotherapeutic regime was given according to the standard protocol at this hospital.

The first measurement were recorded before starting chemotherapy.

	Pre-Treatment		Post-treatment		Difference		*P*
Measurement	Mean	SD	Mean	SD	Mean	SD	
Weight (kg)	75.3	13.4	71.8	13.0	-3.5	1.92	<0.01
Mid arm (non-dominant) circumference (cm)	28.0	3.5	26.9	3.4	-1.1	0.68	<0.01
Biceps skinfold (mm)	7.7	3.4	7.2	3.2	-0.6	0.4	<0.01
Fat (kg)	22.8	10.4	21.7	9.6	-1.1	1.2	<0.1
Lean (kg)	48.5	5.9	46.7	5.5	-1.8	1.5	<0.01
BMI (kg/m^2)	24.6	4.4	23.5	4.0	-0.2	0.7	<0.01
REE (kJ/kg lean body mass)	147.7	38.5	156.3	34.0	8.5	18.0	<0.5

There was a significant correlation (r 0.683, $P<0.05$) of weight loss with loss of lean body mass as assessed by DEXA scan. No such relationship existed for weight changes and body fat content. The ratio of lean tissue to fat lost was 1.71:1. The reduction in weight seen during a single course of chemotherapy for AML thus arises predominantly from lean body mass rather than fat stores in contrast to starvation in healthy obese individuals where weight loss is normally approximately 50% fat and 50% lean (Garrow, 1988). This has quantitative implications for the design of nutritional support for such patients. Nutritional support should aim predominantly to preserve lean body mass in these patients.

Schofield (1985). *Human Nutrition-Clinical Nutrition.* **39** Suppl.1, 5-41
Garrow, J.S. (1988). *Obesity and Related Diseases.* Edinburgh: Churchill Livingstone.

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Nutritional screening: evaluation and implementation of a simple nutrition risk score. By H.M. REILLY[1], J.K. MARTINEAU[1], A. MORAN[2] and H. KENNEDY[1], [1]*Nutrition and Dietetic Department and* [2]*Department of Gastroenterology, Birmingham Heartlands Hospital, Birmingham B95SS*

Undernutrition remains prevalent amongst hospital patients (McWhirter *et al.* 1994). In the present study, a simple nutrition risk score (NRS; simplified version, Table1), was developed to survey the risk of undernutrition in a sample of hospital inpatients and later implemented as a hospital-wide nutritional screening tool.

Table1 *Select one score from each section* (complete NRS includes explanations to aid selection of score)

Paediatrics (0-17 years)

1. Present weight

Appropriate *0*

90-99% of expected *2*

80-89% of expected *4*

<79% of expected *6* (- Q3)

3. Appetite Good *0* Poor *2* Nil *3*

4. Ability to eat /retain food

No problems *0* some difficulty *1* significant problems *2* unable to eat / retain food *3*

5. Stress factor None *0* mild *1* moderate *2* severe *3*

Adults (>18 years)

1. Weight loss in last 3 months (unintentional)

nil *0* 0-3 kg *1* >3-6 kg *2* 6 kg or more *3*

2. Body mass index

20 or more *0* 18-19 *1* 15-17 *2* <15 *3* (- Q3)

Total score:- *0-3* Low risk - no action necessary *4-5* Moderate risk- needs monitoring *6-15* High risk- refer to dietitian

To validate the NRS, twenty inpatients, two from each of ten specialities were assessed to evaluate correlation of the score with a validated nutritional risk index (Wolinsky *et al.* 1990); correlation with the dietitian's clinical impression of nutritional status; reproducibility of scores by dietitians and nursing staff; and ease of use of the NRS. The NRS was found to be easy to use, applicable to all patient categories and ages, and correlated well with other assessment methods (r 0.68, $P<0.001$) and (r 0.83, $P<0.001$). Reproducible scores were obtained between dietitians (r 0.91, $P<0.001$) and between dietitians and nursing staff (r 0.80, $P<0.001$). The NRS was used to assess the nutritional risk of 153 consecutive admissions to medical and surgical specialities. Current action taken to prevent deterioration in nutritional status was assessed.The results are shown in Table 2.

Table 2	Nutrition risk score		
	Low 77/153(50%)	Moderate 36/153(24%)	High 40/153(26%)
Action			
Referred for nutritional support	6/77 (8%)	7/36 (19%)	24/40 (60%)
Monitored (weight checked/supplements given)	10/77 (13%)	6/36 (17%)	4/40 (10%)
No action taken	61/77 (79%)	23/36 (64%)	12/40 (30%)

These figures confirm that poor nutritional status remains an extensive problem amongst hospital inpatients and demonstrate that appropriate action is infrequently taken. The NRS is a simple framework within which to promote a multidisciplinary approach to nutritional support.

In conclusion, many patients are at risk of undernutrition with no demonstrable intervention. The nutrition risk score is suitable for use as a screening tool and has been implemented as part of the standard nursing assessment process at this hospital.

McWhirter, J.P., Pennington, C.R. (1994). *British Medical Journal* **308**:945-948.

Wolinsky, F.D., Coe, R.M., McIntosh, W.M.A., Kubena, K.S., Prendergast, J.M., Chavez, M.N., Miller, D.K., Romeis, J.C., Landmann, W.A. (1990). *Journal of Nutrition* **120**:1549-1553.

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Two-phase randomized controlled clinical trial of oral dietary supplements in surgical patients: results of in-patient phase I. By A.M. KEELE[1], M.J. BRAY[1], P.W. EMERY[2] and D.B.A. SILK[1], *[1]Department of Gastroenterology and Nutrition, Central Middlesex Hospital NHS Trust, Acton Lane, London NW10 7NS and [2]Department of Nutrition and Dietetics, Kings College, London W8 7AH*

It has previously been shown that oral dietary supplements have clinically significant short-term benefits in surgical patients (Rana *et al.* 1992). The aim of the present study was to re-evaluate the short-term clinical efficacy of oral dietary supplements administered postoperatively to in-patients undergoing moderate to major gastrointestinal surgery (phase I), and later in phase II, to investigate the clinical efficacy of oral dietary supplements given during the first 4 months following hospital discharge. In phase I, 100 patients who were scheduled to undergo pre-determined moderate to major open gastrointestinal surgery entered the study. They were randomly assigned to receive a normal ward diet postoperatively or the same diet supplemented *ad libitum* with an oral nutritional supplement (6.3 kJ/ml, 8 mg N/ml). The study period was defined as commencing from the day the patients started ingesting "free fluids" postoperatively, to the day of hospital discharge. Eighty-six patients (forty-three in each group) completed the study. Trial end-points for comparison were nutritional intake, nutritional status, incidence of serious complications and subjective levels of fatigue.

The mean daily energy and protein intakes assessed from daily food records were significantly higher in the treatment group than in the control group at study days 1, 2 ($P<0.0005$) 3 and 4 ($P<0.05$). No significant differences were noted at days 5 and 6. On day 7 protein intake was greater in the treatment group ($P<0.03$). Patients in the treatment group lost significantly less weight than control patients by discharge; 2.2 (SEM 0.5) kg compared with 4.2 (SEM 0.4) kg, ($P<0.001$). Patients in the control group showed a significant reduction in hand grip strength over their hospital stay, ($P<0.02$), whereas patients in the treatment group maintained their hand grip strength. No significant differences in any other nutritional variables were seen. Subjective levels of fatigue, assessed using a 10 point scale (Christensen *et al.* 1982), increased significantly in control patients (n 18) at study day 3 ($p<0.01$) and at discharge ($p<0.02$) compared with preoperative levels. There was no change in fatigue level in the supplemented patients (n 17).

Significantly more patients in the control group (12) developed serious complications (wound infection 7, wound dehiscence 2, gastrointestinal perforation 1, subphrenic abscess 1, multiple complications 1) than in the treatment group (4; wound infection 2, wound dehiscence 1, multiple complications 1; $P<0.05$).

It is concluded that the prescription of oral dietary supplements on an *ad libitum* basis to postoperative patients undergoing moderate to major gastrointestinal surgery results in clinically significant short-term benefits.

Christensen, T., Bendix, T. & Kehlet, H. (1982). *British Journal of Surgery* **69**, 417-419.

Rana, S.K., Bray, J., Menzies-Gow, J., Jameson, J., Payne-James, J., Frost, P. & Silk, D.B.A. (1992). *Clinical Nutrition* **11**, 337-344.

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Comparison of implanted ports with Broviac/Hickman-style tunnelled catheters for long-term home parenteral nutrition: the patient's view. By S. MAGNAY[1], C. WHEATLEY[2], S. WOOD[1], and A. FORBES[1]. [1]*Nutrition Unit, St Mark's Hospital, City Road, London EC1V 2PS and* [2]*PINNT Secretariat, 258 Wennington Road, Rainham, Essex RM13 9UU.*

Patients with intestinal failure may ultimately need long-term parenteral feeding at home. There are two principal means of central venous access: the fully implanted port requiring skin puncture to achieve each venous access, or the Broviac/Hickman type of tunnelled catheter requiring capping off when not in use. Medically based comparisons of these have shown few differences in terms of efficacy, safety or freedom from complication, and ports are less used than Broviac-style catheters in the UK (financial reasons and ease of insertion playing a part, given apparent medical equivalence). There is however a paucity of information on the patient's views and patients are often given little choice as to which method is used. A pilot study has therefore been performed.

Patients with intestinal failure on home parenteral nutrition for at least 6 months with an implanted port were asked to complete an anonymous eighteen item questionnaire examining attitudes to, and satisfaction with, their intravenous access. The questionnaire was designed to address the patients' views as to the need for feeding, the time and manipulations involved, and their perceptions of complication rates, and to assess limitations to lifestyle including sexual functioning and activities of daily living. Answers were reported using a five point Likert scale (e.g. "not at all" - "frequently") yielding a score from 1 to 5 for each question. A global health questionnaire (SF-36) was also completed to provide an externally validated assessment of health status. For each patient with a port, a control patient with a Broviac-type catheter was selected with close matching for age, sex and diagnosis.

Results of the study confirmed that the port and Broviac-style patients were well matched. There were no major differences in responses to the global assessment of health status between the two groups. No differences emerged in patients' perceptions of complication rates (independently validated from the Unit's medical database) with a median of <1 line change in 3 years. Disruption caused by the manipulations necessary to commence and end feeding, and by the time of infusion itself, did not differ between the two groups. When not connected to an infusion bag, patients in both groups felt largely unaware of their device, but in choice of clothing (males and females), limitations of (non-sexual) activities, and embarrassment relating to the presence of the device, patients with ports gave consistently more satisfied responses (median scores: 2 *v.* 4, 1 *v.* 3, and 1 *v.* 4, respectively). Sexual activity and pleasure therefrom for patient and partner was significantly limited with both devices (median scores of 4) with no obvious differences between the two groups. The most marked difference emerged in relation to water contact and self hygiene, where all port patients were able to bathe and shower freely in contrast to Broviac-equipped patients, all of whom returned scores of 4 or 5 indicating frequent limitation of perceived hygiene needs.

The decision to use port or Broviac-style catheter in patients included here was not based on randomized selection, and our conclusion should not therefore be considered necessarily applicable to all patients requiring long-term home parenteral nutrition. However, as the previous medical data show few differences, the greater satisfaction of patients with implanted ports indicates to us (unless controlled data emerge to the contrary) that in most cases the choice of device should be that of the informed patient.

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Changes in enteral feeding: a positive response to clinical audit. By J.M. STANFORD[1], G. FROST[1], J. WALTERS[2], C. MACQUEEN[1], P. MARSHALL[1], S. MARTIN[1], M. KELLY[1] and K. MASTERS[1]. [1]*Nutrition and Dietetic Department and* [2]*Department of Gastroenterology, Hammersmith Hospital, Du Cane Road, London, W12 OHS*

McWhirter *et al.* (1994) identified malnutrition as a continuing problem in hospital and highlighted a need for education on clinical nutrition. Following our initial audit of enteral feeding practice in 1992, which identified a baseline of improvement (Frost *et al.* 1994), a second audit was carried out.

Since 1992 a number of changes have been instigated including the publication of the nutrition policy in the pharmacy list given to all junior doctors, an active nutrition team, new methods of enteral access and a more aggressive approach to promoting enteral feeding and identifying patients and training of nursing staff. These figures demonstrate the changes that have occurred, comparing a 6-month period in 1992 with a similar period in 1994.

Year	No. patients	Age (years) (range)	Days fed (as % patients) 1	2-5	6-10	11-15	16-20	20+
1992	52	67 (34-86)	26	37	11	4	4	15
1994	115	72 (31-94)	8	24	24	10	5	28

Year	Methods of feeding (as % patients) NG	PEG	NJ	JEJ
1992	93	7	0	0
1994	70	15	3	7

NG, nasogastric; PEG, percutaneous endoscopic gastrostomy, NJ, nasojejunal; JEJ, jejunostomy.

The results highlight the following changes: (1) a 113% increase in the number of patients being enterally fed; (2) a 50% reduction in the number of patients fed for less than 5 d and a substantial reduction (220%) in patients fed for less than 1 d; (3) a 107% rise in PEG feeding has allowed a substantial increase in the length of time patients are fed. Of interest is that of the ten patients fed for more than 50 d (range 51-235), two were successfully fed via nasogastric tubes (51 d and 152 d respectively); (4) the introduction of nasojejunal and jejunostomy feeding post-operatively has enabled enteral feeding rather than parenteral feeding to be established.

In conclusion, the changes instigated at the Hammersmith Hospital after the previous audit have allowed more patients to be fed for longer periods and with possible greater benefits to the patient.

Frost, G., Stanford, J., Masters, K., Taylor, T., Ward, L., & Walters, J.R.F. (1994). *Journal of Human Nutrition and Dietetics* 7, 61-68.

McWhirter, J.P. & Pennington, C.R. (1994). *British Medical Journal* **308**, 945-948.

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The use efficacy and monitoring of artificial nutritional support (ANS) in a teaching hospital. By J.P. McWHIRTER[1], K. HILL[2], J. RICHARDS[3] and C.R. PENNINGTON[1], [1]Department of Clinical Pharmacology, [2]Department of Nutrition and Dietetics and [3]Pharmacy Department, Ninewells Hospital and Medical School, Dundee DD1 9SY

Guidelines for the management of ANS were compiled by the Nutrition Advisory Group, circulated to all units and used as the standard against which to audit the implementation, efficacy and monitoring of the ANS service. Standard regimens for parenteral (PN) were used where possible to limit costs, and catheter care protocols were defined. The present study aims to determine the amount of ANS used; the implementation of these guidelines by assessing the extent to which nutritional goals were set and achieved; and recording the levels of morbidity as a result of complications.

Adults receiving ANS in hospital and at home were studied over a 6-month period. The delay between last significant oral intake and initiation of ANS was calculated. Nutritional assessments were carried out using anthropometry at the start of the feeding period and post-feeding measurements were taken in those patients supported for 7d or more. Energy requirements were retrospectively calculated and compared with prescriptions and actual intakes. Complications and interruptions to the regimens which resulted in lost feeding time were recorded, as were patient outcomes.

In total, 167 patients were recorded. Of the eighty-eight patients who received enteral nutrition (EN), twenty-four were fed at home (twenty were not available for study). Patients were fed for a mean of 8.3d (range 1-80d). Forty-nine patients received PN in hospital for a mean of 17.3d (range 1-60d). Ten patients received home PN (HPN) throughout the audit period. The mean time lapse between last significant oral intake and the start of ANS in hospital was 5.4d (range 0-23d). Twenty-nine patients (20%) eighteen EN, eleven PN) were fed for >7d.

The aim of nutritional support was to maintain nutritional status in 82/147 (56%) patients (fifty-five EN and twenty seven PN) and to improve that of sixty-five (44%) (thirty-four EN and thirty-one PN). Nutritional assessments were carried out on 118/147 (80%) of all patients at the start of the feeding period and ninety-one (62%) were re-assessed at the end. Of the patients receiving EN, the aim of ANS was achieved in 37/88 (39%), failed in thirteen (16%) and the outcome was unknown in forty-two (45%) because of death, transfer or early cessation. In the group supported by PN, the aim was achieved in 28/59 (48%), failed in thirteen (22%) and unknown in eighteen (30%).

	Enteral (n 88)	Parenteral (n 59)
Prescription <90% of requirements	27 (31%)	17 (29%)
Delivery 100% of prescription	34 (39%)	31 (52%)
" >90% "	18 (20%)	8 (14%)
" >75% "	21 (24%)	6 (10%)
" >50% "	12 (14%)	11 (19%)
" <50% "	3 (3%)	3 (5%)

A total of 55/2381 (2.3%) feeding days were lost by 69/147 (47%) patients (twenty-four EN, thirty-one PN). Complication of EN in hospital accounted for an overall loss of 564 feeding hours by 40/84 patients (mean 14.1): 92 h tube-related (twenty-two patients); 48 h gastrointestinal intolerance (eleven patients); and 376 h due to delivery problems (sixteen patients). PN complications resulted in 747 lost hours by 19/53 patients (mean 39). In hospital, catheter-related complications occurred in 15/49 patients (579 lost hours) and problems with delivery resulted in 120 h lost by three patients. Metabolic complications occurred in two patients (48 h lost). Catheter-related sepsis was documented for five (three surgical, two intensive care) patients resulting in one line removal and 132 lost hours by the remaining four patients. Two HPN patients were admitted to hospital as a result of catheter complications, one line was accidentally displaced and one line was replaced due to a tunnel infection.

The energy prescription of more than a quarter of patients (26% EN, 32% PN) were insufficient to meet retrospectively assessed requirements. Prescribed nutrient delivery was achieved in two thirds and nutritional aims were achieved in only 44% of patients (26% EN, 43% PN). Only 2.3% feeding time was lost due to complications (1% EN, 1.3% PN). In conclusion, better assessment of requirements, review of current regimens and more appropriate monitoring are required.

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Postoperative analgesia, stress response and urinary nitrogen excretion after upper abdominal surgery. By M.M. SHALABY[1,2], H.M. BASHA[1], M.I. RAMADAN[1], W.K. NARSHY[1] and I.T. CAMPBELL[2], *[1]Medical Research Institute, University of Alexandria, Alexandria, Egypt and [2]University Department of Anaesthesia, University Hospital of South Manchester, Withington, Manchester M20 8LR*

This present study was performed to compare directly the effects of three common postoperative analgesic techniques on the stress response and urinary N excretion after upper abdominal surgery.

Sixty fit adult patients undergoing elective upper abdominal surgery were randomly assigned to receive either intramuscular pethidine (IP group), epidural local anaesthesia (bupivacaine; EB group), or epidural opiate analgesia (fentanyl; EF group), on demand for postoperative pain relief over 48 h. Plasma cortisol, ß-endorphin and blood glucose were measured at 08.00 hours first postoperative day and 08.00 hours second postoperative day. Urea N excretion was measured on the day before the operation, the day of the operation and on the first and second postoperative days. Pain relief was assessed using a 0-100 visual analogue scale. Two-factors repeated measurements analysis of variance were used to test for significant differences between groups.

Pain relief was significantly better in both epidural groups than intramuscular pethidine ($P<0.01$) but with no difference between the two epidural groups. Both cortisol and ß-endorphin rose in all three groups intraoperatively and had returned towards normal concentrations by day 2; they were still significantly elevated in the IP group compared with both epidural groups ($P<0.01$), but with no difference between the epidural groups. Urinary urea N was similar in all three groups preoperatively. Pre- and postoperative urea N excretion values are given in the Table.

	24 h urinary urea N excretion (g)+			
	Preoperative	Operation	Day 1	Day 2
IP	9.73	11.7	11.85	11.65
	(7.55-11.9)	(9.56-13.8)	(9.85-13.9)	(10.1-13.2)
EB	10.1	9.39*	8.30*	8.7*
	(8.27-12.0)	(7.29-11.5)	(6.48-10.1)	(6.60-10.8)
EF	9.93	9.83*	8.23*	7.71*
	(8.47-11.4)	(8.19-11.5)	(7.03-9.43)	(6.59-8.83)

*$P<0.05$ compared with IP group. +Geometric mean +95% CI.

Epidural fentanyl and bupivacaine are equally effective in attenuating the "stress response" to major upper abdominal surgery and are better than intramuscular pethidine; both prevent the rise normally seen in urinary N excretion.

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Thiamin status of people with AIDS. By A.K. HARDIMAN[1], C. BALDWIN[2], A. POZNIAK[2] and P.W. EMERY[1], *[1]Department of Nutrition and Dietetics, King's College London, Campden Hill Road, London W8 7AH and [2]Department of Genitourinary Medicine, King's Healthcare, London SE5 9RS*

Deficiencies of micronutrients such as thiamin may contribute to morbidity in people with AIDS. There have been several case reports of autopsy findings characteristic of Wernicke's encephalopathy in people who have died from AIDS, and in an uncontrolled study Butterworth *et al.* (1991) found biochemical evidence of thiamin deficiency in nine out of thirty-nine people with AIDS and AIDS-related complex. We have therefore measured biochemical indices of thiamin status in twenty-four males with AIDS (as defined by the current Centers for Disease Control criteria) and in an age-matched group of 15 healthy men. Subjects were recruited from both inpatients and an outpatient clinic. Heavy drinkers were excluded, as were those who were too confused to give informed consent and those who were terminally ill. None of the subjects or controls was taking vitamin supplements.

The erythrocyte transketolase (EC 2.2.1.1; TK) activation coefficient was significantly higher in the subjects than the controls (subjects median 1.125 (range 1.029-1.408), controls median 1.091 (range 1.000-1.157); $P<0.01$ by Mann-Whitney test). Taking an activation coefficient of 1.2 as the upper limit of normal six of the subjects, but none of the controls, can be classified as thiamin deficient. This is very similar to the proportion identified by Butterworth *et al.* (1991) as thiamin deficient. Surprisingly, the basal erythrocyte TK activity was significantly higher in the subjects than the controls (subjects median 1.22 (range 0.65-1.98) units/g haemoglobin, controls median 0.82 (range 0.65-1.12) units/g haemoglobin; $P<0.01$ by Mann-Whitney test). This was partly due to low haemoglobin values among the subjects (subjects mean 115 (SE 2.7) g/l, controls mean 141 (SE 2.5) g/l; $P<0.01$ by Student's t test). TK activity expressed per ml blood showed no significant difference (subjects median 0.101 (range 0.046-0.134) units/ml, controls median 0.071 (range 0.060-0.099) units/ml; $P>0.05$ by Mann-Whitney test), although the distribution of values within the subject group was clearly bimodal. This confirms that basal TK acitivity is a less reliable indicator of thiamin status than the activation coefficient, particularly under pathological circumstances. Urinary thiamin excretion was not significantly different between the two groups (subjects mean 225 (SE 57) μg/g creatinine, controls 284 (SE 42) μg/g creatinine; $P>0.05$ by Student's t test), but interpretation of this variable is difficult because the subjects are likely to have lost muscle mass and hence their creatinine excretion would be lower than normal; unfortunately reliable 24 hour urine collections were not made.

Chronic thiamin deficiency normally affects the nervous system, so a simple questionnaire was devised to investigate the occurrence of neurological abnormalities amongst the subjects. The clinical notes were also examined for relevant entries. Almost all the subjects reported unusual tiredness or fatigue, and several also reported short-term memory loss or signs of ataxia or peripheral neuropathy. However, only anorexia and recent weight loss were significantly more common amongst the subjects who were biochemically thiamin deficient than in the subject group as a whole (anorexia was reported by 6/6 deficient subjects *v.* 14/24 in the whole group; recent weight loss was recorded in 6/6 deficient subjects *v.* 11/24 in the whole group; for both variables $P<0.05$ by chi-squared test).

In the present sample 25% of people with AIDS had inadequate thiamin status. This may be indicative of a wider problem in the HIV-positive population. In view of the pathology associated with thiamin deficiency routine screening for thiamin status may be appropriate, particularly in symptomatic patients.

Butterworth, R., Gaudreau, C., Vincelette, J., Bourgault, A-M., Lamothe, F. & Nutini, A-M. (1991). *Metabolic Brain Disease* **6**, 207-212.

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Docosahexaenoic acid (DHA) and visual function in infants with fat malabsorption secondary to liver disease. By C.H. SPRAY[1], S.V. BEATH[2], K.D. WILLIS[2], C. SUTTLE[3], V. TIPPER[3], I.W. BOOTH[2], G.A. HARDING[3] and D.A. KELLY[1]. *[1]The Liver Unit and [2]The Institute of Child Health, The Childrens Hospital, Birmingham B16 8ET and [3]Aston University, Birmingham,B4 7ET.*

DHA is a long chain polyunsaturated fatty acid which is present in high concentrations in retinal rod photoreceptors and is required in the normal development of visual pathways. Newborn babies with dietary deficiency of DHA have been shown to have impaired visual evoked responses and electroretinograms (Birch *et al.* 1992). However, it is not known if persisting biochemical DHA deficiency leads to long-term impairment of visual development, although it is now recommended that pre-term formula milks be supplemented with 1% of total formula fatty acid as DHA, as this approximates to DHA concentration found in breast milk (ESPGAN, 1991).

The aim of the present study was to determine whether infants with fat malabsorption secondary to liver disease had abnormal visual function and to evaluate possible factors such as deficiency of DHA, vitamin A, vitamin E, fat stores and hepatic dysfunction. Six infants age 1-15 months (M=4) were recruited.

Visual function was assessed by visual evoked potential (VEP) and electroretinograms (ERG) in dark- and light- adapted conditions. DHA concentrations in erythrocyte membranes were measured by gas-liquid chromatography, vitamins A and E were measured by HPLC. Body-fat stores were estimated by mid-arm fat area (MAFA) expressed as a z score. Plasma bilirubin was used as an index of severity of liver disease. Fat malabsorption was confirmed by measuring stool fat. All the infants received fat soluble vitamin supplementation routinely.

Subjects	DHA % fat	VEP abn/norm	ERG a-wave latency msec	MAFA z score	Bilirubin μmol/L
Reference range	5.2-7.1*		10-30	na	0-19
1	1.01	norm	35.7	1	22
2	1.05	abn	17.6	-2	445
3	1.17	abn	32.03	-1	234
4	1.40	abn	21.09	-1	142
5	1.53	abn	21.8	-2	46
6	1.54	norm	18.75	1	207
p (correlation with DHA)			0.2	0.9	0.6

* derived from breast fed infants (Makrides *et al.* 1993)

All infants had low red cell DHA concentrations. Only one child had completely normal visual. Two of the infants with the lower DHA concentration had abnormal rod function as indicated by the prolonged latency of the ERG a-wave. VEP was abnormal in 4 infants, but this may reflect an underlying encephalopathy. In this pilot study no clear relationship between; vitamin A and E levels, severity of liver disease and fat stores was found. However, DHA deficiency and abnormal rod photoreceptor function as measured by ERG appears to be common in infants with liver disease and further studies to assess the effect of DHA supplementation on visual function are in progress.

Birch E.E. Birch D.G. Hoffman D.R. and Uauy R. (1992) *Investigative Ophthalmology and Visual Science.* **33**,3242-53.

ESPGAN (1991) *Acta Paediatrica Scandinavia* **80**,887-96.

Makrides M. Simmer K. Goggin M. and Gibson R.A. (1993) *Pediatric Research* **33**,425-7.

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Measuring individual energy requirement in infants:inaccuracy of predictive equations and validation of heart rate monitoring. By M.A.THOMSON, S.BUCOLO AND R.W.SHEPHERD. *Children's Nutrition Research Centre, Royal Children's Hospital, Brisbane, Australia.*

Determination of energy and thereby overall fluid requirements in infants should preferably be determined by individual measurement of energy expenditure (EE) but has hitherto relied on measuring intakes which produce normal weight gain and hydration, which is retrospective and inaccurate. We have measured resting energy expenditure (REE) by indirect calorimetry and total energy expenditure (DLW-TEE) by doubly-labelled water methodology, in healthy infants and those with cystic fibrosis (CF) and evaluated the accuracy and reliability of predictive equations (Harris-Benedict, 1919, FAO/WHO/UNU, 1985, Schofield, 1985), and ambulatory heart rate monitoring (HR-EE). Predictive equations, when applied to the individual varied widely from the measured value [illegible] controls (n 36) by 106-132% and underestimating EE in CF (FAO/WHO/UNU, 84(SD11)%, Schofield, 86(SD11)%, n 9) except Harris-Benedict (152(SD63)% of measured). Measured REE closely correlated over a range of activities (r^2=0.99, P<0.0001), in controls (n 10, mean SD [illegible]). The EE was calculated from continuous heart-rate monitoring (22(SD2)h) [illegible] equations of HR and measured EE. HR-EE and DLW-TEE measurements showed a relative bias (mean difference) of 0.02 (SD 0.01) MJ/kg per 24 h. The reliability of the HR-EE method was unaffected by sex, age, disease state, or state of nutrition. Predictive equations should not be relied on to determine energy and fluid requirement. Heart-rate monitoring with individual HR-EE calibration lines is reliable and non-invasive and is deserving of wider application in determining individual requirements.

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Growth hormone and exogenous glucose utilisation rates in multiple organ failure. By CERI J. GREEN[1], ELLEN O'SULLIVAN[1], S. UNDERHILL[1], L.J. HIPKIN[2], D.P.M. MACLAREN[1] and I.T.CAMPBELL[1], *University Departments of [1]Anaesthesia and [2]Chemical Pathology, Royal Liverpool Hospital, Liverpool L69 3BX*

The normal response to injury and infection includes an increase in growth hormone (GH). In injury and infection there is also an increase in GH in response to glucose infusion, although a subgroup has been described which exhibits a relatively depressed GH response to glucose infusion, and has a higher mortality (Dahn *et al.* 1986). GH responses to a hyperglycaemic glucose clamp have been examined in patients suffering multiple organ failure (MOF).

Fourteen patients aged 18 to 76 (median 66)years were studied. At the time of the study APACHE II scores ranged from 11-32 (median 16). All patients were being artificially ventilated and fed intravenously. Feeding stopped at 18.00 hours the day before the study and all patients were studied between 09.30 and 14.00 hours. D-Glucose (20%) was infused to raise the blood glucose concentration to 12 mmol/l over 20 min where it was maintained (CV 6.3%) for a further 160 min by blood sampling and adjustment of the infusion rate at 5 min intervals in accordance with the algorithm of DeFronzo *et al.* (1979). GH concentrations were measured at 20 min intervals.

Two distinct groups were identified; seven patients in whom GH rose to the 20-50 mU/l range, designated hypersecretors, and seven in whom GH remained <10 mU/l, designated hyposecretors (Dahn *et al.* 1986). Exogenous glucose utilization rates (GUR) were calculated over 20 min epochs between 40 and 180 min. Exogenous GUR was significantly higher in the hypersecretors than the hyposecretors (P=0.022, ANOVA), though not at any specific time point. Exogenous GUR in the hypersecretors did not change over the course of the clamp averaging 34.6 (95%CI 25.3 - 43.7) ug/kg per min, whereas GUR in the hyposecretors decreased from 29.1(95%CI 51.5-16.4) to 19.2 (49.2 - 7.5) umol/kg per min (P=0.05).

There appears to be an association between the GH response to infused glucose in MOF and the ability to utilise exogenous glucose.

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Importance of measuring CO_2 production rate in ^{13}C breath tests. By S.AMARRI[1], W.A. COWARD[2], M. HARDING[2], and L.T. WEAVER[1], *[1]Department of Human Nutrition, University of Glasgow, Yorkhill Hospitals, Glasgow G3 8SJ and [2]MRC Dunn Nutrition Unit, Cambridge CB4 1XJ*

Stable isotope (^{13}C) breath tests offer a safe, repeatable method for nutritional and metabolic research. They involve the administration of a substrate labelled with ^{13}C followed by serial measurements of the ^{13}C:^{12}C ratio in expired CO_2, from which the percentage ^{13}C recovered (PDR) can be calculated. However the results depend on an accurate measure of CO_2 production rate. Many researchers use a standard value of 5 mmol/m^2 per min, derived from extrapolations of measurements in adults (Shreeve *et al.* 1976). The purpose of the present study was to compare results obtained using directly measured CO_2 production rates with those calculated from two predicted values (Shreeve *et al.* 1976 and Schofield, 1985). Ten normal (N) healthy children (age 7-12 years, two males and eight females), twenty-six children with cystic fibrosis (CF) (age 1-12 years, twelve males and fourteen females) and two children with other gastrointestinal diseases (age 5 and 10 years, one male and one female) were studied. The CF patients were studied more than once and the total number of tests performed was 55. After an overnight fast each subject ingested 10 mg/kg body weight of a mixed triacylglycerol (1,3-distearyl, 2[carboxyl-^{13}C]octanoyl glycerol). A baseline breath sample was collected followed by duplicate samples every 30 min for 6 h. ^{13}C sample enrichment was measured by isotope ratio mass spectrometry and the results were expressed as delta ‰ compared with an international standard (PeeDee Belemnite). These data were used to calculate PDR. The volume of CO_2 produced per min (VCO_2) was measured at rest by indirect calorimetry (Deltatrac, Datex Instrumentarium Corp., Helsinki, Finland) for 30 min approximately 3 h after substrate ingestion, and the results were converted to mmol/min. For each subject the expected BMR was calculated from the equation of Schofield (1985), based on weight and height, and these values were converted to CO_2 production rate using equations derived from Elia & Livesey (1988). Using these three CO_2 production rates three different PDR were calculated and compared. The results (median and range) are shown in the Table.

		Measured CO_2	CO_2 by Schofield	CO_2 by Shreeve et al.
CO_2 (mmol/m^2 per min)	N	5.08 (4.01-6.01)	5.14 (4.51-5.42)	5.00
	CF	6.66 (5.20-9.90)	5.62 (4.30-8.11)	5.00
Cumulative PDR (%)	N	29.16 (9.68-41.09)	30.25 (8.68-42.61)	30.56 (9.09-41.96)
	CF	19.80 (0.89-36.74)	16.94 (0.86-49.02)	15.55* (0.67-30.21)

*Significantly different from measured value, P = 0.02.

In healthy children there was a close accordance between measured and predicted CO_2 production rates, but children with CF had a median measured CO_2 production rate 31% higher than normal chidren. Use of predicted CO_2 production rates in children with CF underestimates cumulative PDR by up to 27%. If indirect calorimetry is not available or impossible to perform the VCO_2 obtained from the BMR calculated using the equations of Schofield (1985) or Shreeve *et al.* (1976) can be used in normal children. However CO_2 production rates should be measured when performing ^{13}C-breath tests in conditions where an altered energy expenditure and/or CO_2 production rate is expected, to obtain accurate results of PDR of ingested ^{13}C.

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Energy metabolism in patients treated by continuous ambulatory peritoneal dialysis (CAPD).

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Though protein-energy malnutrition has been found in up to 40% of CAPD patients, energy expenditure (EE) in CAPD patients has not been compared with healthy controls. CAPD patients receive a constant supply of glucose from the dialysate fluid. The impact of this energy load on fasting energy metabolism has not previously been defined.

In the present study EE in twelve stable CAPD patients was compared with that of eleven healthy controls. Studies were performed after an overnight fast with all subjects lying quietly for the study duration (4h). EE was measured using a Deltatrac metabolic monitor (Datex, Helsinki, Finland) at rest and, in the CAPD patients, . Following a baseline measurement, over 5 min periods every 15 min during and for 2h following an exchange of 2 litres of dialysate fluid containing glucose. Controls underwent EE measurements for the same time periods as the CAPD patients. Blood glucose was measured at 0, 2 and 4 h. Glucose absorption from the dialysis fluid was measured during the study dialysis and residual renal urea and creatinine clearance for the 24 h period before the study.

There was no significant difference between groups in respect to weight, height or age. A significant in blood glucose concentration (mmol/L) (4.7 to 4 to 3.6; $P = 0.0001$) was observed in the controls but not in the patients; (6.5 to 6.7 to 6.3). Comparison of energy metabolism in both groups is shown below.

	EE (kJ/24 h) Mean SD	EE % HB Mean SD	V02 (ml/min) Mean SD	RER Mean SD
CAPD	5460 520	0.94 0.08	190 17	0.84 [a] 0.02
CONTROL	5890 730	0.97 0.09	207 25	0.81 [b] 0.03

a,b Significantly different $P = 0.01$.

EE % HB, EE as a percentage of basal energy expenditure predicted from the Harris Benedict equation; V02, oxygen consumption; RER, Respiratory exchange ratio.

CAPD patients absorbed a mean 16(range 12-21)g glucose from the dialysis fluid, providing 251 (range 188-330)kJ over the final 2 h of the study. The RER in the controls decreased over time (0.85 to 0.79), $P = 0.0004$. No significant in RER was observed in the CAPD patients (0.85 to 0.86). No correlations were demonstrated between dialysis dose or residual renal function and energy expenditure in the CAPD patients.

In conclusion CAPD therapy has no significant influence on resting EE especially when this is normalized to basal EE. In addition there was no significant relationship between dialysis dose and energy metabolism. The significant reduction in RER in the control group probably reflects an increased reliance on fat metabolism with continued fasting. In contrast the glucose absorption from the dialysis fluid, which counteracts the reduction in blood glucose seen in fasting controls, appears to prevent such a change in substrate oxidation with preservation of the RER.

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Relationship between muscle thickness measured using ultrasound and fat-free mass measured with dual energy X-ray absorptiometry (DEXA). By D.J.WITHERS[1], J.E.ADAMS[1] and I.T.CAMPBELL[2], *Departments of [1]Radiology and [2]Anaesthesia, University of Manchester, Manchester M13 9PL.*

Patients suffering multiple organ failure (MOF) are often severely oedematous; this confounds all the common methods of assessing changes in body composition. It has been shown that virtually no oedema fluid is retained within the body of muscle in these circumstances (Helliwell *et al.* 1991) and this raised the possibility of monitoring muscle wasting in MOF using ultrasound. It was determined that the muscle thicknesses that correlated best with fat-free mass derived from body weight and skinfold thickness were those measured over the biceps, mid forearm and mid thigh (Watt *et al.* 1993), and measuring muscle thickness at these three sites it has proved possible to identify wasting in oedematous patients independently of oedema (Campbell *et al.* 1994). There is a need to validate the use of these three sites as an index of lean tissue mass using a different technique to assess fat-free mass.

Fat-free mass was measured using DEXA (Lunar DPX-L) in thirty-six patients (14M, 22F; aged 20-74 (median 53)years; weight 46.7-94.8 (median 64.2) kg; height 1.48-1.86 (median 1.635)m) undergoing the procedure for a variety of reasons. Muscle thickness was measured at the three sites using an ALOKA SSD 500 portable ultrasound machine with a 3.5MHz linear array probe.

The correlation coefficient of the sum of the three ultrasound measurements (biceps, forearm and thigh) with fat-free mass measured using DEXA was 0.872 ($P<0.0001$). The correlation coefficient of fat-free mass with DEXA and mid upper arm circumference was 0.494 ($P=0.002$) and for mid thigh circumference 0.332 ($0.1>P>0.05$). The correlation coefficient obtained in the earlier study, using skinfold measurements to derive fat-free mass, was 0.843.

These results confirm the validity of using muscle thickness measurements, made with ultrasound over the biceps, forearm and thigh, as an index of fat-free mass.

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Body composition and physiological measures of fitness during re-feeding of anorexia nervosa patients. By E.G.WALLER[1], A.J.WADE[1], J.TREASURE[2], A.WARD[2], T.LEONARD[2], and J.POWELL-TUCK[1], *[1]Departments of Human Nutrition and Sports Medicine, London Hospital Medical College, Turner Street, London E1 2AD and [2] The Institute of Psychiatry, Denmark Hill, London SE5 8AF*

Several lines of evidence suggest that undernutrition significantly impairs exercise capacity (Desai *et al.* 1984) and physiological function in muscle (Russell *et al.* 1983). On re-feeding, some characteristics of muscle function recover quickly, seen in these experimental studies of brief twitch or isometric contractions. However there is limited information available about the complete recovery of aerobic function in muscle working submaximally.

The present study therefore aimed to examine further the relationship between weight gain and changes in body composition and physiological characteristics of fitness during the recovery from severe anorexia nervosa (AN). Seven hospitalized female AN patients were studied over 9 weeks (weeks 0-8) during supervised re-feeding on a 12.5 MJ/d diet. Body composition was measured using weight, skinfolds (Durnin & Womersley, 1974) and dual energy X-ray absorptiometry (DEXA). Mean BMI (weight (kg) / height^2 (m^2)) value at week 0 was 14.6 (SD 1.3), rising to 18.2 (SD 1.9) at week 8, corresponding to a mean weight gain of 9.6kg (P<0.001). The proportion of this weight recorded as fat varied between the two methods. DEXA gave a body-fat gain of 6 kg (62%), however this was 5.2 kg (54%) when calculated from skinfolds. Changes in percentage body-fat over the 9 weeks as measured by skinfolds and DEXA were significant (P<0.001 and 0.01 respectively). Comparing results given by the two methods for measurement of body-fat showed a significant difference at week 0 (P<0.001) but not at week 8 (P=0.02).This methodological difference requires further investigation.

Peak expiratory flow rate (PEFR) was measured on weeks 0, 5 and 8 in all seven subjects. This increased with weight gain in six of the subjects (P =0.063 for the value of F found in ANOVA with repeat measures; from 303 (SD 89) to 379 (SD 54) litres/min). Several variables relating to aerobic work were also recorded weekly at rest and during cycle ergometry at very low loads (loads imposed 0, 20, 40, and 60 watts, each for 5 min, in a progressive work test). The ability to perform over the full range of workloads improved with recovery in six of the seven patients, although three never reached 60 watts. O_2 uptake increased with weight gain both at rest and at all loads, but was unchanged if expressed per unit body weight. O_2 pulse increased slightly but significantly between weeks 0 and 8 both at rest and at 0 watt load (2.07 (SD 0.61) to 2.73 (SD 0.49), and 3.4 (SD 0.8) to 4.27 (SD 0.75) ml/beat respectively, both P <0.05, n=6, using paired t - test. No similar analysis was possible at 20, 40, or 60 W as insufficient subjects reached these loads on week 0.

In conclusion, the woman who fails to pedal beyond 0 or 20W in this protocol, has a maximum O_2 pulse of less than 5 ml/beat, and has a PEFR below 300-330 litres/min, has very severely impaired aerobic function. Our experience suggests that this will start to improve with a weight gain of only a few kilograms, e.g. 2-3 kg, mainly achieved over an initial period of 3-4 weeks of re-feeding. On the other hand, unlike the complete recovery of muscle function characteristics reported by Russell *et al.* (1983) over 8 weeks, the poor O_2 pulse and performance capacity that we have observed was only slowly returning to normal. It seems evident that functional aerobic capacity will remain significantly impaired for a very much longer period in relation to that expected in a fit, normal-weight woman of the same age. Whether this is simply a question of restoring the wasted muscle mass, or is also related to functional characteristics, perhaps associated with the loss of hormonal (oestrogenic) stimuli in the amenorrheic woman (as has been investigated recently by Phillips *et al.* 1992) remains to be seen.

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Comparison of three simple methods for the detection of malnutrition.

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Malnutrition in hospital is often unrecognized. A nutrition team will teach simple methods to detect malnutrition. On a single day all medical in-patients underwent an assessment. Age, diagnosis and weight 3 months previously were recorded. Weight, height, triceps, skinfold thickness and mid-arm circumference were measured. A clinical examination for fluid retention was performed.

Eighty-four patients (forty-three men), median age 71 (range 28-97) years were assessed. The most common diagnoses were cardiac disease 26, stroke/dementia 12, non-malignant lung disease 9 and malignancy 6. Prevalence of malnutrition by three methods is shown in the table.

Method	assessed n (%)	malnourished n (%)
Percentage weight loss (%WL) >10%	65 (77)	17 (26)
Body mass index (BMI) <19 kg/m²	69 (82)	13 (19)
Mid-arm muscle circumference (MAMC) <5th percentile (Symreng, 1982)	83 (99)	16 (19)

Malnutrition was detected by all three methods in six patients. MAMC identified three who could not be weighed and three with fluid retention. %WL detected four patients who were overweight (BMI >25 kg/m²), yet had lost more than 10% body weight. Thirty-five (42%) patients had evidence of fluid retention; BMI and/or %WL detected malnutrition in six of these and MAMC detected an additional three. Using all three tests 29/84 (35%) patients were assessed as malnourished. BMI and %WL detected most patients but fluid retention may limit their accuracy. MAMC is good for those confined to bed, unable to be weighed or with fluid retention.

Symreng T. (1982). *Clinical Nutrition* 1, 211-219

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Measuring the height of critically ill patients in bed. By T. WATT[1,2], J. CLAYTON[1], M.A. KEEGAN[2] and I.T. CAMPBELL[1,2], *[1]Intensive Care Unit and [2]University Department of Anaesthesia, Withington Hospital, Manchester M20 8LR*

It is often necessary to know the height of a patient in order to express, for example, cardiac output per square metre of body surface area, or energy expenditure as a percentage of basal predicted from age, height and weight. A surveyor's measure has been designed to measure the height of supine individuals, but in a previous study it was shown to overestimate conventional and portable stadiometer measurements by a mean of 1.5 cm (Watt *et al.* 1994). It also compared poorly in accuracy with simply guessing the height, but it was more precise.

It is possible that the surveyors measure overestimated height because of the tendency for supine individuals to lie with their feet partially plantar flexed. The accuracy of the surveyors measure has been re-assessed but with all the individuals using it instructed specifically to measure the distance between the top of the head and the heels, thereby offsetting the problem of a plantar flexed foot producing a falsely high figure.

The heights of eight normal individuals were measured by seven volunteers all accustomed to guessing or measuring by one method or another the height of supine patients. All heights were measured using the conventional stadiometer (graduated in inches) with the subject standing and the surveyor's measure (graduated in cm) with the subject lying supine on a hospital bed. The volunteers were specifically instructed to place the surveyor's measure under the heel of the subject.

The mean difference between the stadiometer measurements and those observed with the surveyors measure was 0.5 cm; stadiometer 163.6 (SD 4.5) cm, surveyor's measure 164.1 (SD 4.7) cm ($P<0.05$).

It is concluded that the surveyor's measure overestimates height in the supine subject compared with a conventional stadiometer with the subject erect even when care is taken to avoid the effects of plantar flexion. The error however appears to be <0.5% and is predictable.

Watt, T., Dodd, F., Keegan, M. & Campbell, I.T. (1994). *Proceedings of the Nutrition Society* **53**, 67A.

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Visual analogue scales: a validated tool for assessing nutritional need in head and neck cancer patients. By C. MACQUEEN, and G. FROST, *Nutrition and Dietetic Department, Hammersmith Hospital, Du Cane Road, London W12 OHS*

The present study reports and validates the use of visual analogue scales as a screening tool to identify nutritional need in head and neck cancer patients.

Twenty patients with squamous cell carcinoma of the head and neck were assessed during their course of radical radiotherapy: before, midway and at the end of treatment, with a 2-month follow-up visit. At each visit anthropometry was measured on four sites. Patients were weighed and completed 3 d food diaries and the visual analogue scales.

The mean age of the group was 64 (range 48-81) years. Fourteen of the patients were male, six were female. Ten of the group had cancer in the larynx, ten in the pharyngeal region.

The visual analogue scales assessed the patients appetite, ability to take solids and liquids, and their sense of taste on a 5-point scale from very poor to very good. Each category followed a similar trend, decreasing at visits 2 and 3 but improving by visit 4. There was no significant difference in visual analogue scale scores between the larynx and pharynx groups at the start of radiotherapy. Neither was there any significant difference in whole group results between visits 1 and 3. However, there was a significant difference for each category in the pharynx group between visits 1 and 3. The difference between the two sub-groups at visit 3 was highly significant for each category. The scores are shown in the Table.

	Appetite Score	Solids Score	Liquids Score	Taste Score
Larynx				
Visit 1	4.4	4.6	4.8	4.3
Visit 3	4.6	4.3	4.5	4.3
Pharynx				
Visit 1	3.7	3.7	4.6	3.5
Visit 3	2.5*	1.7*	3.0*	1.9*

* Significantly different from visit 1 $P < 0.02$.

These scores were correlated with energy intake from food. A high score meant a high energy intake, and a low score meant a low energy intake. When compared using Pearson's coefficient, this correlation was stronger for solids (R 0.71, $P < 0.0001$) and appetite (R 0.65, $P < 0.0001$), than for liquids (R 0.59, $P < 0.0001$) and taste (R 0.54, $P < 0.0001$).

The visual analogue scales described are an easy-to-use, validated tool for screening a patient's need for nutritional support and hence dietetic referral.

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Diurnal variation of plasma glutamine and arginine in normal and fasting subjects. By L.M. CASTELL, C.T. LIU and E. A. NEWSHOLME. *University Department of Biochemistry, Oxford OX1 3QU.*

The plasma glutamine (pGln) and arginine (pArg) concentrations in humans vary widely in different conditions: in most normal humans the range for pGln is 500-700 μM; the mean [pGln] of nine athletes after a 10 h overnight fast was 632 (SE 17) μM , compared with 860 uM for a 10th athlete, who fasted for only 6 h after a very large meal. A bolus of glutamine (0.1 g/kg body weight) given to fasting healthy subjects can increase [pGln] to 1400 μM in 30 min. Stress such as starvation, burns or major surgery decreases [pGln], sometimes to as low as 300 μM (Parry-Billings *et al.* 1990). In healthy young humans, Castillo *et al.* (1994) reported [pArg] of 100-200 μM. After 3 d pre-fed with/without arginine, fasting [pArg] of 169 (SE 13) μM increased after a meal to 208 (SE 26) μM (arginine-rich diet), compared with 164 (SE 23) μM decreased to 142 (SE 28) μM (arginine-free diet).

In order to obtain further information on intra- and inter-individual variations, the diurnal variation of [pGln] and [pArg] was investigated in four healthy subjects during a normal day's activity. Ethical permission was obtained. Apart from a 10-12 h fast, no dietary restrictions were made. Samples were taken at 2-h intervals from about 07.15-21.30 hours. Subject A ingested no food or water from 02.30-18.00 hours. Subject B participated in two experiments: once eating normally during the day and once undertaking a 21 h fast (drinking only water). Subject D drank only black coffee for breakfast. Glutamine was measured enzymically; arginine was measured using a novel technique (Newsholme & Liu, 1994).

In all 3 non-fasting subjects (B, C and D) [pGln] increased 2hr after lunch by 22%, 15%, 29%, and after breakfast and dinner in two subjects: the largest increases occurred after a meal containing protein. In 2 subjects (B and C) [pArg] increased after lunch (27% and 12% respectively) but decreased in subject D by 15%. In subject A [pGln] increased by 10% at mid-day, returning to baseline 4 h later; [pArg] decreased by 15% in the morning, returned to baseline 2hr later and remained low until after dinner. During the 21-hr fast in subject B there was almost no change in [pGln]; [pArg] decreased by 22% in the morning, returned to baseline 2h later where it remained until after dinner. In subject D [pGln] decreased by 16% after three 300ml drinks of black, instant coffee; [pArg] decreased from baseline levels by 18% after lunch, and increased by 36% after dinner.

The results of the study show that plasma glutamine and arginine levels usually increased after eating, particularly when protein was consumed. This suggests that the small intestine does not consume all of the glutamine in a meal, and that food intake can influence the plasma concentration. These findings differ from those of other studies which report little or no change in plasma glutamine after a meal (Elia *et al.* 1988). In the intestine, arginine, together with some of the glutamate derived from glutamine, is converted to ornithine and then to citrulline: citrulline is converted to arginine in the kidney, from where it enters the plasma. In most subjects in the present study [pArg] increased 2 h after a meal: the results agree with those reported by Elia *et al.* (1988).

These findings emphasize the importance of consistency in timing when taking blood samples for plasma amino acid estimations from the same subjects on different days in, for example, clinical and/or longitudinal studies. It is also important to ensure that subjects adhere strictly to a 10 h fast when requested, and to obtain a brief dietary history whenever possible. Insufficient attention is often paid to these details when setting out protocols for experiments on human subjects.

We thank the subjects for their cheerful co-operation.

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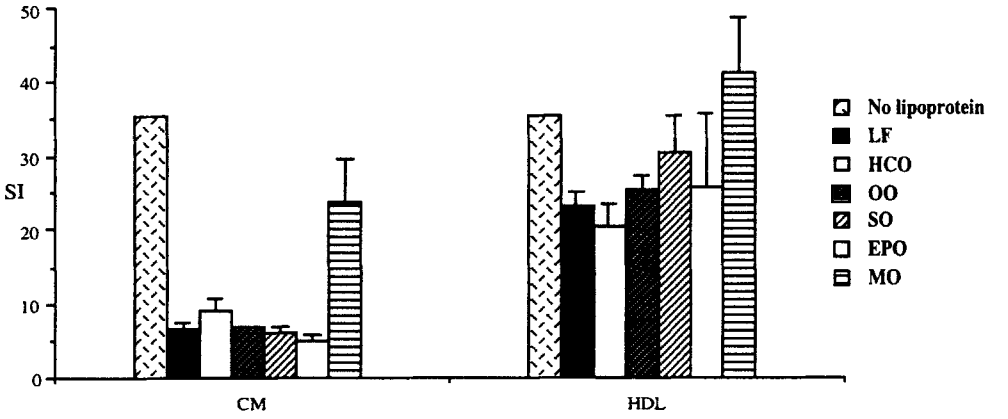
The effect of lipoproteins isolated from rats fed on different dietary lipids upon lymphocyte proliferation. By N.M. JEFFERY, P. YAQOOB, P.C. CALDER and E.A. NEWSHOLME, *Department of Biochemistry, University of Oxford, South Parks Road, Oxford OX1 3QU*

We have previously reported that feeding rats dietary lipid of differing fatty acid composition affects the concentrations of serum lipids (Yaqoob *et al.* 1993) and the composition of individual lipoprotein fractions (Jeffery *et al.* 1994). The *in vitro* proliferation of lymphocytes is also influenced by dietary lipid manipulation (Yaqoob *et al.* 1994). It is possible that circulating lipoproteins elicit an immunomodulatory effect. Therefore, the effect of lipoproteins upon spleen lymphocyte proliferation was investigated *in vitro*.

Male weanling Lewis rats were fed for 10 weeks on a low-fat diet (LF; 20 g/kg) or on high-fat diets containing 200 g/kg hydrogenated coconut oil (HCO), olive oil (OO), safflower oil (SO), evening primrose oil (EPO) or menhaden oil (MO). Chylomicrons (CM), very-low-density (VLDL), low-density (LDL) and high-density (HDL) lipoproteins were prepared from serum by ultracentrifugation. Spleen lymphocytes were isolated from rats fed on the LF diet and were cultured for 66 h in serum-free medium containing lipoprotein (or no lipoprotein in the control) and the T lymphocyte mitogen, concanavalin A (Con A; 0.25 μg/ml). The concentrations of CM and HDL used were 0.5% and 5% of their serum concentrations respectively. Preliminary experiments showed that these lipoprotein concentrations were optimal for lymphocyte proliferation under the conditions used in the present study. Lymphocyte proliferation was measured as [^{3}H]thymidine incorporation over the final 18 h of culture and is expressed as stimulation index (SI). Data are from four animals fed on each diet.



No effects of VLDL or LDL isolated from animals fed on the different diets upon lymphocyte proliferation were observed (results not shown). CM isolated from animals fed on each diet markedly suppressed lymphocyte proliferation compared with that of the control cells which were cultured in the absence of lipoprotein. CM from the rats fed on the MO diet were significantly less inhibitory than CM isolated from animals fed on the each of the other diets. HDL isolated from animals fed on the LF, HCO or OO diets suppressed lymphocyte proliferation compared with HDL from animals fed on the MO diet. Thus, this study shows that lipoproteins can affect lymphocyte proliferation *in vitro*, that different lipoproteins have different effects and that the observed effect is dependent upon the nature of the diet consumed by the animal from which the lipoproteins are obtained.

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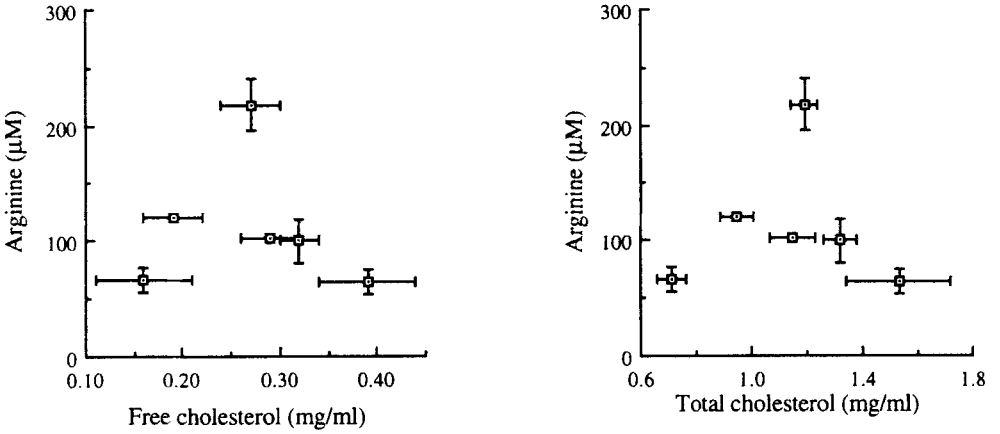
Serum cholesterol levels in rats fed on high-fat diets do not correlate with serum arginine levels. By C.T. LIU, P. YAQOOB, P.C. CALDER and E.A. NEWSHOLME. *Department of Biochemistry, University of Oxford, South Parks Road, Oxford OX1 3QU*

L-Arginine is the precursor of nitric oxide, also known as endothelium-derived relaxing factor, an important regulator of vascular tone in man and other mammals. Hypercholesterolaemia, which is a risk factor for development of atherogenesis, impairs endothelium-dependent relaxation. Experimental and clinical data have suggested that the dysfunction of the endothelium in hypercholesterolaemic subjects can be normalized by supplying arginine. Thus, Jeserich *et al.* (1992) hypothesized that this dysfunction was in part due to arginine deficiency. Rats fed with high-fat diets have been shown to have different serum cholesterol concentrations (Yaqoob *et al.* 1993). Therefore, an animal model was used to test the hypothesis of a link between arginine deficiency and hypercholesterolaemia.

Male weanling Lewis rats were fed for 10 weeks on a low fat diet (LF; 20 g/kg) or on high-fat diets containing 200 g/kg hydrogenated coconut oil (HCO), olive oil (OO), safflower oil (SO), evening primrose oil (EPO) or menhaden oil (MO). At slaughter, blood was collected and serum prepared. Values for free and total cholesterol concentrations are taken from Yaqoob *et al.*(1993). A novel method, based on the enzymic isotope-dilution principle, was used in the determination of arginine in this study (Newsholme & Liu, 1994). Values are means and standard errors from five animals fed on each diet.



The highest serum arginine level was found in rats fed on the SO diet and was about double the concentration of that in rats fed on the LF diet (control rats). The lowest serum arginine levels were found in rats fed on the OO or MO diets. In contrast, the highest serum cholesterol levels (both total and free cholesterol) were reported in rats fed on the OO diet, while the lowest total and free serum cholesterol level were observed in rats fed on the MO diet. We conclude that the serum cholesterol level in this animal model does not correlate with the levels of serum arginine.

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The effect of dietary lipid manipulation on macrophage phospholipid fatty acid composition and membrane fluidity. By E. J. SHERRINGTON, D. J. HARVEY and P.C. CALDER, *Department of Biochemistry, University of Oxford, South Parks Road, Oxford OX1 3QU*

Macrophages are important cells of the immune system which perform a number of vital functions many of which involve the plasma membrane e.g. phagocytosis, secretion, adhesion and receptor expression. Thus, changing the properties of the macrophage plasma membrane could affect the function of these cells. The aim of the present study was to determine the effect of dietary lipid manipulation on the fatty acid composition and plasma membrane fluidity of the macrophage.

Male weanling rats were fed for 10 weeks on a low-fat (20 g/kg) diet (LF) or on diets containing 200 g/kg hydrogenated coconut oil (HCO; rich in saturated fatty acids), olive oil (OO; rich in oleic acid 18:1 *n*-9), safflower oil (SO; rich in linoleic acid 18:2 *n*-6), evening primrose oil (EPO; containing *n*-6 PUFA γ-linolenic acid 18:3 *n*-6) or menhaden oil (MO; containing 20:5 *n*-3 and 22:6 *n*-3). Four days before slaughter, the animals were injected intraperitoneally with thioglycollate to elicit macrophage migration to the peritoneal cavity. Immediately after slaughter, macrophages were collected from the peritoneal cavity. The plasma membrane fluidity was determined by electron spin resonance (ESR) spectroscopy at 310 K, using 5-DOXYL-stearic acid as the ESR probe. The effective order parameter (S_{eff}), which is a measure of the average motion of the acyl chain of the fatty acid within the membrane, was obtained from the ESR spectrum. Lipid was extracted and the phospholipids were separated from the neutral lipid and saponified. The fatty acid composition of the phospholipid was determined by gas chromatography of the fatty acid methyl esters. Results are means from at least three animals fed on each diet.

	S_{eff}		Fatty acid (mol %)												
Diet	Mean	SEM	10:0	12:0	14:0	16:0	16:1 *n*-7	17:0	18:0	18:1 *n*-9	18:2 *n*-6	18:3 *n*-6	20:4 *n*-6	20:5 *n*-3	22:6 *n*-3
LF	0.54	0.01	n.d.	7.4	9.6	35.1	2.1	4.1	17.1	12.4	3.2	6.1	7.6	n.d.	n.d.
HCO	0.51	0.02	2.4	5.3	7.5	30.0	2.6	n.d.	17.5	13.2	5.3	8.2	8.8	n.d.	n.d.
SO	0.56	0.03	1.3	5.2	7.2	30.6	2.5	1.1	19.3	10.1	10.8	6.9	7.2	n.d.	n.d.
OO	0.50	0.04	1.4	4.8	8.2	31.1	1.6	1.6	19.2	13.8	7.0	7.4	7.4	n.d.	n.d.
EPO	0.49	0.02	2.4	3.9	7.5	33.1	1.9	2.4	19.2	11.2	7.2	6.0	5.9	n.d.	n.d.
MO	0.53	0.02	0.7	2.0	5.7	29.1	1.1	1.8	16.7	9.2	4.6	3.8	2.5	2.7	3.5

n.d. indicates not detected

The phospholipid fatty acid composition was affected by dietary lipid manipulation. For example, the phospholipid of macrophages from SO-fed animals had an increased proportion of 18:2 *n*-6 compared with that from animals fed on the other diets. Furthermore, the phospholipid from macrophages of animals fed on the MO diet had decreased proportions of 18:3 *n*-6 and 20:4 *n*-6, and 20:5 *n*-3 and 22:6 *n*-3 were detected in significant proportions. Despite these changes in fatty acid composition there was no difference in the S_{eff} of macrophages isolated from animals fed on the different diets.

Thus, the observed changes in the fatty acid composition of macrophage phospholipid did not result in a change in membrane fluidity, as assessed by ESR spectroscopy. Similar results were found by Lee *et al.* (1985), studying blood monocytes isolated from humans supplemented with MaxEPA capsules (containing 20:5 *n*-3) for 6 weeks. They found an increase in 20:5 *n*-3 in the phospholipid of the monocytes but found no change in membrane fluidity assessed by a fluorescent probe technique. Yaqoob *et al.* (1995) noted that fatty acids supplied to cultured macrophages were preferentially incorporated into intracellular triacylglycerol rather than the phospholipid. In the present study the neutral lipid fatty acid composition of macrophages was more significantly altered by dietary manipulation than the phospholipid fatty acid composition (unpublished results). It may be important for macrophages to maintain a precise level of plasma membrane fluidity. Therefore, exogenously supplied fatty acids are not substantially incorporated into the phospholipid and instead are stored as lipid intracellularly.

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Glutaminase (*EC* 3.5.1.2) and glutamine synthetase (*EC* 6.3.1.2) activities in the gastrointestinal tract of rats. By L.A. JAMES, P.G. LUNN, G. JENNINGS and M. ELIA, *MRC Dunn Nutrition Centre, Cambridge, CB4 1XJ.*

Glutamine is a major fuel for the gastrointestinal (GI) tract, but information about the extent of its metabolism and the distribution of key enzymes involved in its synthesis (glutamine synthetase) and degradation (glutaminase) is incomplete. Enzyme distribution studies can provide insights into glutamine metabolism which are not possible from arterio-venous catheterization studies. The present study aimed to establish the distribution of glutamine synthetase and glutaminase in mucosal and non-mucosal layers of different parts of the rat GI tract, including mouth and oesophagus for which there appears to be no available data.

Male hooded rats, 5-6 weeks of age, 150-200 g, were maintained on a standard diet. Enzyme activities were determined by radiochemical techniques based on the conversion of [^{14}C]glutamine to [^{14}C]glutamate and vice versa. Separation of glutamine and glutamate following incubation was achieved using AG - 1X8 Dowex anion exchange resin.

The Table shows the activity of mucosal scrapings from the small intestine, colon and stomach, the surface layer of the mouth, and whole oesophagus for which mucosal scrapings were not possible.

	Glutamine synthetase (nmol glutamine/min per mg protein)			Glutaminase (nmol glutamate/min per mg protein)		
Tissue	n	Mean	SEM	n	Mean	SEM
Mouth	7	1.24	0.06	7	20.10	2.30
Oesophagus	7	0.85	0.04	7	23.00	2.40
Upper stomach	4	3.75	1.66	7	45.40	5.90
Lower stomach	7	35.43	3.98	7	27.10	2.90
Small intestine	20	0.34	0.02	21	140.40	12.80
Colon	21	4.31	0.30	21	37.46	3.54

The mucosa of the small intestine had the highest glutaminase activity (duodenum, 168.5 > ileum, 132.9 > jejunum, 95.3 nmol glutamate formed/min per mg protein); the mucosa of the stomach and colon intermediate activities, (ascending, 55.5 > transverse, 30.5 > descending colon, 26.4 nmol glutamate formed/min per mg protein); and the mouth and oesophagus the lowest. Glutaminase activity in the non-mucosal layers ranged from 20.1-41.0 nmol glutamate formed/min per mg protein in all parts of the GI tract examined. Glutamine synthetase activity was low in the mucosal (0.34-4.31 nmol glutamine formed/min per mg protein) and non-mucosal layers (0.34-0.84 nmol glutamine formed/min per mg protein) of all parts of the GI tract except the stomach (35.4 and 34.2 nmol glutamine formed/min per mg protein, in the mucosal and non-mucosal layers). This data, which provides the most complete information on the distribution of glutaminase and glutamine synthetase activity in the GI tract of the rat, suggests (1) the mucosal and non-mucosal layers of the lower stomach have substantial potential for synthesizing glutamine, in contrast to the rest of the GI tract, which has little such potential; (2) the potential capacity for glutamine metabolism in the mouth and oesophagus appears to be limited; (3) the mucosa of the small intestine, which has the highest capacity for glutamine metabolism, derives its glutamine from external sources.

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Requirement for both glutamine and arginine by proliferating lymphocytes. By P.C. CALDER, *Department of Biochemistry, University of Oxford, South Parks Road, Oxford OX1 3QU*

Glutamine is necessary for the optimal function of cells of the immune system tested *in vitro* (see review by Calder, 1994) and dietary arginine has been shown to enhance immune functions (see review by Barbul, 1986). In recent years there has been much interest in the provision of glutamine and arginine to subjects in a variety of clinical settings, including those associated with immunosuppression. However, there is little information on the interactions between these two apparently "essential" amino acids.

Rat lymph-node lymphocytes were prepared by standard procedures and were cultured under standard conditions in arginine- and glutamine-free RPMI medium supplemented with 100 ml/l fetal calf serum, antibiotics, 5 μg/ml concanavalin A (a T-lymphocyte mitogen) and various concentrations of arginine and/or glutamine. The cells were cultured for 66 h, with [^{3}H]thymidine being present for the final 18 h; thymidine incorporation is a measure of lymphocyte proliferation. Results are mean thymidine incorporation from six determinations.

	[Arginine] (mM)											
	0		0.01		0.05		0.1		0.5		1	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
No glutamine	105	8	96	12	122	55	67	25	101	12	100	12
2 mM glutamine	618	19	3531	524	36014	6756	39192	5686	37592	3734	41296	2383

	[Glutamine] (mM)											
	0		0.01		0.05		0.1		0.5		2	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
No arginine	100	6	132	35	474	30	648	62	551	48	569	171
1 mM arginine	107	37	166	27	521	55	952	150	13580	2951	31816	3896

In the absence of glutamine, arginine cannot promote lymphocyte proliferation. In contrast, in the absence of arginine, glutamine is able to support sub-optimal lymphocyte proliferation; there is a six-fold increase in proliferation attained at a glutamine concentration of 0.1 mM. In the presence of 1 mM arginine, lymphocyte proliferation is dependent upon glutamine concentration; there is a nine-fold enhancement of proliferation at a glutamine concentration of 0.1 mM while at 2 mM glutamine proliferation is enhanced almost 300-fold. In the presence of 2 mM glutamine, lymphocyte proliferation is dependent upon arginine concentration; optimal proliferation (an enhancement of almost 70-fold) is attained at an arginine concentration of 0.05 mM. These results indicate that at the normal physiological concentrations of arginine and glutamine in the circulation (0.15 mM and 1 mM in the rat respectively) optimal lymphocyte proliferation will probably occur, but that if the concentration of either amino acid is decreased, then lymphocyte function could be compromised. Furthermore, it seems that the provision of both amino acids in combination, rather than alone, may be a more effective means of restoring immune function in immunocompromised patients.

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Influence of cell culture conditions on diet-induced changes in lymphocyte fatty acid composition. By P. YAQOOB and P. C. CALDER, *Department of Biochemistry, University of Oxford, South Parks Road, Oxford OX1 3QU*

We have previously reported that dietary lipid manipulation can affect rat lymphocyte proliferation and that this effect can be totally or partially masked by culturing the cells in the presence of fetal calf serum (FCS) rather than autologous serum (Yaqoob *et al.* 1994*b*). The present study investigates the influence of cell culture conditions on changes in lymphocyte fatty acid composition introduced by dietary lipid manipulation (see Yaqoob *et al.* 1994*a*).

Weanling Lewis rats were fed for 10 weeks on a low-fat (LF; 20 g/kg) diet or on high-fat diets containing 200 g/kg of either hydrogenated coconut oil (HCO), olive oil (OO), safflower oil (SO), evening primrose oil (EPO) or menhaden oil (MO). Lymphocytes were prepared from spleen and cultured for 48 h in the presence of autologous serum, FCS or a serum-free supplement (Nutridoma-SP) and 5 μg/ml concanavalin A (Con A). At the end of the culture period the cells were washed with phosphate-buffered saline, the lipid was extracted and the fatty acid composition was analysed by gas chromatography. Values are means with their standard errors for three animals fed on each diet.

Diet	Culture condition	Fatty acid (mol %)											
		16:0		16:1*n*-7		18:0		18:1*n*-9		18:2*n*-6		20:4*n*-6	
		Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
LF	Autologous	33.6	1.3	3.4	0.1	22.2	0.6	20.6	0.2	7.8	0.6	3.5	0.7
	FCS	34.3	1.0	3.5	0.3	22.2	1.8	19.4	1.4	2.1	0.3	1.7	0.3
	Serum-free	30.2	2.4	8.0	1.1	17.5	0.0	26.5	2.1	2.6	0.9	2.8	1.3
HCO	Autologous	29.2	0.5	2.9	0.5	28.0	1.5	17.7	1.8	5.1	0.5	6.0	0.8
	FCS	36.4	0.2	3.7	0.6	22.7	0.4	21.7	1.1	1.4	0.3	1.3	0.4
	Serum-free	29.2	0.8	9.5	0.2	18.1	0.4	30.1	0.6	1.7	0.3	2.3	0.8
OO	Autologous	35.8	1.6	2.3	0.2	25.5	2.0	21.7	0.4	3.0	0.5	2.8	1.0
	FCS	35.2	0.9	2.4	0.2	23.0	2.1	20.0	2.0	2.0	0.1	1.2	0.1
	Serum-free	31.0	0.9	7.8	0.8	15.6	0.6	30.1	0.2	1.5	0.5	1.8	0.8
SO	Autologous	34.9	4.0	2.2	0.1	26.8	2.7	14.1	1.9	5.4	1.2	2.8	0.4
	FCS	35.5	2.7	3.9	1.3	20.3	0.8	20.0	0.7	2.0	0.3	2.1	0.4
	Serum-free	33.8	2.4	6.7	1.3	14.6	2.3	24.4	0.3	3.1	1.0	2.4	0.8
EPO	Autologous	40.7	1.0	1.5	0.1	20.4	1.0	21.8	2.8	5.3	0.2	1.1	0.1
	FCS	38.8	0.8	3.2	0.2	25.8	0.9	20.8	1.0	3.0	0.2	1.2	0.3
	Serum-free	35.7	3.0	6.6	1.3	8.8	0.3	33.8	0.6	2.4	0.0	1.5	1.1
MO	Autologous	41.8	0.7	3.8	0.4	26.2	0.3	15.4	1.5	1.8	0.2	0.9	0.2
	FCS	38.1	1.1	4.2	0.1	24.0	0.5	19.6	2.3	1.1	0.6	0.6	0.2
	Serum-free	33.3	2.4	4.6	2.6	18.0	1.1	25.9	3.2	1.6	0.6	0.7	0.4

Culturing lymphocytes in autologous serum allowed some, but not all, of the diet-induced changes in fatty acid composition to be maintained. In particular, lymphocytes from rats fed on the OO or MO diets maintained very low proportions of linoleic and arachidonic acids. The effects of dietary lipid manipulation were totally reversed when lymphocytes were cultured in FCS. This is partly because FCS itself contains lipid which is different from that present in the diets and partly because stimulation with Con A brings about some distinct changes in fatty acid composition, which may override the effects of the diets; in particular, mitogenic stimulation is associated with a decrease in palmitic, stearic, linoleic and arachidonic acids and an increase in oleic acid (Calder *et al.* 1994). In addition, it appears that culturing lymphocytes in serum-free medium not only reverses any effects of dietary manipulation, but also markedly increases the appearance of palmitoleic and oleic acids, at the expense of palmitic and stearic acids, suggesting activation of the Δ^9 desaturase when these cells are cultured in the absence of exogenous lipid.

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The effect of dietary fatty acid composition on postprandial glycogen and lipid synthesis in tumour-bearing rats. By P.M.C. HO, O.A. OBEID and P.W. EMERY, *Department of Nutrition and Dietetics, King's College London, Campden Hill Road, London W8 7AH*

We have previously observed that the rate of hepatic glycogen synthesis following a standard meal was almost twice as great in cachectic tumour-bearing rats as in normal controls (Emery *et al.* 1993). In view of the growing body of evidence that dietary fatty acid composition can modulate the metabolic response to inflammatory stimuli we have now investigated the effect of different dietary fats on postprandial glycogenesis.

Groups of eight male Fischer 344 rats were fed for 21 d following subcutaneous injection of Leydig cell tumour cells (TB), or sham injections (C), on semisynthetic diets containing 100 g maize oil/Kg or 100 g lard/Kg as the sole source of fat. They were then fasted overnight and given a 16 KJ liquid meal (of the same composition as their previous diet) by oral administration together with an intraperitoneal injection of ^3H_2O. The rats were killed 1 hour later and the livers and epididymal fat pads (EFP) were analysed for ^3H incorporation into glycogen and saponifiable lipid.

	Maize Oil		Lard		
	TB	C	TB	C	SEM
Hepatic glycogenesis†	43.5**	21.7	24.8	27.0	4.8
Hepatic glycogen content (mg/g)	15.2	13.6	12.6	15.3	1.1
Hepatic lipogenesis‡	9.6*	11.3	9.4*	11.5	0.8
EFP lipogenesis‡	4.7	4.2	4.8	4.3	0.8

Significantly different from control group fed on same diet; * $P<0.05$, ** $P<0.01$.
† μmol ^3H_2O incorporated into glycogen/g tissue per h. ‡ μmol ^3H_2O incorporated into saponifiable lipid/g tissue per h.

In the maize-oil-fed rats the rate of incorporation of isotope into glycogen was increased by tumour growth, in line with our previous observations, but this increase did not occur in the rats fed on lard. However, liver glycogen content was not significantly increased in the tumour-bearing rats on either dietary treatment, suggesting that the main effect of tumour growth in the rats fed on the maize oil diet was a switch to the indirect pathway of glycogen synthesis involving increased gluconeogenesis. Hepatic lipogenesis was suppressed to a similar degree in both groups of tumour-bearing rats, while adipose tissue lipogenesis was not significantly different between any of the groups. Thus, although there was no significant difference in tumour growth rate, body weight or food intake between the two groups of tumour bearing rats, it appears that the expression of some of the metabolic changes which occur in response to tumour growth is dependent on qualitative aspects of dietary fat intake.

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