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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

A meeting of the Nutrition Society was held at St Angela's College for Home Economics, Lough Gill, Sligo, Republic of Ireland, on Thursday and Friday, 6/7 September 1990, when the following papers were presented.

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Validation of energy and protein intakes assessed by diet history and weighed records against energy expenditure and 24 h urinary nitrogen excretion. By ALISON E. BLACK, SUSAN A. JEBB and SHEILA A. BINGHAM, *MRC Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge CB2 1QL*

Eleven successful slimmers (one male, ten female) kept weighed diet records for 21 d using both traditional scales (WI) and the PETRA system of recording weight and food description on cassette tape; the changeover was on day 11. Subjects were weighed on days 1 and 21. Two diet histories were taken by different dietitians (DHA, DHB), one before and one after the study; the technique was not standardized. Energy (EI) and nitrogen (NI) intakes were calculated from food tables. Total energy expenditure (TEE) was measured by the doubly-labelled water method (Coward, 1988). Five 24 h urine collections validated by PABACHEK (Bingham & Cummings, 1985) were obtained to measure urine N; seven individual collections with PABA recovery <85% were rejected.

Multiple regression analysis of WI and PETRA intakes showed no effect of survey method or order of survey, but significantly higher intakes on Friday to Sunday; mean intakes were adjusted to allow for variation in days of the week covered. The Table shows the measured TEE and 24 h N, also EI and NI expressed as ratios of these values. The data were examined by paired *t* tests.

Mean (SD) intakes from WI (7·10 (3·16) MJ, 10·3 (3·7) gN) and PETRA (7·22 (2·87) MJ, 10·4 (3·9) gN) were not significantly different. DHA estimated mean intakes lower (6·45 (2·66) MJ, 10·1 (3·2) gN) and DHB higher (7·79 (2·42) MJ, 11·9 (3·4) gN) than weighed methods, but not significantly. Differences between DHA and DHB just failed to reach significance at 5% for energy, but the results do suggest observer bias in the diet history method.

	ΔWt	TEE	EI/TEE				Urine N	NI/1·25 × urine N			
Subject	(kg)	(MJ/d)	WI	PETRA	DHA	DHB	(g/d)	WI	PETRA	DHA	DHB
CH	−0·30						9·72	0·46	0·30	0·61	0·51
EW	−0·40	7·75	0·47	0·46	0·38	0·70	12·78	0·56	0·39	0·45	0·69
EL	−2·20	10·30	0·68	0·56	0·56	0·62	11·02	0·54	0·64	0·65	0·87
RP	−0·30	11·14	0·61	0·73	0·42		16·43	0·72	0·77	0·49	
BP	−2·20	9·21	0·58	0·74	0·48	0·55	9·01	0·79	0·78	0·72	0·65
SD	−0·80	9·82	0·80	0·54	0·64	0·80	10·16	0·76	0·68	0·91	1·02
PC	−0·60	9·64	0·77	0·83	0·64	1·04	9·63	0·85	0·84	0·75	1·11
CD	+0·50	9·31	0·92	1·02	0·86	0·96	10·60	0·83	1·02	0·73	1·14
PG	−1·60	13·72	0·95	0·95	0·98	0·91	15·51	0·99	0·84	0·98	0·93
DE	−0·60	8·72	0·95	0·88	0·79		8·76	0·86	0·96	0·92	
SJ	+0·20	7·63	0·61	1·14		1·15	7·98	0·82	1·18		1·14
Mean	−0·75	9·72	0·74	0·79	0·64	0·84	11·06	0·75	0·76	0·72	0·90
SD	0·89	1·77	0·17	0·22	0·20	0·21	2·75	0·16	0·26	0·18	0·23

Assuming stable weight, EI/TEE should equal 1·00 (95% confidence limits) (CL 0·71–1·29). Urine N is 80% of intake (Bingham & Cummings, 1985), thus, in N balance, NI/1·25 × urine N should equal 1·00 (95% CL 0·74–1·26). All methods gave mean ratios significantly lower than expected values, indicating underestimation of food intake. Energy and N were underestimated to a similar extent. Weight change (Δ Wt), mean −0·75 (+0·2 to −2·2) kg suggested both dieting and under-reporting. Within-subjects all methods tended to give similar results, suggesting that personal factors strongly influence 'cooperation'.

Bingham, S. A. & Cummings, J. H. (1985). *American Journal of Clinical Nutrition* **42**, 1276–1289.
Coward, W. A. (1988). *Proceedings of the Nutrition Society* **47**, 209–218.

No evidence of lower energy expenditure in post-obese women. By G. R. GOLDBERG, A. E. BLACK, A. M. PRENTICE and W. A. COWARD, *MRC Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge CB2 1QL*

The post-obese (PO) have lower absolute energy requirements compared with their obese state because of the change in weight. Many claim that they have to consume a lower energy intake (EI) to maintain the same weight as those who have never been obese. A study by Geissler *et al.* (1987) suggested that EI and energy expenditure (EE) were lower in PO women and McNeill *et al.* (1990) found a lower EI, but no significant differences in EE, between lean and PO subjects. Black *et al.* (1990) presented EI values from PO subjects and on the basis of these alone, energy requirements would appear to be low. However, when compared with total free-living EE (TEE) measured by doubly-labelled water (DLW), it was evident that the intakes of some of these subjects were under-reported.

In the present paper we analyse data from nine weight-stable (mean 18 months) PO women (mean self-reported weight loss 27 kg) who had EE measured by whole-body indirect calorimetry and DLW. The data are compared with those obtained from two age- and height-matched groups of nine lean (L) and nine obese (OB) women who, although participating in different studies, underwent identical protocols with respect to calorimeter and DLW measurements. Mean (SD) ages and heights of the PO, L and OB groups were 33·7 (8·9), 33·3 (5·2) and 34·9 (5·3) years; 1·65 (0·08), 1·65 (0·08) and 1·63 (0·03) respectively. Values for body composition and EE are shown in the Table.

	PO		L		OB	
	Mean	SD	Mean	SD	Mean	SD
Wt (kg)	64·6	8·2	58·5	7·8	87·9***†††	14·3
FFM‡ (kg)	44·5	5·3	42·4	4·6	49·1*	5·2
BMI (kg/m^2)	23·8*	2·5	21·4	1·6	32·9***†††	4·6
BMR (kJ/d)	5867	596	5815	369	6710***†††	611
BMR/kg FFM (kJ/kg per d)	132·9	15·6	138·0	9·0	137·0	9·0
24 h EE (kJ/d)	7644	713	7634	626	8967***†††	886
TEE (kJ/d)	9330	1083	8973	1419	10298	1398
TEE−BMR (kJ/d)	3463	671	3157	1203	3588	1167

FFM, fat-free mass.
Significantly different from L: *P<0·05, ***P<0·001. Significantly different from PO: †††P<0·001.
‡ Calculated by isotope dilution.

Data were analysed by t tests. With the exception of body mass index (BMI) all significant differences were between L and OB or PO and OB groups. There were no significant differences between L and PO in any variables, including 24 h EE and TEE expressed as multiples of basal metabolic rate (BMR) measured under standard conditions as in Goldberg *et al.* (1988).

These results do not support the contention that absolute EE or EE corrected for weight, fat-free mass or BMR are lower in PO compared with lean subjects.

Black, A. E., Jebb, S. A. & Bingham, S. A. (1990). *Proceedings of the Nutrition Society* (This Meeting).
Geissler, C. A., Miller, D. S. & Shah, M. (1987). *American Journal of Clinical Nutrition* **45**, 914–920.
Goldberg, G. R., Prentice, A. M., Davies, H. L. & Murgatroyd, P. R. (1988). *European Journal of Clinical Nutrition* **42**, 137–144.
McNeill, G., Bukkens, S. G. F., Morrison, D. C. & Smith, J. S. (1990). *Proceedings of the Nutrition Society* **49**, 14A.

Metabolic and behavioural responses to altered energy intake in man. 1. Experimental overfeeding. By E. DIAZ, A. M. PRENTICE, G. R. GOLDBERG, P. R. MURGATROYD and W. A. COWARD, *MRC Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge CB2 1QL*

Potential adaptive responses to overeating were investigated by measuring all components of the energy balance equation during forced overfeeding. The normal energy needs of nine healthy young men (six lean, three overweight) were determined by assessing metabolizable energy intake (ME) and total energy expenditure (TEE by doubly-labelled water (DLW)) during a 3-week weight-stable baseline period. They were then overfed a mixed diet (13% protein, 42% fat, 45% carbohydrate by energy) at 150% of baseline for 6 weeks. A 6-week free-diet period (FD 1) followed the overfeeding period. Subjects slept and ate in a long-stay metabolic suite throughout the experiment, but were otherwise unrestricted. Changes in body composition were assessed by densitometry and deuterium dilution. Metabolic responses were assessed at the end of each period by whole-body calorimetry which yielded data on basal metabolic rate (BMR), 24 h energy expenditure on a fixed protocol (CAL EE), and 24 h activity-plus-thermogenesis (CAL A&T derived as CAL EE–BMR). Combined metabolic and behavioural responses were assessed during the final 2 weeks for each period by DLW which provided data on free-living TEE and free-living activity-plus-thermogenesis (DLW A&T derived as TEE–BMR).

	Baseline (B)		Overfeeding (O)		FD 1		O minus B		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	P^*
Energy intake[1]	13·3	1·3	19·5	2·0	—	—	6·2	1·9	<0·001
Body mass[2]	73·7	9·5	81·4	9·6	76·6	8·5	7·6	1·6	<0·001
FFM[2]	57·4	4·0	60·7†	4·2	58·7†	3·4	3·0†	0·9	<0·001
Fat mass[2]	16·3	7·0	20·3†	7·4	17·6†	6·8	4·6†	2·1	<0·001
BMR[1]	7·3	0·5	8·2	0·3	7·3	0·4	0·9	0·4	<0·001
BMR[3]	128	9	137†	9	125†	7	9†	10	<0·05
CAL EE[1]	10·6	0·6	12·4	0·6	10·6	0·6	1·8	0·5	<0·001
CAL EE[4]	146	17	154	15	140	16	9	4	<0·001
CAL A&T[1]	3·3	0·2	4·2†	0·4	3·4†	0·3	0·9†	0·4	<0·001
TEE[1]	13·4	1·3	14·8†	1·6	12·5†	1·8	1·4†	2·0	NS
TEE[4]	187	35	186†	35	167†	34	−1†	25	NS
DLW A&T[1]	6·1	1·5	6·6†	1·4	5·4†	1·6	0·5†	1·8	NS

[1]MJ/d; [2]kg; [3]kJ/kg FFM per d; [4]kJ/kg per d; FFM, fat-free mass; NS, not significant.

* Significance tested by paired *t* test between baseline and overfeeding.

† *n* 8 due to missing data.

The rise in BMR only dissipated 14·5% of the energy overload, and the maximum amount dissipated by thermogenesis was 14·5% (CAL A&T) or 8·1% (DLW A&T) giving a total of 29%. The remaining 71% caused a virtually linear 7·6 kg weight gain (39% fat-free mass, 61% fat) at an energy cost of 24 kJ/g. Increases in energy expenditure were almost entirely explainable by changes in body weight and fat-free mass, and by the anticipated increase in diet-induced thermogenesis associated with the extra 6·2 MJ/d. There were no significant differences between lean and obese subjects. This study found little evidence of any active metabolic or behavioural adaptive processes which may help to maintain energy balance when people overeat.

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Metabolic and behavioural responses to altered energy intake in man. 2. Experimental underfeeding. By E. DIAZ, A. M. PRENTICE, G. R. GOLDBERG, P. R. MURGATROYD and W. A. COWARD, *MRC Dunn Clinical Nutrition Centre, 100 Tennis Court Road, Cambridge CB2 1QL*

Potential energy-sparing adaptive responses to underfeeding were investigated by measuring all components of the energy balance equation during experimental dietary restriction. Eight of the subjects described in Diaz *et al.* (1991) (five lean, three overweight) were underfed on a mixed diet (15% protein, 41% fat, 44% carbohydrate by energy) for 6 weeks immediately following the overfeeding period and free-diet 1 period. Each subject's intake equalled their basal metabolic rate (BMR). A second 6-week free-diet period (FD 2) followed the underfeeding. Subjects slept and ate in a long-stay metabolic suite throughout the experiment, but were otherwise unrestricted. Changes in body composition were assessed by densitometry and deuterium dilution. Metabolic responses were assessed at the end of each period by whole-body calorimetry which yielded data on BMR, 24 h energy expenditure on a fixed protocol (CAL EE), and 24 h activity-plus-thermogenesis (CAL A&T derived as CAL EE–BMR). Combined metabolic and behavioural responses were assessed during the final 2 weeks of each period by doubly-labelled water (DLW) which provided data on free-living total energy expenditure (TEE) and free-living activity-plus-thermogenesis (DLW A&T derived as TEE–BMR).

	FD 1		Underfeeding (U)		FD 2		U minus FD 1		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	*P**
Energy intake[1]	13·3†	1·3	7·6	0·6	—	—	−5·7	1·1	<0·001
Body mass[2]	76·6	8·4	70·7	9·0	72·5	7·9	−5·6	0·3	<0·001
FFM[2]	58·7	3·4	56·3	4·4	56·2†	3·3	−2·6	1·7	<0·01
Fat mass[2]	17·6	6·8	14·4	7·0	16·5†	5·7	−4·6	2·1	<0·01
BMR[1]	7·3	0·4	6·7	0·6	7·5	0·3	−0·6	0·3	<0·001
BMR[3]	125	8	119‡	8	126‡	7	−6‡	4	<0·01
CAL EE[1]	10·6	0·6	9·6	0·7	10·2	0·4	−1·0	0·2	<0·001
CAL EE[4]	140	15	137	15	142	14	−3	2	<0·05
CAL A&T[1]	3·3	0·4	2·9	0·4	3·2‡	0·3	−0·4	0·2	<0·01
TEE[1]	12·5	1·8	10·9	1·3	12·5‡	0·8	−1·6	2·3	NS
TEE[4]	167	34	157	29	174‡	27	−10	28	NS
DLW A&T[1]	5·2	1·5	4·2	1·1	5·4‡	0·9	−1·0	2·0	NS

[1]MJ/d; [2]Kg; [3]kJ/kg FFM per d; [4]kJ/kg per d; FFM, fat-free mass; NS, not significant.

* Significance tested by paired *t* test between free-diet 1 and underfeeding.

† Data from initial baseline (see Diaz *et al.* 1991).

‡ *n* 7 due to missing data.

Compared with the FD 1 period, BMR/kg FFM was only decreased by 5%. CAL EE/kg and TEE/kg were only decreased by 2% and 6%. On average, energy-sparing mechanisms only reduced the 5·7 MJ/d energy gap by a maximum of 1·6 MJ/d or 28%. We conclude that human metabolism has a very limited auto-regulatory adaptive capacity in response to altered energy intake, and that changes in body mass are the major outcome.

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Diaz, E., Prentice, A. M., Goldberg, G. R., Murgatroyd, P. R. & Coward, W. A. (1991). *Proceedings of the Nutrition Society* **50**, 110A.

Vitamin E supplementation and vitamin E status of premature infants and the occurrence of retinopathy of prematurity. By PAUL M. MATHIAS, E. B. COLEMAN and K. O'SULLIVAN, *Department of Biological Sciences, Dublin Institute of Technology, Kevin Street, Dublin 8* and M. HOGAN, *Coombe Hospital, Dublin 8, Republic of Ireland*

To date there is much conflicting information on the efficacy as well as safety of vitamin E supplementation in premature infants, especially in the prevention of retinopathy of prematurity (ROP) (Kretzer & Hittner, 1988). The aim of the present pilot study was to examine the effect of a current feeding regimen on the vitamin E status of premature infants, and to record the incidence of ROP.

Twenty-eight infants (sixteen male and twelve female) born at the Coombe Maternity Hospital, Dublin, were studied. Their birth weights ranged from 620 to 2290 g (mean 1313 g) and gestational age from 26 to 36 weeks (mean 30 weeks). Thirty-two per cent of infants weighed <1000 g at birth, 39% between 1000 and 1500 g and 29% >1500 g. During routine management of these infants a vitamin E supplement of 20 mg oral α-tocopheryl acetate was given daily whenever possible. Serum samples from a selection of these infants were taken at birth, 2–3 weeks, 4–5 weeks and 6–7 weeks, and were analysed for α-tocopherol (α-T), cholesterol (C), and the α-tocopherol:cholesterol ratio (α-T:C) calculated. The results are shown in the Table.

Time (weeks)	*n*	α-T (μmol/l)		C (mmol/l)		α-T:C ratio (μmol/mmol)	
		Mean	SE	Mean	SE	Mean	SE
Birth	15	7·9	1·7	2·56	0·90	3·10	1·00
2–3	12	26·2	4·3	2·98	0·76	8·79	3·10
4–5	4	29·5	4·0	3·08	0·71	9·58	2·17
6–7	7	39·1	4·6	3·47	0·99	11·27	2·98

At birth there was no correlation between either birth weight or gestational age and α-T, but there was a significant positive correlation between α-T and C (r 0·790, $P<0·001$). α-T levels rose to adult-type values within 2–3 weeks, with improvement of vitamin E status confirmed by the appreciable rise in α-T:C values. Seven (27%) infants developed ROP: five of these weighed <1000 g at birth. No child developed beyond grade iii (severe) ROP. This incidence rate for ROP compares favourably with other studies (e.g. Phelps *et al.* 1987), but with less vitamin E supplements used (20 mg/d *v.* e.g. 100 mg/kg per d), and a lower plasma vitamin E level attained (39·1 *v.* e.g. 81·4 μmol/l). Considering the risks associated with excessive vitamin E supplementation in premature infants, such as necrotizing enterocolitis (Johnson *et al.* 1988), the present study indicates that a less aggressive approach to vitamin E therapy could be adopted.

Johnson, L., Quinn, G. E., Abbassi, S., Otis, C. & Bowen, F. W. (1988). *Paediatrics* **81**, 329–331.
Kretzer, F. L. & Hittner, H. M. (1988). *Archives of Diseases of Childhood* **63**, 1151–1167.
Phelps, D. C., Rosenbaub, A. L., Isenburg, S. J., Leake, R. D. & Davey, F. J. (1987). *Paediatrics* **79**, 489–500.

Effect of tea on iron and zinc absorption in suckling rats. By M. REDDY, A. FLYNN and F. O'LOUGHLIN, *Department of Nutrition, University College, Cork, Republic of Ireland*

Tea has been shown to inhibit non-haem iron absorption (Disler *et al.* 1975). However, it is unclear if this inhibition is influenced by the addition of milk to tea or if tea influences the absorption of other trace elements. This report outlines the effect of tea, with or without milk, on Fe and zinc absorption in suckling rats.

Tea infusion was prepared by adding 200 ml boiling water to 3 g tea leaves (Lyons Mauve Label) and allowing to stand for 5 min before filtering through a tea strainer. Fe and Zn contents of tea infusion were 0·10 and 0·13 mg/l respectively. Tea infusion was mixed with water (4:1) or milk (4:1) and control solutions of water and water+milk (4:1) were prepared. All solutions were labelled with $^{59}FeCl_3$ and $^{65}ZnSO_4$ to give final concentrations of 6 mg/l for both Fe and Zn and 1 μCi/ml for both ^{59}Fe and ^{65}Zn, and 0·4 ml was given by gavage to 16-d-old rats, previously fasted for 18 h. Animals were killed 6 h later and stomach, small intestine (SI), caecum–colon and liver removed. SI was perfused with 6 ml 0·15 M-sodium chloride. ^{59}Fe and ^{65}Zn were determined in a well gamma counter using a channels ratio method.

Table. *Uptake (% of dose) of ^{59}Fe and ^{65}Zn from water, water+milk, tea and tea+milk*

	Water (*n* 6)		Water+milk (*n* 6)		Tea (*n* 6)		Tea+milk (*n* 6)	
Organ/tissue	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
^{59}Fe								
Stomach	1·2	0·1	1·1	0·2	0·7	0·1	0·3	0·1
SI	7·6	1·1	6·8	1·0	10·7	2·7	7·2†	1·7
SI perfusate	1·4	0·3	1·3	0·2	2·6	1·6	2·8	1·7
Caecum–colon	6·9	1·0	6·5	0·8	57·8**	5·7	72·9**†	6·3
Liver	10·8	0·3	10·8	0·6	3·0**	0·4	1·8**†	0·2
Absorbed‡	90·5	0·9	91·2	0·8	39·0**	4·3	24·1**†	6·2
Carcass§	82·9	0·9	84·5	1·7	28·3**	2·7	17·0**	5·7
^{65}Zn								
Stomach	1·3	0·1	1·7	0·3	1·0	0·1	0·9	0·1
SI	30·6	3·5	25·3	0·7	25·5	1·6	20·9*	1·0
SI perfusate	6·5	1·3	8·3	0·8	2·8	0·8	3·3	0·5
Caecum–colon	7·0	1·7	10·1	2·1	26·3**	3·7	32·4**	5·5
Liver	19·8	0·7	19·2	1·1	14·5**	0·9	15·4	2·3
Absorbed‡	85·3	1·9	80·0	2·5	69·9*	3·7	63·5**	5·7
Carcass§	54·6	2·3	54·8	2·4	44·4*	2·2	42·7	5·3

Significantly different from water (Student's paired *t* test): $*P<0·05$, $**P<0·01$.
Significantly different from tea: $†P<0·05$.
‡ Absorbed = 100 − (stomach + SI perfusate + caecum–colon) (%).
§ Carcass = absorbed − SI.

These results show that tea is inhibitory to absorption of both non-haem Fe and, to a lesser extent, Zn and this effect is exacerbated by milk for Fe but not Zn. These effects are probably due to chelation of Fe and Zn with phenolic compounds in the gastrointestinal tract (Brune *et al.* 1989).

Brune, M., Rossander, L. & Hallberg, L. (1989). *European Journal of Clinical Nutrition* **43**, 547–558.
Disler, P. B., Lynch, S. R., Charlton, R. W., Torrance, J. D., Bothwell, T. H., Walker, R. B. & Mayet, F. (1975). *Gut* **16**, 193–200.

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Absorption of manganese from tea in suckling rats. By A. L. FRAILE and A. FLYNN, *Department of Nutrition, University College, Cork, Republic of Ireland*

Tea contains a large amount of manganese, 610 (350–900) mg/kg dry tea (Wenlock *et al.* 1979), and contributes ~21% of the total Mn in the British household food supply (Lewis & Buss, 1988). However, the bioavailability of Mn in tea is unknown. Since there is evidence that tea inhibits the absorption of iron and zinc (Reddy & Flynn, 1991) the present study was carried out to investigate the bioavailability of Mn in tea.

Tea infusion was prepared by adding 200 ml boiling water to 3 g tea leaves (Lyons, Mauve Label) and allowing to stand for 5 min before filtering through a tea strainer. The Mn content of tea infusion was 2·1 mg/l. Tea infusion was mixed with water (4:1) or milk (4:1) and control Mn solutions (1·5 mg/l) were prepared with $MnSO_4$ in water and in water+milk (4:1). All solutions were extrinsically labelled with ^{54}Mn (1 μCi/ml) and 0·4 ml was given by gavage to 16-d-old rats, previously fasted for 18 h. Animals were killed 6 h later and stomach, small intestine (SI), caecum–colon and liver removed. SI was perfused with 6 ml 0·15 M-sodium chloride. ^{54}Mn in tissues was determined in a well gamma counter.

Table. *Uptake (% of dose) of ^{54}Mn from water, water+milk, tea and tea+milk*

	Water (*n* 5)		Water+milk (*n* 5)		Tea (*n* 5)		Tea+milk (*n* 4)	
Organ/tissue	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Stomach	2·2	0·2	3·1	0·7	6·4	2·7	3·0	1·1
SI	57·3	3·5	57·7	2·9	64·7	3·4	59·8	1·9
SI perfusate	4·5	1·1	4·3**	0·7	2·3	0·9	5·4*	0·9
Caecum–colon	0·2	0·1	0·5	0·1	0·4	0·1	0·5	0·1
Liver	16·8*	0·6	18·6	0·9	19·3	1·4	15·8	1·9
Absorbed†	93·1	1·1	92·1	0·8	90·9	2·5	91·1	1·9
Carcass‡	35·8	4·2	34·4	3·5	26·2	2·1	31·1	2·8

Significantly different from tea (Student's paired *t* test): *$P<0·05$, **$P<0·01$.
† Absorbed=100–(stomach+SI perfusate+caecum–colon) (%).
‡ Carcass=absorbed–SI.

When ^{54}Mn was given in water, over 93% of the dose was absorbed and about 36% transferred to the carcass with about 17% in the liver. There was a high proportion of dose retained in the SI, mainly in the duodenum. ^{54}Mn absorption from tea and tea with milk were very similar to that for ^{54}Mn in water.

These results show that Mn in tea is highly absorbable in suckling rats and is unaffected by addition of milk. In contrast to Fe and Zn (Reddy & Flynn, 1991), phenolic compounds in tea do not affect Mn absorption. Thus tea appears to be a rich source of bioavailable Mn.

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Lewis, J. & Buss, D. H. (1988). *British Journal of Nutrition* **60**, 413–424.
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Adolescent attitudes towards body physique. By MAURA FOX, *St Angela's College of Education for Home Economics, Sligo, Republic of Ireland*

The slim figure as the ideal of beauty is portrayed by all forms of the media in today's Western society. The present study examines attitudes of adolescent girls to their own body sizes and compares these with anthropometric measurements of the same subjects. Data were obtained by questionnaire from 437 adolescent (15–19 years) girls (65·4% of contacted group). Age, social class by father's occupation (Census of the Population, 1981), attitudes towards body size and aspects of slimming, together with direct measurement (single observer) of height, weight and triceps fatfold (Holtain skin calipers) were recorded.

Anthropometric and socio-economic data were analysed by the Kolmogorov–Smirinov/Lillifors test, product-moment correlation coefficient and analysis of variance, and attitudes data by chi-square.

Overall mean body mass index (BMI) was 21·3 kg/m^2 with 71% of the sample within the acceptable range (18·0–22·9 kg/m^2; Royal College of Physicians, 1983), 6% less than 18·0 kg/m^2, 15% with BMI between 23·0 and 24·9 kg/m^2 and 8% with BMI 25·0–33·1 kg/m^2; BMI and triceps fatfold were positively correlated (r 0·84, $P<0·001$). Social class had no significant ($P=0·065$) relationship with BMI. Self-estimated BMI was highly correlated (r 0·73, $P<0·001$) with measured BMI.

A majority (93%) of subjects irrespective of age, social class or body size wished to be slim. The slim figure was perceived to be significantly ($P<0·001$) associated with status factors including career success, sexual attractiveness and admiration by female friends. Attempts at slimming were frequently reported, with 70% of subjects trying to restrict food intake in the previous year and 20% and 10% doing so over 2 and 3 years respectively. Reported weight loss was 3·1 to 28·5 kg with 62% admitting to regaining lost weight. Methods of slimming included energy-intake restriction with exercise (42% of dieters), energy restriction alone (30%), exercise alone (9%), smoking (1%) and the use of laxatives (1%). Slimming was not medically monitored and reported associated complications included anxiety (6%), amenorrhoea (6%), sleeplessness (13%) and depression (17%).

Results suggest that irrespective of anthropometry, adolescent subjects were unhappy with body physique and many were attempting to slim unnecessarily.

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Weight change in subjects voluntarily enrolling in successive annual 'sponsored slims'. By JOYCE HUGHES[1], GILL FRANKS[2] and MICHAEL STOCK[1], [1]*Department of Physiology, St George's Hospital Medical School, London SW17 0RE* and [2]*Department of Dietetics, St George's Hospital, London SW17 0RE*

Four successive (1987, '88, '89, '90) sponsored slims (SpS) (9–11 weeks duration) involving 90–130 slimmers were organized by dietitians and a research nutritionist at St George's Hospital to help raise money for a medical charity.

Forty-four subjects enrolled in more than one SpS, referred to as repeat slimmers (RS); thirty-three for two, seven for three and four for all four SpS. Data on these subjects were analysed to determine the weight change during, between and at the end of several attempts to lose weight to see the net effect of repeated weight loss regimens. The RS lost slightly less weight than the other participants in '87 and '88. However, in '89 and '90 RS lost more weight. The difference in mean weight loss between the RS and the other participants was significant ($P<0{\cdot}01$) in 1990, but not in previous SpS. Not all RS were successful in losing weight, two in '87, four in '89 and two in '90 gained between 0·1 and 3·2 kg.

Weight loss (kg) during sponsored slims

Slimmers . . .	All*		Repeat		Single	
	Mean	SD	Mean	SD	Mean	SD
1987	3·7	3·1	3·3	3·3	3·8	3·0
1988	3·3	2·5	3·1	2·1	3·5	2·7
1989	2·4	2·4	2·8	3·3	2·3	2·1
1990	2·2	2·0	3·3	2·2	1·9**	1·8

* Drop-outs excluded.
** $P<0{\cdot}01$ compared with Repeat slimmers.

Of the four RS who took part in all four SpS, three finished with a net weight gain (6·1, 5·1 and 1·7 kg) and only one finished with a net weight loss (4·3 kg). Those taking part in three SpS fared better, five with net weight losses (0·6–15·4 kg) and only two with net weight gains (both 1·8 kg). For RS taking part in just two SpS there were twenty-six net losses (0·4–9·4 kg), two with no change and five with net gains (0·8–9·0 kg). Most RS (100% between '87 and '88; 87% between '88 and '89; 94% between '89 and '90) experienced a net weight gain in the interval between the repeat SpS, and twenty-four of the '2×SpS' group (*n* 33) finished heavier (0·6–13·7 kg) at the end of their second SpS than at the end of their first SpS.

The relatively small mean weight loss during each 10 week 'sponsored slim', even for dedicated repeat slimmers, indicates that sponsored slimming, with professional dietary advice, fortnightly weight checks and the incentive of sponsorship is an unsatisfactory method for the obese, of which there were thirteen RS with an initial body mass index (BMI) >30 kg/m^2 and one RS with an initial BMI >40 kg/m^2. However, for some RS, who were in the normal to overweight category (BMI 23–30 kg/m^2), this small mean weight loss may be sufficient to prevent development of obesity in the long term.

Nutrient intakes of medically-advised and self-selected slimmers: results from the Scottish Heart Health Study. By C. BOLTON-SMITH, *Cardiovascular Epidemiology Unit, Ninewells Hospital and Medical School, Dundee DD1 9SY*

The accurate assessment of dietary intake is a major problem in any group, but especially difficult for slimmers. Slimmers may be more likely to underestimate portion sizes, and to omit sweets and snacks than non-slimmers. While food frequency questionnaire (FFQ) data are known to underestimate total energy intake compared with 7 d weighed records, energy-adjusted nutrient values agree reasonably well for the two methods (Bolton-Smith & Milne, 1991).

Dietary data were obtained by FFQ, using standard portion sizes (Yarnell *et al.* 1983), as part of the Scottish Heart Health Study: a cross-sectional study of risk factors for coronary heart disease in men and women aged 40–59 years (Smith *et al.* 1989). A question on special diet was included, and the nutrient intakes of those reporting to be on a medically-advised slimming diet (Med-slim) and those on a self-selected slimming diet (Self-slim) were compared with those on no special diet (Non-slim).

	Men							Women						
n . . .	Non-slim 4592		Med-slim 37		Self-slim 167			Non-slim 4224		Med-slim 89		Self-slim 572		
	Mean	SD	Mean	SD	Mean	SD	*P*	Mean	SD	Mean	SD	Mean	SD	*P*
BMI	25·9	3·4	30·7	5·0	28·4	3·0	***	25·3	4·4	30·2	5·2	27·8	4·9	***
Energy (MJ)	9·96	2·55	7·84	2·90	7·97	2·09	***	7·53	1·97	6·87	1·81	6·50	1·76	***
Protein (% energy)	15·0	2·1	18·0	3·8	16·7	2·6	***	16·9	2·6	19·3	4·1	19·2	3·1	***
Fat (% energy)	34·8	5·7	36·0	7·0	35·3	7·6	NS	39·7	6·0	39·6	6·4	38·3	6·6	***
Starch (% energy)	27·4	5·9	25·4	6·6	27·0	7·3	NS	24·9	5·5	23·9	6·3	24·0	5·8	***
Sugar (% energy)	16·6	5·5	14·2	4·3	14·6	4·4	***	16·0	5·5	15·4	5·0	15·3	4·2	*
Alcohol (% energy)	6·3	6·5	6·5	6·1	6·3	6·7	NS	2·5	3·3	1·8	2·9	3·2	4·0	***
P:S	0·25	0·31	0·30	0·29	0·32	0·35	*	0·25	0·26	0·30	0·27	0·27	0·34	*
Fibre (g ND)	9·1	3·2	11·7	4·1	12·0	4·3	***	10·8	3·9	12·3	3·8	13·1	4·0	***
Vitamin C (mg ND)	23·1	10·8	35·5	16·1	32·1	17·5	***	29·5	16·0	40·0	21·9	40·9	19·1	***
Vitamin E (mg ND)	4·2	2·3	4·6	2·9	5·0	3·0	***	4·9	2·7	5·4	3·3	5·1	2·9	**
Retinol (μg ND)	316	164	487	371	368	209	***	396	212	472	339	459	277	**
β-Carotene (μg ND)	1398	982	2384	1641	1860	1553	***	1948	1441	2582	1829	2389	1723	***

BMI, body mass index (weight, kg/height, m^2); P:S, ratio of polyunsaturated to saturated fat; ND, nutrient density (amount/4·18 MJ (1000 kcal)); *P*, significance level by analysis of variance on appropriately ln and $(\text{arcsine})^{1/2}$ transformed data.
$^*P<0·05$, $^{**}P<0·01$, $^{***}P<0·001$.

The lower mean energy intakes in the slimming groups would appear to agree with their 'special diet' status and may suggest that frequency of food consumption is adequate to differentiate between the groups. However, biased reporting between the groups cannot be ruled out completely for the FFQ has not been specifically validated in slimmers. The only significant differences (by ANOVA) between the nutrient values for the Med-slim and Self-slim groups were, for men, % energy from protein $P<0·05$, and for women, % energy from alcohol $P<0·001$.

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Hunger and food cravings in successful weight losers. By ELIZABETH EVANS, *Slimming Magazine Clubs, London SW7 3HG*

Obese people find it difficult to lose and maintain weight, yet little attention has been focused on successful weight losers who are subject to the same physiological, psychological, social and environmental influences as they were when overweight. Analysis of the strategies adopted by such people to cope with hunger and food cravings is of potential value in improving the quality of advice given to overweight people, and so improve the currently poor rate of success in the treatment of obesity.

The hunger and food cravings experienced by two groups of successful weight losers were recorded retrospectively by questionnaire. Group A consisted of fifty women (41·6 (SE 1·3) years) who had lost weight (maximum body mass index (BMI) 29·1 (SE 0·6) kg/m^2) and maintained weight (BMI 21·8 (SE 0·1) kg/m^2) for 2–25 years (mean 9·2 (SE 0·7)). Group B was made up of sixty-two (sixty female, two male) individuals aged between 25 and 74 years who had all lost at least 45·5 kg (maximum BMI 36·3–62·1 kg/m^2). Twenty-two still had weight to lose (BMI 29·1 (SE 1·1) kg/m^2), the remainder had lost all their excess weight and had maintained at this lower body-weight (BMI $<25·0$ kg/m^2) for up to 12 years. Both groups lost weight by following an energy-restricted diet of not less than 4184 kJ (1000 kcal)/d while attending a commercial slimming group.

Ranked in order of importance, beverages – tea, coffee or diet drinks – keeping busy and consuming more vegetables were the main strategies used by both groups to curb hunger and to keep in energy balance. For those in group B who were still losing weight, keeping busy was most important.

The majority in both groups (78% A, 82% B) claim to have had food cravings while dieting but only half continued to do so once slim. Although many could not ascribe their food cravings to any particular time or situation, premenstrual tension and eveningtime were also important triggers. The foods most craved were chocolate (20% A, 40% B), biscuits (14% A, 15% B), cheese (14% A), cakes (10% A, 16% B) and bread (12% A, 11% B). Cravings were assuaged either by avoiding the food altogether (less common with chocolate) or by including into the daily allowance or by selecting a low-energy alternative.

Weight maintenance in both groups was effected by adherence to a low-fat, low-sugar, high-fibre diet. A better understanding of the energy and nutritional value of food enables these individuals to manage their food intake and avoid weight regain.

Failure to demonstrate brown adipose tissue thermogenesis in adult man using infra-red thermography during both cold- and glucose-induced thermogenesis. By ELIZABETH M. WHITE and J. F. ANDREWS, *Department of Physiology, Trinity College, Dublin 2, Republic of Ireland*

Does brown adipose tissue (BAT) retain thermogenic capacity into adulthood in man? An histological investigation in cadavers (Heaton, 1972) showed partial but not total loss of BAT-like tissue with ageing, BAT being retained at the neck site particularly. Biochemical and other evidence support some minimal retention of functional capacity (Lean, 1989). These approaches cannot confirm thermogenic function. Infra-red thermography showed promise as a means of demonstrating BAT activity (Rothwell & Stock, 1979).

Young adult men and women were studied seated in still air in an environmental chamber at either 30° or 11°. Whole-body metabolic responses were determined after either a glucose load (100 g in 500 ml water, lemon flavoured) or a sham feed of 500 ml of a saccharine-sweetened lemon-flavoured drink. Metabolic rate was determined indirectly as oxygen consumption using a face mask and an open-circuit system. A Bofors infra-red camera was used to determine the changing thermal patterns on the back of the subject related in time to the whole-body metabolic rate. Two areas were selected for further analysis: (1) neck, over a putative BAT site and (2) a neutral off-centre lumbar site. Image analysis was used to calculate mean surface temperatures at these sites. The accuracy of the thermographic process was confirmed by spot thermocouples placed at the sites.

Temperature . . .	30° (*n* 22)						11° (*n* 11)					
	Lumbar (°)		Neck (°)		Difference† (°)		Lumbar (°)		Neck (°)		Difference† (°)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Pre-drink	32·0	0·2	33·7	0·2	1·7	0·2	23·1	0·5	27·2	0·5	4·1	0·5
Post-drink*	32·6‡	0·3	34·1‡	0·2	1·6	0·1	23·1	0·6	27·0	0·6	4·1	0·5

* For 30–90 min period post-injection, time of maximum metabolic stimulation.
† Mean of individual differences.
‡ $P<0·01$ compared with pre-drink, Student's paired *t* test.

The results in the Table show a temperature gradient warmest to coldest: neck>lumbar. This would be expected from the physical chimney effect. At 30° glucose caused a post-ingestion thermogenesis as expected and a parallel generalized increase in surface temperature. Individual site temperatures increased, but gradients, particularly neck to lumbar did not. At 11° there was no effect of glucose ingestion on surface temperature, which would be expected as the metabolic data showed the post-ingestion thermogenesis to be subsumed in to the ongoing cold-induced thermogenesis. The gradient, neck to lumbar, did increase but since neck temperature fell from warm to cold (by 5·5°) this was not taken to be an indication of underlying BAT thermogenesis, rather an enhancement of the chimney effect.

These results do not support the possibility that BAT thermogenesis can occur to any great extent in adult man in response either to cold or to dietary energy intake. However, if, as some evidence suggests, only internal sites retain activity (Lean, 1989) infra-red thermography would fail to show any effect. Further, good perfusion of an active site of thermogenesis could minimize its temperature increase relative to that of surrounding tissues and again infra-red thermography would fail to detect this activity.

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The effect of dietary α-tocopherol supplementation on the storage stability of chicken muscle. By P. J. A. SHEEHY, P. A. MORRISSEY and A. FLYNN, *Department of Nutrition, University College, Cork, Republic of Ireland*

Lipid oxidation of meat is an important consideration for both consumer and producer. Oxidation products cause off-flavour development and adversely affect colour, nutritive value and safety of meats. Vitamin E (α-tocopherol) functions as a lipid-soluble antioxidant (Burton & Ingold, 1981), and recent studies have shown that dietary α-tocopherol supplementation is effective in increasing muscle tocopherol concentration and limiting oxidation of pork during storage (Monahan *et al.* 1990). The objective of the present study was to investigate the effect of dietary α-tocopherol on the storage stability of chicken.

Male chicks (age 1 d) were randomized into four groups and fed on diets containing 5, 25, 65 and 180 μg α-tocopherol/g feed. The chicks were killed by cervical dislocation on day 24. Diet and muscle tocopherol concentrations were determined by high-performance liquid chromatography. Representative samples of raw or cooked (75°×60 min) muscle were stored under refrigerated conditions (4°) and oxidative changes monitored after 0–7 d, using the thiobarbituric acid method of Ohkawa *et al.* (1979). Similar samples were stored in a freezer (−20°) and their oxidative stability determined after 2 months. The results are shown in the Table.

Sample	Dietary α-tocopherol (μg/g)	Muscle α-tocopherol (μg/mg protein)		TBARS (nmol malondialdehyde/mg protein)											
				Days at 4°										Months at −20°	
				0		2		4		5		7		2	
		Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Raw	5	0·016[d]	0·001	0·40	0·04	0·53	0·05	0·65	0·07	–		0·92	0·08	3·38[a]	0·42
	25	0·038[c]	0·007	0·32	0·06	0·44	0·03	0·73	0·11	–		0·90	0·13	3·30[a]	0·27
	65	0·103[b]	0·008	0·52	0·10	0·59	0·09	0·79	0·10	–		1·19	0·15	1·98[b]	0·22
	180	0·260[a]	0·065	0·42	0·07	0·47	0·005	0·74	0·11	–		1·10	0·23	1·69[b]	0·28
Cooked	5	–		4·21[a]	0·90	3·89[a]	0·28	–		6·37[a]	0·66	–		7·28[a]	0·94
	25	–		3·28[ab]	1·23	2·67[b]	0·47	–		5·89[a]	1·08	–		5·80[ab]	0·72
	65	–		3·23[ab]	0·89	2·04[bc]	0·33	–		4·43[ab]	0·88	–		3·81[b]	0·62
	180	–		1·72[b]	0·43	1·54[c]	0·13	–		3·07[b]	0·54	–		1·71[c]	0·07

[a–d] Mean values in columns with different superscript letters are significantly different ($P<0·05$).

Muscle α-tocopherol concentration increased linearly with dietary tocopherol level. Thiobarbituric acid-reacting substances (TBARS) in raw muscle stored at 4° increased over a 7 d period, although values between groups did not differ significantly, probably because of the low rate of lipid oxidation in all samples during this time. In contrast, TBARS in samples from chicks fed on the lower levels of α-tocopherol were significantly elevated following frozen storage for 2 months.

TBARS in cooked samples were higher than those in corresponding raw samples. High dietary α-tocopherol supplementation significantly reduced TBARS during both refrigerated and frozen storage. In general, therefore, the results indicate that the shelf-life of chicken is extended by dietary α-tocopherol supplementation.

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The effect of a non-absorbable lipid, lactitol polyester on fat-soluble vitamin and cholesterol levels in the rat. By A. O. CONNELL, J. F. CONNOLLY[2], A. FLYNN[1] and P. A. MORRISSEY[1], [1]*Department of Nutrition, University College, Cork* and [2]*Teagasc, Moorepark, Fermoy, Co. Cork, Republic of Ireland*

Sugar polyesters are synthetic lipids with physical and organoleptic properties virtually identical to those of triacylglycerols. They differ from triacylglycerols in one important and unique property in that they are not hydrolysed in the intestine of either man or animal and hence are not absorbed. By providing a persistent lipophilic phase in the intestine, the polyesters tend to reduce the absorption of lipophilic substances such as cholesterol and fat-soluble vitamins (Jandacek, 1984).

In the present study, lactitol polyester (LPD) was synthesized from the sugar alcohol, lactitol, and fatty acid methyl esters of partially hydrogenated soya-bean oil. LPE had a hydroxyl value of 88·44 which is equivalent to an average of about 6·0 ester groups per molecule of lactitol, and contained less than 1% free fatty acids.

Twenty-four male rats were randomly divided into four groups and fed on diets containing (g/kg): 150 coconut oil (control), or 100 coconut oil plus 50 LPE, or 50 coconut oil plus 100 LPE, or 150 coconut oil plus 50 LPE. After 28 d, the rats were anaesthetized and decapitated, and plasma tissues and faeces were prepared for determination of lipid (Soxhlet extraction), cholesterol (commercial enzyme kits; Boehringer-Mannheim, Mannheim, W. Germany) and α-tocopherol and retinol (high-performance liquid chromatography).

The effect of LPE on plasma, liver and faecal α-tocopherol and retinol, plasma and faecal cholesterol and faecal lipids in rats

	Control (*n* 6)		50 g LPE/kg (*n* 6)		100 g LPE/kg (*n* 6)		Control + 50 g LPE/kg (*n* 6)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Plasma:								
α-Tocopherol (μg/ml)	6·96	0·62	5·01*	1·18	3·63**	0·87	4·26**	0·45
Retinol (μg/ml)	0·45	0·11	0·58	0·12	0·56	0·10	0·68	0·16
Cholesterol (mg/l)	721	61	762	145	753	92	678	42
Liver:								
Retinol (μg/g)	10·1	3·3	11·7	4·9	12·7	3·4	11·8	3·9
α-Tocopherol (μg/g)	147·3	13·6	89·9**	16·5	56·7**	8·5	86·4**	9·4
Faeces:								
Lipid (g/g total solids)	0·04	0·001	0·21**	0·03	0·32**	0·05	0·25**	0·04
α-Tocopherol (μg/g total solids)	72·4	14·7	202·2**	7·6	185·5**	25·8	150·5**	43·6
Retinol (μg/g total solids)	0·53	0·31	0·67	0·42	0·44	0·13	0·37	0·25
Cholesterol (mg/g total solids)	1·55	0·12	1·82	0·24	1·84**	0·10	1·78	0·20

Significantly different from control: *$P<0·05$, **$P<0·01$.

The substitution of dietary fat by LPE or the additional supply of LPE led to an increase in faecal lipid excretion, and in the case of the 100 LPE group, a significant increase in cholesterol excretion. Plasma cholesterol levels, however, were not affected by the presence of LPE in the diet. All treatment groups showed significant decreases in plasma and liver α-tocopherol concentrations, and faecal α-tocopherol concentrations were found to be significantly increased. The presence of LPE in the diets had no significant effect on retinol concentration in any of the above variables. The mean body-weights remained similar among all groups, but LPE-fed animals showed a very significant decrease in feed conversion efficiency.

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The effect of propargylglycine feeding on copper status in the rat. By J. C. W. BROWN and J. J. STRAIN, *Biomedical Sciences Research Centre, University of Ulster at Jordanstown, Newtownabbey, Co. Antrim BT37 0QB, Northern Ireland* and P. YOUNG, *Veterinary Research Laboratories, Stormont, Belfast BT4 3SD, Northern Ireland*

Homocysteine feeding can lower copper status in the rat (Brown & Strain, 1990), but Cu deficiency does not affect plasma homocysteine (Hcy) levels (Brown *et al.* 1990). In the current study propargylglycine (PPG), an inhibitor of cystathionase (*EC* 4.4.1.1), was used to block the transsulphuration pathway and the effect of the resultant increased Hcy and cystathionine levels on Cu status in the rat was investigated.

Four groups (*n* 8) of male weanling Sprague-Dawley rats were provided with deionized water and fed on the experimental low-Cu diets for 21 d. Group A was fed on the basal diet with the additions of PPG (6 mmol/kg) and DL-cysteine (2 g/kg). Two control groups were fed on the same amount of basal diet as group A, either with DL-cysteine (2 g/kg) added (B) or alone (C). Group D was fed on the basal diet *ad lib*. Hepatic and cardiac levels of Cu and activities of the Cu-dependent enzyme cytochrome *c* oxidase (CCO; *EC* 1.9.3.1) were measured together with hepatic Cu/Zn superoxide dismutase (Cu/Zn-SOD; *EC* 1.15.1.1) and plasma caeruloplasmin (CP; *EC* 1.16.3.1) as indicators of Cu status.

Group . . .	A†		B		C		D	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Body-wt (g)	66·2^{a}	2·3	86·7^{b}	4·7	82·9^{ab}	4·7	172·6^{c}	9·9
Heart-wt (% body wt)	0·48^{ab}	0·02	0·42^{b}	0·01	0·43^{b}	0·01	0·51^{a}	0·03
Liver-wt (% body wt)	6·55^{a}	0·18	4·37^{b}	0·32	4·23^{b}	0·27	5·92^{a}	0·18
Liver Cu (μg/g dry wt)	11·95^{ab}	0·49	12·31^{ab}	1·09	12·91^{b}	0·97	9·86^{a}	1·06
Heart Cu (μg/g dry wt)	12·90^{a}	0·65	13·08^{a}	0·50	13·15^{a}	0·36	10·72^{b}	0·48
Liver CCO (U/mg protein)	2·36^{ab}	0·42	2·83^{ab}	0·37	3·33^{b}	0·36	1·74^{a}	0·53
Heart CCO (U/mg protein)	2·84^{a}	0·43	2·70^{a}	0·18	3·13^{a}	0·26	1·34^{b}	0·09
Liver Cu/Zn-SOD (U/mg protein)	69·0^{a}	3·9	69·9^{a}	3·5	71·5^{a}	4·8	64·4^{a}	4·2
Plasma CP (U/l)	ND		ND		ND		ND	
Plasma Hcy (μmol/l)	57·6^{a}‡	0·83	9·6^{b}‡	0·56	10·2^{b}‡	0·63	13·1^{b}	0·67

ND, not detectable.

$^{a-c}$ Means in horizontal rows with different superscript letters are significantly different by the least significant difference test: $P<0·05$.

† *n* 6; ‡*n* 2.

Although plasma homocysteine levels were significantly increased by PPG feeding, the induced homocysteinaemia did not markedly affect Cu status over the 21 d period. This group (A) exhibited marked growth retardation compared with the other groups and PPG feeding also resulted in hepatic hypertrophy. The major differences in Cu status among the groups, however, was the marked decrease in indicators of Cu status in group (D) compared with the other groups. The lowered Cu status of group (D) could have been due to the much higher growth rates of this group compared with those of the PPG and control groups.

Brown, J. C. W., Mazdai, G. & Strain, J. J. (1990). *Proceedings of the Nutrition Society* **49**, 106A.
Brown, J. C. W. & Strain, J. J. (1990). *Journal of Nutrition* (In the Press).

Dietary intakes and essential fatty acid levels in patients with cystic fibrosis. By J. M. THOMSON, R. MOORE, C. MCMASTER and J. A. DODGE, *Department of Child Health, Queen's University, Institute of Clinical Science, Grosvenor Road, Belfast BT12 6BJ, Northern Ireland*

Deficiency of the essential fatty acid linoleic acid has been widely reported in cystic fibrosis (CF). Although its aetiology is unclear, it is possible that malabsorption in conjunction with a low-fat diet may aggravate any metabolic defect in fatty acid metabolism. We looked at the erythrocyte membrane fatty acids in thirty-six patients with CF (twenty-one males, fifteen females), mean age 12·6 years (SD 6·29) ranging from 2 to 25 years, with a body mass percentile of 93%. Forty-eight healthy controls (age range 1–30 years, twenty males and twenty-eight females) were also recruited. Thirty-four patients had pancreatic insufficiency and were receiving enzyme-replacement therapy.

Table 1. *Erythrocyte membrane fatty acids (%)*

	18:2 *n* 6		18:3 *n* 6		20:3 *n* 6		20:4 *n* 6	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
CF patients	13·36*	2·73	0·05	0·12	1·60	0·43	14·18	1·70
Controls	14·89	1·59	0·02	0·09	1·53	0·45	13·29	1·70

*$P<0·01$ compared with controls, Student's t test.

There was no evidence of clinical or biochemical deficiency in any of the subjects. Twenty-four of the subjects agreed to record their nutrient intake by means of a 7 d weighed dietary inventory. Calculation of the macronutrient content of the diets was made using a computerized food composition database.

Table 2. *Macronutrient intakes of patients with CF*

	% RDA		% Energy			P:S ratio
	Energy	Protein	Protein	Fat	Carbohydrate	
Mean	95·9	133·1	13·1	34·1	52·8	0·22
SD	18·5	36·5	2·4	4·8	5·5	0·16

RDA, recommended daily amounts (Department of Health and Social Security, 1979); P:S, polyunsaturated:saturated fats.

Patients attending this clinic no longer follow a diet restricted in fat. Arbitrary recommendations for diet and CF state that patients require 120–150% RDA for energy and 150–200% RDA for protein, which clearly we have not achieved. It has been suggested that total energy is the important factor in maintaining essential fatty acid levels (Parsons *et al.* 1988). Perhaps the normal- to high-fat diet will also lead to a reduction in the incidence of essential fatty acid deficiency, provided that an adequate energy intake is maintained.

Department of Health and Social Security (1979). Recommended daily amounts of food energy and nutrients for groups of people in the United Kingdom. *Report on Health and Social Subjects* no. 15. London: H.M. Stationery Office.

Parsons, H. G., O'Loughlin, E. V., Forbes, D., Cooper, D. & Gall, D. G. (1988). *Pediatric Research* **24** (3), 353–356.

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Dietary calcium and fibre intakes in adolescents and in the elderly in Dublin. By LOUISE BYRNE, DEIRDRE KELLY and N. P. KENNEDY, *Division of Nutritional Sciences, Department of Clinical Medicine, Trinity College, Dublin, Republic of Ireland*

The risk of osteoporosis in later life is partly dependent on dietary calcium intake, most especially during bone growth and in postmenopausal women. The present work was carried out to estimate the intake of Ca and fibre by adolescents and by elderly subjects in the Dublin area, to compare the intake of these nutrients in the two age groups and to compare their intake in adolescents from high- and low-income families.

Dietary assessments were carried out in thirty-two subjects aged over 65 years (sixteen male, sixteen female) selected from the general medical services (GMS) lists of two general practitioners (GPs), using food frequency questionnaires for Ca (Nelson *et al.* 1988) and fibre.

The same questionnaires were used to evaluate the diet of twenty adolescents aged between 11 and 14 years who also weighed and recorded their dietary intakes for 7 d following the questionnaire assessment. Nine children (four male, five female) were selected from the GMS list of a GP in a low-income area and eleven (three male, eight female) from a fee-paying primary school in a high-income area. Results are shown in the Table.

					Ca				Fibre			
			Energy (MJ/d)		FFQ (mg/d)		WI (mg/d)		FFQ (g/d)		WI (g/d)	
		n	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Elderly	Male	16	—		745	371	—		23·2	7·1	—	
	Female	16	—		475	193	—		13·2	3·9	—	
Adolescents	Male	7	8·6	2·3	885	536	989	337	20·0	8·0	20·5	7·8
	Female	13	6·9	1·9	660	413	824	335	17·3	4·0	16·8	5·7
Adolescents	GMS	9	8·6	1·9	908	468	1001	254	19·8	4·5	19·2	5·6
	Fee	11	6·7	2·1	621	445	781	396	17·3	6·8	17·2	5·0

FFQ, food frequency questionnaire; WI, average 7 d weighed intake.

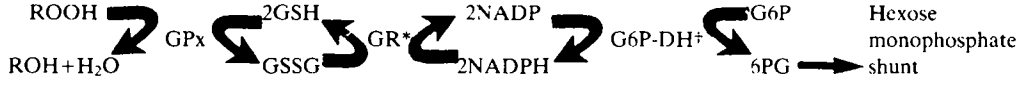
Although the CA intake of adolescents as determined by FFQ correlated well with weighed intake estimates (Pearson's correlation coefficient, r 0·79), there was 40% misclassification (by quartiles), most marked in individuals consuming little dairy produce or a lot of fruit. Overall, the FFQ underestimated Ca intake by 14·9%. FFQ determinations of fibre intake in this age group correlated less well with weighed intake estimates (r 0·28) displaying greater misclassification (60%), especially when fibre intake was high, despite mean overestimation by only 1·8% overall. In view of these findings, it is not possible to make valid comparisons of fibre intakes in our study groups.

In keeping with the results of other studies, Ca intakes appeared greater in adolescents than in the elderly and greater in males than in females in both age groups. However, when Ca intakes in adolescents were corrected for energy intake the sex difference disappeared (Ca intake (WI) in mg/10MJ: males, n 7, mean 1146, SD 775; females, n 13, mean 1194, SD 674). Adolescents from GMS-eligible families had higher energy and Ca intakes than those from fee-paying schools, but energy-adjusted Ca intakes were similar.

Nelson, M., Hague, G. F., Cooper, C. & Bunker, V. W. (1988). *Journal of Human Nutrition and Dietetics* **1**, 115–127.

Tissue activities of glutathione peroxidase (*EC* 1.11.1.9) and related enzymes in cattle depleted of vitamin E or selenium, or both. By D. M. WALSH, S. KENNEDY and D. G. KENNEDY, *Veterinary Research Laboratories, Stormont, Belfast BT4 3SD, Northern Ireland*

Dietary deficiencies of vitamin E (E) and selenium are etiopathogenetically important factors in nutritional degenerative myopathy development. Low dietary Se reduces tissue activity of the seleno-enzyme glutathione peroxidase (*EC* 1.11.1.9; GPx). This enzyme uses reduced glutathione (GSH) to effect the reduction of peroxides. An adequate supply of GSH is therefore essential for the normal action of GPx. This is ensured by the efficient functioning of a series of reactions:



*GR, glutathione reductase (NAD(P)H, *EC* 1.6.4.2); †G6P-DH, glucose-6-phosphate dehydrogenase (*EC* 1.1.1.49).

Some members of a group of drug-detoxifying enzymes, collectively referred to as glutathione S-transferase (*EC* 2.5.1.18; GSHT), possess a non-Se-dependent GPx activity. The effects of low dietary concentrations of E or Se, or both, on tissue activities of GPx and related enzymes were investigated.

Four groups of four calves were given diets low in E, low in Se, low in both E and Se, or supplemented with E and Se as described by Walsh *et al.* (1991).

		Heart		M. gluteobiceps		M. supraspinatus		M. masseter	
Group	Analyte*	Mean	SE	Mean	SE	Mean	SE	Mean	SE
1 (+E+Se)	GPx	363[a]	18·5	38[a]	2·25	59[a]	5	160[a]	14·5
	GSHT	32	2·25	31	1·5	39	2	43	1·5
	GR	4·75	1·8	2·0	0·4	3·5	0·55	12·9	0·4
	G6P-DH	5·5[a]	0·55	0·4[a]	0·2	1·85[a]	0·35	1·7[a]	0·25
2 (−E+Se)	GPx	393[a]	61·5	32[a]	7·5	49·5[a]	7·4	84[b]	9·5
	GSHT	28·5	4	37	3	38	3·5	31	5
	GR	3·75	0·95	2·0	0·57	3·75	0·05	10·0	1·65
	G6P-DH	7·45[a]	1·2	1·6[b]	0·25	1·7[a]	0·35	4·4[b]	1·45
3 (+E−Se)	GPx	148[b]	24·5	13[b]	3	23[b]	7	56[b]	9·5
	GSHT	32	2·5	28	3·5	37	4	33	4·5
	GR	5·15	2·05	2·75	0·47	4·25	0·5	12·25	1·05
	G6P-DH	5·9[a]	0·65	0·2[a]	0·05	1·1[a]	0·3	1·5[a]	0·3
4 (−E−Se)	GPx	216[b]	27·75	19[b]	6	28[b]	7·75	57[b]	8
	GSHT	31	2·5	36	3	37	5·5	39·5	4·5
	GR	4·4	1·05	2·6	0·55	3·8	0·65	10·3	0·75
	G6P-DH	6·3[a]	0·3	1·7[b]	0·37	1·8[a]	0·6	2·9[a]	0·97

[a,b] Values for GPx and G6P-DH in the same column but with different superscript letters are significantly different: $P<0·05$.

* All enzyme activities are expressed in international units/g soluble protein.

The results show that tissue GPx activity was dependent on dietary Se, but independent of dietary E concentration. GSHT and GR activities were not significantly affected by dietary concentrations of either nutrient. G6P-DH activities in both biceps and masseter from E-deficient calves were elevated compared with controls. The consequences of this last result are not clear, but would merit further investigation.

Walsh, D. M., Kennedy, S. & Kennedy, D. G. (1991). *Proceedings of the Nutrition Society* **50**, 68A.

The effect of garlic on blood lipids and fibrinolytic activity. By K. M. SHEIL and K. M. YOUNGER, *Division of Nutritional Sciences, Department of Clinical Medicine, Trinity College Medical School, St James's Hospital, Dublin 8, Republic of Ireland*

Garlic preparations are now widely available and are advocated as a 'natural' means of reducing risk of cardiovascular disease (CVD). Garlic supplementation has been reported to lower serum cholesterol and serum triacylglycerol (Bordia, 1981), and although this effect was not observed by Arora *et al.* (1981) they did find garlic supplementation to be associated with an increase in blood fibrinolytic activity. The aim of the present study was to investigate the effect of garlic supplementation for 4 weeks in healthy volunteers on components of blood implicated in CVD. These included apoprotein Al, apoprotein B and lipoprotein (a) (Apo Al, Apo B, and Lp(a)) which are now considered to be better indicators of disease risk than low-density and high-density lipoproteins (LDL and HDL) (measured in earlier studies). Also, as an indicator of blood fibrinolytic activity, tissue plasminogen activator (tPA, involved in clot lysis and hence important in prevention of thrombosis in CVD) was measured.

Thirteen healthy volunteers (seven male, six female, mean age 35·2 (SEM 3·13) years, range 23–57 years) were recruited. After measurement of height and weight, they were provided with six 500 mg capsules/d of odour-controlled pure dried garlic (Nature's Way, Sheastown, Co Kilkenny, Republic of Ireland). Compliance was monitored by visits and odour on breath. Diet (3 d record by written recall) was assessed for factors which might affect blood lipids. Fasting venous blood was drawn immediately before and after the 4 week supplementation period and analysed for blood lipids by standard enzymic techniques. Apo Al and Apo B were analysed immunoturbidometrically, and Lp(a) and tPA by ELISA (Biopool AB, Umea, Sweden).

Table. *Blood lipids, apoproteins and tPA antigen before and after garlic supplementation*

	Pre-supplement		Post-supplement		
	Mean	SEM	Mean	SEM	Significance
Triacylglycerol (mmol/l) (*n* 12)	0·99	0·155	0·75	0·139	NS
Total cholesterol (mmol/l)	5·8	0·42	6·0	0·41	NS
HDL cholesterol (mmol/l)	1·4	0·13	1·4	0·08	NS
LDL cholesterol (mmol/l)	3·9	0·41	4·1	0·39	NS
Apo Al (mg/l)	247·3	13·2	301·1	22·6	$P \leq 0{\cdot}05$
Apo B (mg/l)	87·7	9·4	107·2	10·2	$P \leq 0{\cdot}01$
Apo B:Apo Al	0·37	0·046	0·38	0·044	NS
tPA antigen (ng/ml)(*n* 11)	5·4	0·72	6·6	0·97	$P \leq 0{\cdot}02$
Lp (a) (mg/l)	2032	694	2285	692	NS

NS, not significant, Wilcoxon signed rank test for paired data.

After garlic supplementation, serum total cholesterol, triacylglycerol, HDL and LDL, Apo B:Apo Al ratio and Lp(a) were unchanged. However, a 22% rise in Apo Al was observed and there was a similar rise in Apo B. Fibrinolytic activity also increased (tPA), which, together with the increased Apo Al, can be interpreted as beneficial as regards risk of CVD or thrombosis. Nevertheless, the increased Apo B could be regarded as a cause for concern, and further work is needed in order to establish whether these effects persist once garlic is discontinued. Considering also the side-effects experienced by eight out of thirteen of the subjects (flatulence, alteration in bowel habit, minor gastric upset and nausea) the use of garlic supplements perhaps should be approached with caution.

Arora, R. C., Arora, S. & Gupta, R. (1981). *Atherosclerosis* **40**, 175–179.
Bordia, A. (1981). *American Journal of Clinical Nutrition* **34**, 2100–2103.

The effect of D-fenfluramine on body-weight of lean and obese Aston strain mice. By M. M. MURPHY, C. M. MURPHY and J. F. ANDREWS, *Department of Physiology, Trinity College, Dublin 2, Republic of Ireland*

D-Fenfluramine reduces appetite by central effects on the serotonergic pathway (Neill & Cooper, 1989). In the present study D-fenfluramine was administered chronically in drinking water at the rate of approximately 20 mg/kg per animal per d. Mice were housed at thermoneutrality (30°) in a 12 h light–12 h dark cycle. Aston mice were studied and effects compared in lean and genetically obese animals. Adult animals were either given lab chow (Nutec Ltd) or lab chow supplemented with chocolate (Cadbury, Dairy Milk), both *ad lib.* for 3 weeks before and during drug administration. Chocolate stimulates a hyperphagia with respect to the chow alone (Younger & Trayhurn, 1984). Food intake and body-weight were determined by weighing on a daily basis. Energy intake was calculated from this (chow, 12·1 kJ/g; chocolate, 22·5 kJ/g).

Three weeks on the chocolate-supplemented diet had made the lean animals mildly obese and the genetically obese animals grossly obese before drug treatment. Drug treatment caused weight loss in all animals in the first week, being greater in absolute terms in the lean than in the obese animals but only reaching significance in the lean chow-fed group. Absolute weight loss continued in the second and third weeks of treatment in the lean animals on both diets, albeit at a reduced rate. Weight gain was resumed in the two obese groups but at a much reduced rate compared with rate of weight gain before treatment. D-Fenfluramine caused a considerable reduction in food intake in week 1. In chow-fed animals food intake was partially restored in week 2 and in week 3 fully restored or more. In chocolate-fed animals the drug appeared to have a more sustained effect in reducing food intake.

				Pre-treatment week		Week 1		Week 2		Week 3	
Animal	Diet	*n*		Body -wt*	Food intake†	Body -wt*	Food intake	Body -wt	Food intake	Body -wt	Food intake
Lean	Chow	4	Mean	39	52	36‖	27	36	39	35	69
			SEM	1	9	1	8	1	5	1	5
	Chow +chocolate	4	Mean	46‡	130	44	115	43	70	41	75
			SEM	5	20	4	16	4	12	4	12
Obese	Chow	4	Mean	52	59	50	51	53	59	54	69
			SEM	6	6	6	8	5	4	6	4
	Chow +chocolate	4	Mean	80‡	85	78	63	81	63	83	15
			SEM	8	5	7	4	6	4	7	2

* Body-weight (g) at end of week.
† Food intake (kJ/d) during week.
‡ Significantly different from chow fed by Student's *t* test: $P<0·001$.
‖ Significantly different from pretreatment body-weight: $P<0·01$.

In conclusion, D-fenfluramine stimulated weight loss more effectively in the lean than the obese animals in terms of both short-term and sustained effects. Diet also influenced response, it being greater in association with the chocolate hyperphagia. This is achieved at least in part by reduced food intake, but other factors, possibly increased whole-body metabolic rate must play a part.

D-Fenfluramine was a gift from Servier Laboratories (Irl) Ltd.

Neill, J. C. & Cooper, S. J. (1989). *Psychopharmacology* **97**, 213–218.
Younger, K. M. & Trayhurn, P. (1984). *Irish Journal of Medical Science* **153**, 409.

Malnutrition is an infrequent feature of inflammatory bowel disease in remission. By M. A. O'CONNELL[1], D. KELLEHER[2], D. G. WEIR[2], P. W. N. KEELING[2] and P. FLOOD[1], [1]*Department of Nutrition and Dietetics, St. James's Hospital, Dublin 8,* and [2]*Department of Clinical Medicine, Trinity College, Dublin 2, Republic of Ireland*

It has been reported that active chronic inflammatory bowel disease (CIBD i.e. Crohn's disease and ulcerative colitis) is associated with malnutrition as a result of reduced intake and absorption and increased utilization and intestinal loss of essential nutrients. Studies have suggested that malnutrition may also be associated with the chronic phases of the disease (Hodges *et al.* 1984). However, rational drug therapy has significantly altered both the morbidity and mortality associated with CIBD. The aim of the present study was to perform a comprehensive analysis of the nutritional status of patients with CIBD in remission.

Twelve patients with ulcerative colitis (UC), ten patients with Crohn's disease (CD) and twenty-two healthy controls (HC), age and sex matched with patients, were studied. Control subjects were healthy or in hospital for reasons unconnected with bowel function. There was no significant difference in body mass index (kg/m^2), usual body-weight and ideal body-weight between patients and controls. Mid-arm circumference (CIBD 293 (SD 45) *v.* HC 310 (SD 35) mm), mid-arm muscle area and grip-strength were not significantly reduced in CIBD patients. Indices of body fat, including triceps skinfold thickness (CIBD 15·7 (SD 7·3) *v.* HC 18·3 (SD 8·3) mm), total skinfold thickness (CIBD 48·5 (SD 15·0) *v.* HC 58·3 (SD 25·3) mm), and % body fat were not significantly reduced in patients compared with controls. Six patients and four controls had triceps skinfolds <25th percentile.

Macronutrient intakes per d

	Energy (MJ)		Protein (% energy)		Fat (% energy)		Carbohydrate (% energy)		Fibre (g)	
Group	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
CIBD patients	11·7	2·6	13·6	1·6	40·6	5·1	43·4	5·7	22·0	8·0
Controls	10·7	3·5	14·4	2·2	39·8	5·9	42·7	4·8	24·0	10·0

Dietary intakes of macronutrients and micronutrients were assessed using the dietary history method. Energy intakes, protein % energy, fat % energy and dietary fibre intake were normal in patients with CIBD. Total carbohydrate intake and sugar intake were higher in patients with CIBD but this did not reach statistical significance. Patients' intakes of micronutrients including iron, folic acid, vitamins B_6, B_{12}, C, D and E and calcium did not differ from controls. Biochemical indices of nutrition including albumin, and total protein levels were normal in patients with CIBD. Seven patients (five female, two male) and six controls (all female) had haemoglobin levels below normal (i.e. 130 g/l for males, 120 g/l for females).

The results indicate that patients with CIBD in remission are not significantly malnourished relative to normal controls. Patients with CIBD appear to tolerate a normal intake of dietary fibre. These results suggest that modern, rational pharmacological and nutritional therapy of patients with CIBD in the acute phases minimalizes the development of chronic malnutrition.

Hodges, P., Gee, M. & Grace, M. (1984). *Journal of the American Dietetic Association* **12**, 1460–1464.

Relation between levels of linoleic acid in plasma non-esterified fatty acids and plasma and adipose tissue triacylglycerols in healthy adults consuming normal diets or diets high or low in linoleic acid. By BERNICE CORRIEDAN and MICHAEL J. GIBNEY, *Division of Nutritional Sciences, Department of Clinical Medicine, Trinity College Medical School, St James's Hospital, Dublin 8, Republic of Ireland*

Kearney & Gibney (1989) showed that the level of linoleic acid (18:2) in plasma non-esterified fatty acids (NEFA) did not reflect adipose tissue levels of 18:2 when individuals were placed on either low- or high-linoleic acid diets. The present study extends this line of investigation to take account of habitual intakes of 18:2 and of plasma triacylglycerol 18:2.

Abdominal adipose tissue biopsies were taken from healthy adult volunteers (eight female and six male, aged 19–46 years). An initial fasting blood sample was taken while the subjects were pursuing their habitual diet and two further fasting samples taken each at the end of 2 weeks on a diet high or low in 18:2. The fatty acid compositions of NEFA and both plasma and adipose tissue triacylglycerols were determined by gas–liquid chromatography.

		Fatty acid (% wt/wt)					
		14:0	16:0	16:1	18:0	18:1	18:2
Adipose tissue	Mean	5·0	22·4	4·8	2·4	43·7	16·9
	SD	0·8	1·4	1·8	0·6	2·7	3·7
Low 18:2 intake:							
NEFA	Mean	2·5	27·2	2·2	10·0	37·6	16·8
	SD	0·5	1·1	0·9	2·4	2·8	2·6
Plasma triacylglycerol	Mean	3·7	26·9	4·2	3·4	41·5	15·5
	SD	0·9	3·1	1·9	0·9	4·3	4·0
High 18:2 intake:							
NEFA	Mean	2·4	26·1	2·2	6·2	38·9	20·1
	SD	0·4	1·6	1·1	2·3	3·4	2·8
Plasma triacylglycerol	Mean	2·1	24·5	2·2	2·4	37·4*	27·1*
	SD	0·5	3·2	0·9	0·8	5·6	6·2
Habitual 18:2 intake:							
NEFA	Mean	3·0	27·1	2·7	8·3	38·9	17·4
	SD	0·8	1·8	1·0	2·4	3·1	3·8
Plasma triacylglycerol	Mean	4·1	23·4	2·5	3·6	40·9*	20·6*
	SD	1·4	4·1	1·1	1·3	3·4	5·3

* Significantly different from adipose tissue ($P<0·05$).

As with the previous study (Kearney & Gibney, 1989) no significant differences were found between adipose tissue 18:2 and that of NEFA 18:2 on the low 18:2 diet, while with the high 18:2 diet, the differences were significantly greater ($P<0·05$). In the present study no differences were observed in 18:2 levels between adipose tissue and NEFA with habitual diets. A similar pattern was found for 18:2 between adipose tissue and plasma triacylglycerols. NEFA 18:2 was significantly correlated (r 0·88, $P<0·05$) with adipose tissue 18:2 on the habitual diet but not on either the low (r 0·22) or high (r 0·26) 18:2 diets. A broadly similar pattern was found for the relation between NEFA 18:2 and plasma triacylglycerol 18:2. It is concluded that on an habitual diet an equilibrium exists between the levels of 18:2 in NEFA and in adipose tissue and plasma triacylglycerols.

Kearney, J. & Gibney, M. J. (1989). *Proceedings of the Nutrition Society* **48**, 29A.

The effect of iron status on antioxidant enzyme activities in male and female rats. By HEATHER E. BRISTOW, J. J. STRAIN and R. W. WELCH, *Biomedical Sciences Research Centre, University of Ulster at Jordanstown, Newtownabbey, Co. Antrim BT37 0QB, Northern Ireland*

Kawabata *et al.* (1989) suggested that iron loading of mice stimulates lipid peroxidation and oxidation of glutathione. This study investigated the effects of Fe on antioxidant enzymes in rats. Two groups (*n* 6) of female and male weanling Sprague-Dawley rats were housed individually, and were fed *ad lib.* on synthetic diets containing 15 mg or 400 mg Fe/kg. Haemoglobin (Hb), transferrin saturation (TS) and hepatic Fe were measured as indices of Fe status. The antioxidant enzymes plasma caeruloplasmin (*EC* 1.16.3.1, CPL), whole blood glutathione peroxidase (*EC* 1.11.1.9, GSH-Px), erythrocyte glucose-6-phosphate dehydrogenase (*EC* 1.1.1.49, G6PDH) and superoxide dismutase (*EC* 1.15.1.1, SOD), hepatic total SOD, catalase (*EC* 1.11.1.6, CAT), GSH-Px, G6PDH, glutathione-S-transferase (*EC* 2.5.1.18, GST) and glutathione reductase (*EC* 1.6.4.2, GR) activities were assayed, together with hepatic malondialdehyde (MDA) as a measure of lipid peroxidation.

	Low-Fe diet				High-Fe diet				Main effects		Inter-action
	Female		Male		Female		Male				
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Fe	Sex	Fe×sex
Whole blood											
Hb (g/l)	113	2	93	3	132	4	127	3	***	***	*
GSH-Px (U/g Hb)	852	65	872	70	734	27	792	80	NS	NS	NS
Plasma											
CPL (U/l)	209	14	123	9	186	20	128	14	NS	***	NS
TS (%)	41	7·2	19	5·7	55	4·0	39	1·4	**	**	NS
Erythrocyte											
G6PDH (mU/mg protein)	68	13	80	7	29	9	54	8	**	NS	NS
SOD (U/mg protein)	16	0·75	14	0·41	13	0·46	14	1·3	NS	NS	NS
Liver											
GST (U/mg protein)	0·78	0·05	0·97	0·05	0·69	0·04	0·85	0·07	*	**	NS
GSH-Px (U/mg protein)	0·20	0·02	0·21	0·02	0·21	0·02	0·23	0·03	NS	NS	NS
GR (mU/mg protein)	9·1	2·1	8·6	1·3	5·9	1·2	8·7	2·9	NS	NS	NS
CAT (U/mg protein)	0·39	0·06	0·71	0·08	0·44	0·06	0·68	0·09	NS	**	NS
Total SOD (mU/mg protein)	2·3	0·09	2·1	0·06	2·3	0·11	2·2	0·10	NS	NS	NS
Fe (μg/g)	141	20	111	8	1155	267	304	30	***	***	***
MDA (μmol/g)	2·1	0·29	1·5	0·53	2·9	0·74	3·7	1·06	NS	NS	NS

NS, not significant.
Significantly different by two-way analysis of variance: *$P<0.05$; **$P<0.01$; ***$P<0.001$.

Results indicated that Fe status was significantly higher in rats fed on the high-Fe diet and that Fe status was significantly higher in females. Female rats also had significantly higher CPL and lower GST and CAT activities than males. Although some antioxidant and related enzymes were significantly decreased in the rats fed on the high-Fe diet, there were no significant differences in hepatic MDA levels.

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Estimation of energy expenditure by heart-rate monitoring in healthy children and adolescents: a validation study. By M B. E. LIVINGSTONE[1], A. M. PRENTICE[2], W. A. COWARD[2], P. S. W. DAVIES[2], C. A. MAHONEY[1], J. A. WHITE[1] and J. J. STRAIN[1], [1]*Biomedical Sciences Research Centre, University of Ulster, Coleraine, Northern Ireland* and [2]*MRC Dunn Nutrition Unit, Cambridge*

We have previously demonstrated good agreement between group estimates of total energy expenditure (TEE) in adults measured by the doubly-labelled water (DLW) method and heart-rate (HR) monitoring (Livingstone *et al.* 1990).

In the present study TEE was measured simultaneously in thirty-six (nineteen male, seventeen female) free-living healthy children aged 7, 9, 12 and 15 years over 10–15 d by the DLW method and between two and three separate days by HR monitoring. Individual HR-oxygen consumption ($\dot{V}O_2$) calibration curves were derived from five varying levels of activity, and a 'FLEX' HR which discriminated between resting and exercise HR was identified (Livingstone *et al.* 1990). Daytime HR was monitored over 16 h periods. Individual calibration curves were used to assign an energy value to minute-by-minute recorded HR above FLEX HR. For periods of inactivity below the FLEX HR where HR is known to be a poor predictor of $\dot{V}O_2$, energy turnover was estimated from individually derived values for resting metabolic rate (RMR). This was calculated as the mean of the $\dot{V}O_2$ for supine, sitting and standing activities. Sleeping energy expenditure was assumed to be equal to measured basal metabolic rate (BMR).

Age (years)	*n*	FLEX HR		RMR:BMR		DLW TEE (MJ/d)		HR TEE (MJ/d)	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
7	11	96	6	1·17	0·06	7·60	1·45	7·30	1·44^{NS}
9	9	91	5	1·18	0·04	8·89	1·33	8·13	1·11***
12	10	93	4	1·14	0·03	10·29	0·99	10·15	1·39^{NS}
15	6	89	6	1·22	0·08	10·27	2·00	10·68	2·32^{NS}

NS, not significant.
Significantly different from DLW TEE (paired *t* test): ***$P<0·001$.

The mean difference between HR TEE and DLW TEE was −0·28 (SED 0·14) MJ/d (paired *t* test 1·93, not significant).

Although individual estimates of HR TEE lack precision, these results confirm that HR monitoring is a cost-effective, objective and socially acceptable method for providing a close estimation of the TEE of population groups.

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Energy expenditure by minute-by-minute heart-rate in cystic fibrosis patients. By A. O'RAWE and J. A. DODGE, *Department of Child Health, Queen's University, Institute of Clinical Science, Grosvenor Road, Belfast BT12 6BJ, Northern Ireland*, A. O. B. REDMOND, *Royal Belfast Hospital for Sick Children, Belfast*, M. B. E. LIVINGSTONE, *Biomedical Sciences Research Centre, University of Ulster, Coleraine*, and P. S. W. DAVIES, *MRC Dunn Nutrition Unit, Cambridge*

Measurements of basal metabolic rate (BMR) and total energy expenditure (TEE), were made in thirty mild and moderately affected cystic fibrosis (CF) patients, aged 6–26 years (twenty male, ten female). TEE was measured in free-living individually calibrated subjects, using the minute-by-minute heart-rate method (HR TEE) over 2–3 d, as previously described (Livingstone *et al.* 1990). In addition, simultaneous assessment was made in four subjects over a 15 d period by the doubly-labelled water method (DLW TEE) and heart-rate monitoring over 6–7 d. There was close association between the two methods (Table 1), HR TEE appearing to provide an objective estimation of TEE in the disease state.

Table 1. *Comparative values for HR TEE and DLW TEE*

Patient	Age (years)	HR TEE (MJ/d)	DLW TEE (MJ/d)	% difference
A	17	13·9	12·97	−7
B	13	15·2	15·31	+0·7
C	10	7·1	7·62	+7·2
D	19	9·79	10·82	+10·82

Increased energy expenditure is documented in CF. Patients homozygous for the common mutation (DF 508) have a median BMR of 25% above predicted (95% confidence limits 6–36%). Those heterozygous for this mutation demonstrate a median BMR of +10% (3–18%) and those without the common mutation have a median BMR of +2% (−14 to +19%) (O'Rawe *et al.* 1990).

We wanted to examine physical activity levels (PAL) to determine adequate energy intake in free-living subjects. There was no significant difference in PAL from those of healthy children (Food and Agriculture Organization/World Health Organization/United Nations University, 1985).

		Age (years)		HR (beats/min)		PAL (HR TEE/BMR)	
	n	Mean	SD	Mean	SD	Mean	SD
Male	20	15·3	4	98	11	1·79	0·21
Female	10	13·4	4	101	10	1·64	0·34

Thus, although CF patients with the DF 508 mutation may demonstrate elevated BMR (O'Rawe *et al.* 1990), they appear to have normal levels of physical activity.

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Effect of antioxidant vitamin supplementation on the peroxyl radical trapping ability of plasma. By C. W. MULHOLLAND, *Ulster Hospital, Dundonald, Belfast BT16 0RH* and J. J. STRAIN, *Biomedical Sciences Research Centre, University of Ulster, Coleraine BT52 1SA, Northern Ireland*

Experimental evidence obtained by several groups (Barclay *et al.* 1983; McCay, 1985) suggests that there is a synergistic relation between ascorbate and α-tocopherol, at least in in vitro systems. These studies, however, have had to rely on models utilizing either simple, single-phase systems or lipid micelles. It has yet to be determined if an interaction between these vitamins occurs in vivo. The present study attempted to demonstrate this synergism ex vivo and to obtain quantitative data on any such interaction.

Thirty-two subjects, sixteen males and sixteen females (age 18–40 years) were randomized into four groups of eight. Each group received the following for a period of 28 d: group 1 (control) placebos; group 2, 1 g ascorbate/d and placebo; group 3, 1000 mg α-tocopherol/d and placebo; group 4, 1000 mg α-tocopherol and 1 g of ascorbate/d. Blood samples were collected on day 0 and day 29 and analysed for ascorbate, α-tocopherol and for radical trapping ability using the TRAP technique (Wayner *et al.* 1988).

Group . . .		1		2		3		4	
	Day	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
α-tocopherol (μmol/l)	0	23·6[a]	1·7	26·5[a]	2·4	29·3[a]	2·2	27·8[a]	1·5
	29	23·0[a]	1·7	26·7[a]	2·5	56·9[b]†	3·8	53·0[b]†	4·4
Ascorbate (μmol/l)	0	76[a]	7·1	62[a]	10·2	82[a]	9·5	79[a]	4·0
	29	69[a]	7·6	115[b]†	13·1	62[a]	7·8	104[b]†	9·5
TRAP (μmol/l)	0	1065[a]	52·3	905[a]	37·1	974[a]	88·7	1037[a]	75·1
	29	1069[a]	108·2	1165[a]†	79·9	1168[a]†	53·4	1178[a]	101·6

[a–b] Means in horizontal rows with different superscript letters are significantly different by Newman Kuels test: $P<0·05$.

† Significantly different from day 0 at $P<0·01$ (paired t test).

Although analysis of variance did not demonstrate any significant difference in post-supplement TRAP values between the four groups, there was a significant difference between pre- and post-supplement TRAP values in groups 2 and 3 but not in group 4. This finding does not necessarily exclude a synergistic relationship as the increase in experimental TRAP values in groups 2 and 3 were much larger than those expected from the increases in the plasma concentration of ascorbate and α-tocopherol in each group respectively.

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Homocysteine toxicity towards human cell lines. By G. MAZDAI, B. M. HANNIGAN and J. J. STRAIN, *Biomedical Sciences Research Centre, University of Ulster, Coleraine BT52 1SA, Northern Ireland*

Homocysteine (Hcy), the non-essential sulphydryl amino acid metabolite of methionine, is considered to be an atherogenic agent and recent reports suggest that mild to moderate homocysteinaemia is associated with premature vascular disease (Brattström *et al.* 1988). Hcy has also been shown to lower copper status in rats (Brown & Strain, 1990) whilst the reaction between Hcy and Cu resulting in the formation of hydrogen peroxide was first demonstrated by Starkebaum & Harlan (1986).

The present study investigated the interaction of Hcy with a number of trace elements and minerals and the combined effect of these on the growth of three cell lines. Hcy at 350 μM in combination with Cu (6 μM) as either cupric chloride ($CuCl_2$), Cu-histidine, or Cu-albumin were found to retard growth of Molt-3 (T-lymphoid), Raji (B-lymphoid) and HL-60 (myeloid) leukaemic cell lines. Initial cell concentrations were 4×10^5 cells/ml in the culture medium (RPMI–1640+10% fetal calf serum) and cell growth was determined after incubation for 48 h. Molt-3 cells were the most sensitive and HL-60 cells were the least sensitive to Hcy and Cu combinations. The percentage growth of the treated (Hcy and $CuCl_2$) cells compared with respective controls where saline (9 g sodium chloride/l) only was added to the culture medium are given in the Table. Catalase (*EC* 1.11.1.6) activity in freeze–thawed cells was measured by the method of Abei (1984). In duplicate experiments catalase activities were found to be lowest in Molt-3 cells and highest in HL-60 cells.

Zinc chloride, magnesium chloride, calcium chloride and ferrous and ferric chlorides (at equivalent molarities to that of $CuCl_2$) in combination with Hcy did not cause any profound growth retardation on the Molt-3 cells (the most sensitive of the three cell lines). Also when applied individually, neither Hcy or Cu had any inhibitory effect on the growth of these cells.

	Molt-3		Raji		HL-60	
	Mean	SD	Mean	SD	Mean	SD
Catalase activity (mU/ml per 10^7 cells)	6·38	0·82	12·00	3·76	45·39	9·21
% Growth of the treated cells (350 μM Hcy + 6 μM $CuCl_2$) compared with respective controls	2·54	1·46	48·70	2·42	82·54	17·00

The results suggest that Hcy and Cu react together to decrease cell viability, perhaps by formation of H_2O_2, which was detected in the culture medium at approximately 1 μM by the method of Pick & Keisari (1980). It is postulated that H_2O_2 may play a central role in cellular damage as intracellular catalase activity appeared to be correlated with cellular growth rate in the presence of Hcy/Cu. Thus a significant part of the cell's defence against oxidant damage may well lie in the competence of its endogenous systems to detoxify excessive amounts of H_2O_2.

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Insulin-like growth factor 1 concentrations in serum, colostrum and milk of ewes with single, twin and triplet lambs. By A. R. G. WYLIE, *Food and Agricultural Chemistry Research Division, Department of Agriculture for Northern Ireland and The Queen's University of Belfast, Newforge Lane, Belfast BT9 5PX* and D. M. B. CHESTNUTT, *Agricultural Research Institute of Northern Ireland, Hillsborough, Co. Down and the Department of Agriculture for Northern Ireland and The Queen's University of Belfast*

Insulin-like growth factor 1 (IGF-1) concentration in colostrum may be relevant to intestinal protein synthesis in the suckled neonate (Mirand *et al.* 1990) and thereby to gut closure. Maternal serum IGF-1 has been linked to fetal number and sex in heifers (Holland *et al.* 1988) and may influence mammary IGF-1 secretion. The present study compares IGF-1 levels in serum, colostrum and milk of animals with widely different fetal loads.

Forty Greyface (Blackface × Border Leicester) ewes were oestrus synchronized and crossed with Suffolk rams. An *ad lib.* precision-chop, grass silage diet was supplemented with rolled barley (300 g/ewe per d) 4 weeks before lambing, and rolled barley (600 g/ewe per d) and fish meal + soya-bean meal (500 g crude protein/kg; 70 g/ewe per d) in the final 2 weeks. Ewes were blood-sampled on day 140 of pregnancy (2nd–5th parity) and induced on day 142. Colostrum was sampled immediately after lambing and defatted. Lambs were sexed and weighed. IGF-1 analysis was by radio-immunoassay of neutralized, acid–ethanol extracts. Correlation analysis was by multiple linear regression after adjustment for number of lambs and parity where appropriate.

				IGF-1 (ng/ml)					
		Lamb wt (kg)		Day 140 serum		Colostrum		Colostrum protein (g/l)	
No. of lambs	*n*	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
1	12	5·67	0·151	177	15·1	503	83·4	184	11·3
2	24	4·41	0·107	182	10·6	603	59·0	231	8·0
3	4	3·41	0·262	176	26·1	454	144·5	210	19·5
Significance of difference		***		NS		NS		**	

NS, not significant.
**$P<0·01$, ***$P<0·001$.

Mean colostral IGF-1 (520 ng/ml) and range (230–1235 ng/ml) exceeded those of Simmen *et al.* (1988) for colostrum taken up to 12 h post partum (200–500 ng/ml). Mean milk IGF-1 was 15·6 ng/ml after 5 d. Neither day 140 serum IGF-1 nor colostral IGF-1 were related to number, total weight or mean weight of lambs. Colostral IGF-1 was correlated with both colostral protein (r 0·34; $P<0·05$) and day 140 serum IGF-1 (r 0·51; $P<0·001$) and day 140 serum IGF-1 levels increased ($P<0·05$) with the proportion of female lambs.

The results suggest that fetal number or mass do not influence maternal or colostral IGF-1 levels.

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