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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

A meeting of the Nutrition Society (Scottish Group/Energy and Protein Group) was held at Craigie College of Education, Ayr, on Thursday and Friday, 2/3 April 1992, when the following papers were presented.

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Site-specific differences in the fatty acid composition of human adipose tissue. By P. C. CALDER[1], D. J. HARVEY[1], J. EVANS[2], C. M. POND[3] and E. A. NEWSHOLME[1], [1]*Department of Biochemistry, University of Oxford, Oxford OX1 3QU*, [2]*Derriford Hospital, Plymouth PL6 8DH* and [3]*Department of Biology, The Open University, Milton Keynes MK7 6AA*

There have been several studies of the fatty acid composition of adipose tissue from different regions of the human body. However, many previous studies have been limited to a small number of sites, chosen for their surgical accessibility rather than for site-specific properties, and the results have been contradictory. Therefore, it was of interest to compare the fatty acid composition of adipose tissue from many different sites in humans.

Adipose tissue samples were collected as soon after death as possible (<24 h) from seven males (aged 25–70 (mean 43) years) who had died accidently. The anatomical sites sampled were the intra-abdominal depots, perirenal, omental and mesenteric, the superficial depots, buttock, breast, clavicular, superficial abdominal, anterior thigh, medial thigh, posterior arm and anterior arm (all *n* 7), and the intermuscular depots, popliteal (*n* 7), calf and trapezius (both *n* 6) and pericardial (*n* 5). These depots are homologous with those studied previously in rodents. Samples were kept frozen until lipid extraction and fatty acid analysis which were performed as described elsewhere (Calder *et al.* 1990).

In all sites from all individuals, seven fatty acids, myristic (14:0), palmitic (16:0), stearic (18:0), palmitoleic (16:1*n*-7), oleic (18:1*n*-9), *cis*-vaccenic (18:1*n*-7) and linoleic (18:2*n*-6), comprised over 90% of the fatty acids; in most samples a further twenty-two fatty acids could be identified. In all samples, oleic acid was the most abundant fatty acid (approximately 35–45%), followed by palmitic acid (approximately 20–35%). There was variation in the fatty acid composition between different individuals, probably arising from differences in diet. Some consistent site-specific features were detectable. The adipose tissue of the calf had the lowest content of myristic, palmitic and stearic acids, a low content of linoleic acid and the highest content of palmitoleic and oleic acids. Pericardial adipose tissue had the highest content of palmitic acid, a low content of oleic acid and the lowest content of linoleic acid. There were no statistically significant site-specific differences in the content of myristic, palmitic or linoleic acids. However, some site-specific differences in the contents of palmitoleic, oleic and stearic acids were statistically significant ($P<0{\cdot}05$).

Fatty acid	Site-specific differences
Palmitoleic	Calf>Trapezius, Perirenal, Mesenteric
Oleic	Calf>Trapezius, Perirenal, Breast
Stearic	Calf<Trapezius, Perirenal, Mesenteric, Breast, Clavicular Buttock<Perirenal, Mesenteric, Clavicular Anterior thigh<Perirenal

These data confirm previous conclusions that the fatty acid composition is similar in most of the large superficial adipose depots, but they also show that minor depots such as calf, trapezius and perirenal have specialized features. The homologous depots in rodents have specialized metabolic properties.

Calder, P. C., Bond, J. A., Harvey, D. J., Gordon, S. & Newsholme, E. A. (1990). *Biochemical Journal* **269**, 807–814.

Adipose tissue fatty acids: risk factors for coronary heart disease (CHD)? By C. BOLTON-SMITH, M. WOODWARD (Honorary Research Fellow), R. TAVENDALE and H. TUNSTALL-PEDOE, *Cardiovascular Epidemiology Unit, University of Dundee, Ninewells Hospital and Medical School, Dundee DD1 9SY*

Studies have supported the view that low adipose tissue (AT) levels of linoleate (18:2) may be markers of or risk factors for CHD (Wood *et al.* 1987; Tavendale *et al.* 1992). Recently, no significant difference in CHD risk was observed according to level of polyunsaturated fat in the diet (Bolton-Smith *et al.* 1992). This report investigates whether AT linoleate may be more discriminatory.

Men (*n* 2185; 40–59 years) who participated in the Scottish Heart Health Study provided an AT sample from their outer upper arm (3 mm punch biopsy; Tavendale *et al.* 1992). Diet was assessed by a food frequency questionnaire and subjects were categorized as: diagnosed CHD (*n* 151), undiagnosed CHD (*n* 320), or controls (*n* 1640) as described previously (Bolton-Smith *et al.* 1992). Odds ratios (OR) and 95% confidence intervals (CI) for the risk of diagnosed and undiagnosed CHD were calculated relative to the lowest quintile (Q) of AT fatty acids using multiple logistic regression analysis. OR were adjusted for total blood cholesterol, diastolic blood pressure, smoking, age, BMI, and dietary per cent energy from linoleate and saturated fat.

	Diagnosed CHD							
	16:1		18:0		18:1		18:2	
Quintile	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Q2	1·15	(0·61, 2·19)	0·71	(0·41, 1·20)	2·63	(1·37, 5·03)	0·58	(0·32, 1·05)
Q3	2·10	(1·13, 3·91)	0·45	(0·28, 0·84)	2·04	(1,04, 3·99)	0·56	(0·31, 1·03)
Q4	1·41	(0·74, 2·70)	0·56	(0·31, 0·99)	2·24	(1·15, 4·35)	0·29	(0·14, 0·59)
Q5	2·89	(1·54, 5·41)	0·31	(0·16, 0·59)	2·73	(1·39, 5·36)	0·55	(0·29, 1·07)
	$P<0·01$		$P<0·01$		$P<0·05$		$P<0·05$	
	Undiagnosed CHD							
	16:1		18:0		18:1		18:2	
Quintile	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Q2	0·69	(0·45, 1·06)	0·61	(0·41, 0·91)	1·07	(0·69, 1·65)	0·71	(0·48, 1·05)
Q3	0·77	(0·50, 1·17)	0·62	(0·42, 0·93)	1·03	(0·67, 1·59)	0·71	(0·48, 1·05)
Q4	1·16	(0·78, 1·73)	0·82	(0·55, 1·20)	1·20	(0·78, 1·84)	0·37	(0·23, 0·58)
Q5	1·39	(0·93, 2·09)	0·68	(0·45, 1·03)	1·82	(1·21, 2·74)	0.47	(0·29, 0·76)
	$P<0·01$		NS		$P<0·05$		$P<0·001$	

P, significance level (Chi-square); NS, not significant.

AT fatty acids showed significant effects on the risk of CHD before and after adjustment for dietary fats. High AT 18:2 and low 18:1 were associated with reduced risk of CHD and this requires further investigation.

Bolton-Smith, C., Woodward, M. & Tunstall-Pedoe, H. (1992). *European Journal of Clinical Nutrition* **46**, 75–84.

Tavendale, R., Lee, A. J., Smith, W. C. S. & Tunstall-Pedoe, H. (1992). *Atherosclerosis* (In the Press).

Wood, D. A., Riemersma, R. A., Butler, S., Thomson, M., MacIntyre, C., Elton, R. A. & Oliver, M. F. (1987). *Lancet* **i**, 177–183.

Lipid content and fatty acid profiles of pig meat. By D. B. LOWE, A. J. KEMPSTER, M. W. FOGDEN and C. F. WHITE (Introduced by J. P. LAMBERT), *Meat and Livestock Commission, Winterhill House, Snowdon Drive, Milton Keynes MK6 1AX*

A study was carried out to update the information on the chemical composition of pig meat required to advise consumers on dietary issues and as a basis for labelling legislation. A sample of sixty carcasses structured by sex (entire males, castrated males and gilts), carcass weight (52, 72 and 92 kg) and P_2 fat thickness (fat depth measured at the last rib) was taken from four commercial abattoirs.

At the same carcass weight and P_2 fat thickness, entire males had more lean tissue and less fatty tissues in the side than the castrates or gilts. The three sexes also differed in their fatty acid profile. Castrates had less mono- and polyunsaturated fats in all of the joints and the combined side. Gilts and entire males are equally represented in the slaughter population, castrates now comprising less than 5% of the slaughter population, so averaged over the two sexes the fatty acid profile of a typical side is shown in the Table.

Lipid content (g/kg) and fatty acid profile (% total fatty acids) of the side tissue

	Lean		Lean and intermuscular fat		Lean and fatty tissues	
	Mean	SE	Mean	SE	Mean	SE
Lipid content . . .	37	1·5	79	2·6	194	6·1
Fatty acid						
14:0	—		1·5	0·05	1·6	0·05
16:0	—		24·6	0·43	24·5	0·45
16:1	—		2·2	0·12	2·1	0·13
18:0	—		14·2	0·38	14·1	0·36
18:1	—		34·1	0·88	34·4	0·92
18:2	—		13·1	0·78	13·7	0·89
18:3	—		1·4	0·12	1·5	0·12

There is also variation in composition within the side. The lipid content ranges from 13% in the lean and fatty tissues of the ham joint to 24% in the streak joint.

Over the past 10 years the average pig carcass slaughtered has become physically leaner: the separable fat in the side has decreased by 6% (Kempster *et al.* 1986). Based on the regression equations from the current data, the lipid content of all the tissues has also decreased from 5·2% to 3·7% in the lean, and from 75·1% to 69·7% in the fat. The P:S ratio for lipid in the fat now stands at 0·45, the value recommended for the total diet in the COMA report (DHSS, 1984).

Department of Health and Social Security (1984). *Committee on Medical Aspects of Food Policy. Report on Health and Social Subjects* no. 28. London: H.M. Stationery Office.

Kempster, A. J., Cook, G. L. & Grantley-Smith, M. (1986). *Meat Science,* **17,** 107–138.

Factors affecting the freezing point depression of milk. By H. MOHAMMEDI, P. C. THOMAS and M. I. BARCLAY, *Scottish Agricultural College, Auchincruive, Ayr KA6 5HW*

It is widely accepted that the average freezing point depression (FPD) of cow's milk is normally approximately −543m°H, and a value above −530m°H is regarded as indicative of the presence of extraneous water. However, there are a number of documented field observations of herd milks providing 'abnormal' FPD values where extraneous water contamination is not implicated (FERNS, personal communication).

To investigate further the natural variations in FPD values, a.m. and p.m. milk samples were examined from cows under a variety of dietary and management regimes. These samples showed substantial variations in FPD within cows between milkings, within cows between days and between cows. Moreover, there were clear indications that these differences were influenced by diet and management regime.

In more detailed experiments conducted with lactating dairy goats, milk samples were taken at intervals over a period of 6 h with feeding at three different times (Fig. 1), and over a period of 6 h during which the animals were with or without access to drinking water, so as to obtain variation in the pattern of daily water intake. The mean values obtained ranged from −590m°H when water was not accessible to −572m°H when it was, with SD values of 16·93 and 7·49 respectively.

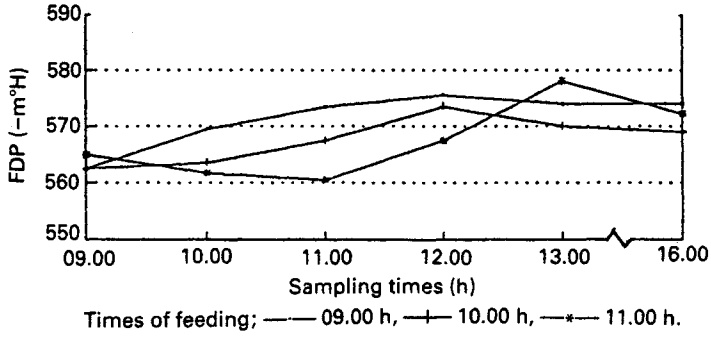

Fig. 1. Effect of time of feeding on the FPD.

Blood samples taken at the times of milking showed a close correlation between blood plasma FPD and milk FPD, confirming earlier observations (Wheelock *et al.* 1966).

The data available suggest that under practical conditions it may be possible to encounter feeding and management regimes which result in 'abnormal' values for FPD in cow's milk without extraneous water being implicated.

Wheelock, J. V., Rook, J. A. F. & Dodd, F. H. (1966). *Journal of Dairy Research* **32**, 79–88.

Absorption of water and solute in the human jejunum from sucrose–electrolyte solutions containing calcium and magnesium. By J. B. LEIPER[1], F. BROUNS[2] and R. J. MAUGHAN[1], [1]*Department of Environmental and Occupational Medicine, Aberdeen University, Aberdeen AB9 2ZD* and [2]*Nutrition Research Centre, University of Limburg, The Netherlands*

Several sports drinks and oral rehydration solutions (ORS) contain small amounts of calcium and magnesium in the belief that replacement of losses during exercise or diarrhoea is necessary. However, Mg in high concentrations is known to induce diarrhoea, and its absorption is thought to be reduced in the presence of Ca. We have measured net water and solute transport from seven sucrose–electrolyte solutions (SES), differing only in the concentrations of Ca and Mg, using a steady-state perfusion technique in the normal fasted human jejunum (*n* 6) on two separate occasions. A triple lumen perfusion set incorporating a mixing segment (150 mm) and a test segment (300 mm) was positioned with the perfusion port just distal to the ligament of Trietz. All solutions contained sucrose (222 mmol/l) and sodium chloride (50 mmol/l). The perfusion rate was 15 ml/min and passage through the mixing segment altered the composition of all test solutions. Sodium and chloride concentrations increased while those of sucrose, Ca and Mg decreased; the changes in the sucrose, Na and chloride concentrations were similar for all SES. Ca and Mg concentrations are shown in the Table.

Solution . . .	A	B	C	D	E	F	G
Ca (mmol/l)	0	1·2	2·0	0	0	1·1	2·1
Mg (mmol/l)	0	0	0	2·2	4·0	3·8	2·2

All solutions were acidic (mean pH (SD) 4·7 (1·4)) and had a similar mean osmolality (341 (SD 13) mOsmol/kg). On the first treatment day the subjects were perfused with solution A and three other SES; on the second day solution A and the remaining three SES were perfused. The order of treatment was randomized. Absorption data were not normally distributed and values are expressed as median (range). Statistical analysis was performed using the Kruskal–Wallis test followed by the Wilcoxon 2-sample rank test where appropriate. Net water absorption in the test segment (ml/cm per h) from solution A was similar on the first (3·6 (2·6–6·3)) and second treatment days (3·8 (3·5–5·7); P=0·925). There was no difference in water absorption (P=0·989) between solutions tested (4·0 (2·6–7·2), overall median). Solute absorption is expressed in μmol/cm per h. Net Na transport (44 (−215–564)) was similar from all solutions (P=0·129), as was carbohydrate absorption (2263 (1275–4497); P=0·984). Ca absorption from solutions C (22 (13–16)) and G (22 (13–54)) was similar (P=0·873), but faster (P<0·05) than that from solutions B (9 (7–28)) and F (9 (7–27)). Mg absorption from solutions E (36 (27–68)) and F (27 (16–61)) was similar (P=0·378), but faster (P<0·01) than that from solutions D (14 (12–22)) and G (12 (8–23)). No net movement of Ca or Mg occurred in solutions which originally did not contain these salts. The presence of Mg did not appear to influence Ca absorption. There was a tendency for Mg absorption to be less when Ca was present in the perfusate, but this was not significant. There was no difference in the relative rates of absorption of Ca and Mg at the same concentration. The highest concentrations of Ca and Mg used in this study are greater than those used in sports drinks and ORS, but even at these levels, Ca and Mg had no effect on water, Na or carbohydrate absorption. The Ca and Mg absorption findings can be most readily explained as the result of solvent drag caused by transport of carbohydrate and Na from the jejunal lumen.

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Effect of dietary molybdenum supplementation or depletion on *Nippostrongylus brasiliensis* infection in the rat. By E. L. ORTOLANI, D. P. KNOX, G. F. J. NEWLANDS and N. F. SUTTLE, *Moredun Research Institute, 408 Gilmerton Road, Edinburgh EH17 7JH*

Since molybdenum is an essential part of the enzyme xanthine oxidase (*EC* 1.1.3.22; XO) that is capable of generating superoxide (O_2^-), and since free radicals play an anthelmintic role against parasites (Kazura & Mesnick, 1984), it was postulated that Mo could increase the generation of superoxide at the site of infection and reduce worm burden.

Three groups of sixteen rats were each fed a commercial cubed diet containing 146·8 μmol Cu/kg dry matter (DM) and 7·4 μmol Mo/kg DM: one group was supplemented with 104·2 μmol Mo/l in the drinking water (+Mo); a second group received two Mo antagonists, i.e. 1·9 mmol/l of tungsten as sodium tungstate and after the fourth day of infection 6·6 mmol/l of allopurinol (−Mo); a third group received no supplements (C). After 40 d the rats, except six in group C (CNI), were infected with 3600 L_3 stage larvae of *N. brasiliensis*. Four rats from each infected group were killed on days 7, 10, 12 and 14 after infection. Two groups of non-infected rats were killed on days 10 and 12. Biochemical indices of inflammation were measured in small intestine homogenates (XO: xanthine dehydrogenase (*EC* 1.1.1.204; XDH) and superoxide dismutase (*EC* 1.15.1.1; SOD) activities and malonyldialdehyde (MDA) concentration) and plasma (mast cell proteinases, RMCPII). Liver Mo concentrations were determined and the number of worms (NW) retrieved on days 7 and 10 were recorded. Food and water intakes were unaffected by infection or other treatments. Key results for infected rats are given in the Table.

	NW Median			XO: XDH Ratio		SOD (U/mg protein)		MDA (nm/mg protein)		RMCPII (μg/ml)		Liver Mo (μm/kg DM)
Days after infection . . .												
	7	10		10	12	10	12	10	12	10	12	7–14
Group												
+Mo	510[b]	0[c]	Mean	0·77[a]	0·90[a]	2·03	2·79[a]	3·17	3·52[a]	5·0[a]	3·4	46·7[a]
			SE	0·04	0·02	0·25	0·40	0·43	0·77	0·4	0·4	3·58
C	900[ab]	9[b]	Mean	0·83[a]	0·86[a]	1·50	1·92[b]	3·15	3·25[a]	4·7[a]	3·6	29·0[b]
			SE	0·08	0·15	0·26	0·15	0·32	0·13	0·6	1·0	1·85
−Mo	941[a]	40[a]	Mean	0·23[b]	0[b]	1·67	0·76[c]	2·53	1·71[b]	2·4[b]	3·6	20·5[c]
			SE	0·23	0	0·40	0·11	0·44	0·24	0·3	0·5	1·70

[a,b,c] Values in the same column with different superscripts were significantly different, $P<0·05$.

The overall mean (SE) values for group CNI were: XO:XDH 0·65 (0·1), SOD 1·64 (0·29), MDA 2·03 (0·12), RMCPII 0·73 (0·15) and Mo concentration 25·8 (0·37). In parasite homogenates, SOD activity (+Mo 4142; C 2052; −Mo 2379; U/mg protein) was higher in those retrieved from the +Mo group. The results show that in infected rats, as Mo status increased, worm burden fell, XO:XDH ratio, SOD and RMCPII activities increased and MDA concentrations rose. The results support the hypothesis that Mo may reduce worm burden by an increase in O_2^- generation. The increments in SOD activities in both intestine homogenates and parasites could reflect adaptation to raised O_2^- concentration.

Kazura, J. W. & Mesnick, S. R. (1984). *Molecular Biochemical Parasitology* **10**, 1–10.

Natural mixed nematode infections can induce sodium deficiency in grazing Finnish Landrace lambs. By C. OOSTERHUIS, K. MCLEAN and N. F. SUTTLE, *Moredun Research Institute, Edinburgh EH17 7JH*

Excessive consumption of minerals by grazing Finnish Landrace (FL) lambs prior to dosing with anthelmintic may have been attributable to salt depletion during nematodiasis (Suttle & Brebner, 1990). Specific effects of nematode infection on sodium status were therefore sought. Four groups of eight or nine FL lambs were allocated to a paddock of high (H) or low (L) larval burden and given (A) or not given (O) anthelmintic (2·5 ml Oramec (MSD AGVET)/kg every 3 weeks). Treatments commenced on June 19 and continued for 18 weeks, lambs being weaned after 4 weeks. Mixed saliva samples were obtained by buccal suction and analysed for Na and potassium using ion specific electrodes. Faecal samples were also taken for parasite egg counts (epg).

Epg were higher in HO than LO lambs but did not indicate large worm burdens, and there was generally a low incidence of diarrhoea and normal Na status for the first 3 months. However, two lambs (A and B) developed very low Na:K ratios in saliva after a rise in *Nematodirus battus* epg (see Table).

Contrasts in N. battus *egg count/g faeces (epg) and saliva Na:K in two lambs from group HO with their cohorts*

	Week . . . Lamb	4	5	6	7	8	9	11
Epg	A	90	450	234	48	72	12	108
	B	522	1026	180	567	864	171	0
	C–H*	3	3	41	15	24	81	108
	SE	2·1	2·5	4·4	13·8	13·8	68·5	102·7
Na:K	A	21·7	7·8	9·7	2·2	1·5	23·5	26·1
	B	23·9	10·8	4·6	1·9	2·0	2·8	14·2
	C–H*	20·5	25·3	23·4	22·4	20·9	24·7	20·4
	SE	5·5	4·4	5·3	6·6	8·2	4·7	7·6

* Mean value for six cohorts.

Diarrhoea developed subsequently in most HO and LO lambs and all but one of the eleven sampled after 18 weeks (in HO, value 28·1) had low saliva Na:K ratios (mean 1·8 (SE 0·38)). By this time *N. battus* was no longer prevalent but other strongyle epg were higher (median epg 1265 in HO, 1823 in LO) than before (peak median epg 598 after 9 weeks in HO). With two exceptions (one in HA, value 2·7; one in LA, value 2·0; both with diarrhoea), saliva Na:K was much higher in lambs given anthelmintic (mean 17·6 (SE 2·0) in HA; *n* 5: 10·5 (SE 3·0) in LA; *n* 4). Highly significant negative relationships were found between final saliva Na:K and both severity of diarrhoea and weight loss for the last month (no data excluded). Shifts in Na:K were caused by reciprocal movements in Na and K of 15 to 75 mmol/l with Na+K constant at 150–160 mmol/l saliva. It is concluded that mild nematode infections can induce Na deficiency in lambs grazing pasture 'adequate' in Na (9–13 g/kg dry matter) and that susceptibility to deficiency increases in the autumn. Intestinal parasites (e.g. *N. battus*) may be essential for Na depletion to occur but the responsible species must change.

Suttle, N. F. & Brebner, J. (1990). *Proceedings of the Nutrition Society* **49**, 220A.

Inhibition of ovine erythrocyte superoxide dismutase activity (ESOD; *EC* 1.14.1.1) in vivo by parenteral ammonium tetrathiomolybdate. By N. F. SUTTLE, J. BREBNER, J. SMALL and K. MCLEAN, *Moredun Research Institute, Edinburgh EH17 7JH*

Tetrathiomolybdate (TTM) (5 μM) strongly inhibits ESOD and caeruloplasmin (CP; *EC* 1.16.3.1) in vitro (Chidambaram *et al.* 1984) but acute effects on ESOD in vivo have not been studied. Nine Texel rams, of 75 kg average live weight (LW) and previously given a compound feed which raised liver copper to 12·6 mmol/kg dry matter (DM), were given a diet low in Cu based on whole barley (Field *et al.* 1988). Two rams were given no TTM (controls) and seven were given ammonium-TTM (Aldrich Chemical Co.) either subcutaneously (s.c.; three sheep, 1·7 mg TTM/kg LW) or intramuscularly (i.m.; four sheep, two at 1·7 and two at 3·4 mg TTM/kg LW): three s.c. doses were given on alternate days and five i.m. doses on consecutive days. After 28 d, three of the TTM-treated rams and one control were given three further i.m. doses of 1·7 mg TTM/kg LW on alternate days. ESOD activity was measured at 30° using an assay kit (Randox Laboratories, Co Antrim) and plasma CP activity by a method modified from Smith & Wright (1974).

In the seven rams initially given TTM, ESOD activity (U/mg Hb) fell to a minimum after 4 d but there was no effect of route or level of administration. Between 7–28 d ESOD activity showed a linear rate of recovery to pre-treatment values. The second course of TTM reduced ESOD activity by 68% after 24 h and 82% after 3 d (see Table); there was no reduction in erythrocyte Cu (data not shown).

Effects on Texel rams of a second series of tetrathiomolybdate injections (TTM; 1·7 mg/kg LW on days 0, 2 and 4) on mean (SE) ESOD and CP activity in erythrocytes and plasma respectively

	ESOD (U/mg Hb)*					CP (o.d./min per l plasma at 37°; 0·5 cm path length)				
Day . . . TTM (2nd series)	0	1	2	3	7	0	1	2	3	7
Controls (*n* 5)	328	310	342	271	286	544	581	551	597	559
SE	14·4	25·0	17·7	14·0	20·3	74·1	97·6	91·9	103·0	92·0
i.m. (*n* 4)	308	98	126	55	58	470	480	507	573	723
SE	7·2	17·0	17·6	3·0	3·6	36·7	41·5	38·1	29·3	81·0

* One unit of activity is that which inhibits the oxidation reaction by 50%.

Inhibition of ESOD activity in washed erythrocytes by TTM (1–10 μm) in vitro was confirmed, but in whole blood ESOD was less inhibited. CP was unaffected by TTM in vivo (see Table) and minimally affected in whole blood in vitro. Plasma binding probably protects ESOD and CP from inhibition and in vitro studies in systems lacking albumin are poor indicators of the physiological properties of TTM. The marked inhibition of ESOD after repeated TTM injections indicates that TTM must be used conservatively in treating Cu toxicity in sheep and man.

Chidambaram, M. V., Barnes, G. & Frieden, E. (1984). *Journal of Inorganic Biochemistry* **22,** 231–239.
Field, A. C., Suttle, N. F., Brebner, J. & Gunn, G. (1988). *Veterinary Record* **123,** 97–100.
Smith, B. S. W. & Wright, H. (1974). *Clinica Chimica Acta* **50,** 359–366.

Slimming with 5 MJ (1200 kcal): 58% *v.* 35% energy from carbohydrate. By M. E. J. LEAN[1], A. AVENELL[2], I. A. ROSS[2], T. RICHMOND[2], T. NICHOLSON[3], and T. PRVAN[3], [1]*Department of Human Nutrition, University of Glasgow,* [2]*Diabetic Clinic, Aberdeen Royal Infirmary* and [3]*Department of Statistics, University of Glasgow*

Overweight women (*n* 110) recruited to a slimming clinic were randomly allocated to two diets which both contained 5 MJ (1200 kcal)/d. The % energy contributions of carbohydrate, fat and protein respectively were 58, 21 and 21 for HC diet and 35, 35 and 30 for LC diet. The effects on weight, body fat content and various coronary risk factors were monitored over a 6 month period. Total drop-outs were 19 at 3 months, 28 at 6 months, with no difference between diets. Smokers (only) and younger subjects (under 50) were more likely to drop out.

Baseline weight was 84 (SD 16) kg, age 51 (SD 14) years. Mean weight loss for the entire study was 6·2 kg and there was no significant difference between the HC diet (95% CI; 4·0, 7·1 kg) and the LC diet (5·2, 8·4 kg). The same pattern was found at 3 months when mean weight loss was 4·9 kg. Various baseline factors were assessed for interaction with diet composition on the effects on weight loss. Age under 50 years was found to interact ($P<0·1$), leading to greater weight loss on the LC diet. For women over 50 years there was no difference in weight loss between the diets.

Total serum cholesterol and LDL-cholesterol showed desirable changes over the study period of weight loss. At 3 months the reductions in both total and serum cholesterol and LDL-cholesterol were greater with the HC diet prescription. At 6 months, when most active weight loss had ceased, the total serum cholesterol remained significantly reduced on the high carbohydrate diet ($P=0·0025$). Serum thyroxine fell significantly on the LC diet from 104 (SEM 3) to 98 (SEM 3) nmol/l at 3 months, while it was maintained at 102 (SEM 3) nmol/l on the HC diet.

		Total cholesterol (mmol/l)						LDL-cholesterol (mmol/l)				
		Baseline		3 months		Significance*		Baseline		3 months		Significance*
Diet	*n*	Mean	SEM	Mean	SEM	*P*<	*n*	Mean	SEM	Mean	SEM	*P*<
HC	42	6·8	0·2	6·4	0·2	0·005	42	4·6	0·2	4·3	0·1	0·03
LC	38	6·8	0·2	6·6	0·2	0·1	36	4·6	0·2	4·6	0·2	NS
				6 months						6 months		
HC	41	6·9	0·2	6·5	0·2	0·003	37	4·7	0·2	4·5	0·2	0·05
LC	38	6·9	0·2	6·8	0·2	NS	34	4·6	0·2	4·6	0·2	NS

* Significance of difference between 3 and 6 month values and the baseline value.
NS, not significant.

In conclusion, slimming diets of widely different macronutrient compositions do not have a major effect on rate of weight loss but there is some evidence to prefer a high carbohydrate composition for its metabolic influence on coronary risk factors.

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Insulin-like growth factor-1 and the fibroblast growth factors as autocrine/paracrine regulators of adipocyte precursor DNA synthesis. By S. C. BUTTERWITH, C. D. PEDDIE, C. GODDARD, I. E. O'NEILL, J. BOSWELL and D. BURT (Introduced by M. MCLEOD), *AFRC Institute of Animal Physiology and Genetics Research, Department of Cellular and Molecular Biology, Roslin, Midlothian EH25 9PS*

Adipocyte hyperplasia occurs by the proliferation and differentiation of adipocyte precursor cells. The mature fully-differentiated adipocyte has no capacity for cell multiplication in vivo (Van, 1985). We have previously demonstrated that the insulin-like growth factors (IGF's), transforming growth factor β and platelet-derived growth factor are stimulators of adipocyte precursor DNA synthesis (Butterwith & Goddard, 1991). In the present study we have investigated the role of acidic and basic fibroblast growth factors (aFGF, bFGF) in the regulation of DNA synthesis and their interaction with (IGF-1). We have also determined whether adipocyte precursor cells in vitro can express IGF-1 or bFGF.

Chicken adipocyte precursor cells were prepared and cultured in Medium 199 (M199) containing 4% Ultroser (Butterwith & Griffin, 1989) for 66 h. They were then washed and incubated in M199 for a further 24 h before addition of growth factors and [^{3}H]thymidine. Incorporation of thymidine into DNA was determined as described previously (Butterwith & Goddard, 1991). Addition of aFGF stimulated DNA synthesis sixfold with a maximum at 100 ng/ml, while bFGF stimulated it fourfold with a maximum at 1 ng/ml. Consistent with our previous studies, IGF-1 stimulated DNA synthesis 1·5–twofold at a concentration of 5 ng/ml. When added together there was a significant synergistic interaction between aFGF and IGF-1 resulting in a stimulation of thirty-twofold (ANOVA $P<0·001$). There was also a synergistic interaction between bFGF and IGF-1 resulting in a stimulation of sevenfold (ANOVA $P<0·001$).

Using a reverse transcriptase polymerase chain reaction technique for bFGF and Northern blotting with a riboprobe for IGF-1 we have demonstrated that both of these growth factors are expressed in proliferating adipocyte precursors. We, therefore, conclude that IGF-1, bFGF and possibly aFGF are potentially important autocrine/paracrine regulators of adipocyte precursor proliferation.

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The indirect effects predicted by a model of adipose tissue metabolism. By J. I. DAVIES and B. M. GRAIL, *School of Biological Sciences, University of Wales: Bangor, Bangor, Gwynedd LL57 2UW*

In order to assess the influence of substrate metabolism on fat deposition in adipose tissue, knowledge is required of both the overall rate of substrate utilization and the relative contributions of the individual pathways involved. We have developed a model which can be used to analyse the latter. Glucose utilization is seen as leading to the production of five major products (triacylglycerol-fatty acids, triacylglycerol-glycerol, lactate, pyruvate and carbon dioxide), the formation of which is due to the activities of six metabolic pathways, each with its characteristic stoichiometry with respect to ATP and NADH. The derived equations include:

$$LA + LB + ET + OX + GL + GP = 1$$

where LA and LB are the proportions of utilized glucose-C entering two lipogenic pathways with different stoichiometries, and ET, OX, GL and GP are the equivalent terms for the fatty acid esterification pathway, the tricarboxylic acid cycle and the glycolytic pathways leading to lactate and pyruvate respectively. Two further equations evaluate the balances between synthesis and utilization in ATP and cytoplasmic NADH (M and N respectively) that result from the metabolism of glucose:

$$26/14\ LA + 122/16\ LB - 14\ ET + 32\ OX + 2\ GL + 2\ GP = M - 3\ N$$

$$2\ OX + 2\ GP - 1/14\ LA - 1/16\ LB - 2\ ET = N$$

The model predicts that the relationships among the pathway proportions LA, LB, etc. have limited flexibility. They are, nevertheless, influenced by the ATP balance, M, which is governed by the activities of a variety of energy-consuming processes including protein synthesis and ion transport. This implies that pathway proportions may be manipulated by agents which have no direct influence on enzymes or the glucose-metabolizing pathways.

Fat deposition rate (FDR), which together with fat mobilization rate (FMR) dictates the overall rate of fat storage, can be expressed as follows:

$$FDR = T\ (1/14\ LA + 1/16\ LB + 2\ ET)$$

where T is the overall rate of glucose utilization. It can be shown that this predicts an inverse relationship between lipogenesis and fat deposition (FDR). It also predicts that fat storage (the difference between FDR and FMR) in adipose tissue can be decreased not only by stimulating lipolysis, but also by increasing the lipogenesis proportions LA and LB, and decreasing total glucose utilization (T), the ATP balance M and the fatty acid esterification proportion ET.

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Factors stimulating proliferation of sheep adipocyte precursor cells. By KATHRYN S. ADAMS, D. J. FLINT and R. G. VERNON, *Hannah Research Institute, Ayr KA6 5HL* and A. CRYER, *Department of Biochemistry, University of Wales, Cardiff CF1 1ST*

Adipocytes develop from small, undifferentiated precursor cells which can proliferate and differentiate into adipocytes in vitro as well as in vivo. Factors regulating the proliferation and differentiation of such precursor cells show species variation. Despite the interest in producing leaner sheep for human consumption, little is known about the factors regulating adipocyte formation in this species.

The stromal-vascular fraction of adipose tissue which contains the precursor cells was prepared as described by Cryer *et al.* (1987) from sheep of various ages from 100 d of gestation to 5 years of age. Cells ($2{\cdot}5\times10^4$) were plated out in 1 ml of Medium 199 containing Earles salts and supplemented with 100 μg/ml streptomycin, 100 IU/ml penicillin, 2 mM-acetate, 4·8 mM-L-glutamine and 20% (v/v) fetal calf serum. Cells were maintained in an atmosphere of 5% carbon dioxide in air at 37°. The culture medium was changed after 24 h and then at further 48 h intervals. Cell numbers were determined microscopically on days 1, 2, 3, 6, 7 and 8 of culture. For studying the effects of hormones and growth factors, the culture medium described above was replaced with DMEM/Hams F12 (1:1) medium (Gibco BRL, Paisley) supplemented with L-glutamine, antibiotics and acetate, as described above, plus 1% (v/v) fetal calf serum, 33 μM-biotin, 10 μg/ml transferrin, 17 μM-pantothenate and 0·5 μl/ml lipid supplement. [^{3}H]thymidine (0·25 μCi/ml) and unlabelled thymidine (100 nmol/ml) were added and the amount of radioactivity incorporated into DNA determined 72 h later by a method based on that of Butterwith & Goddard (1991).

Growth curves showed that for cells grown in 20% fetal calf serum, cell numbers decreased over the first 72 h of culture and then increased exponentially with a doubling time of 0·9 d, regardless of the age of sheep or adipose tissue depot used.

Platelet-derived growth factor (PDGF) and fibroblast growth factor (FGF) both stimulated cell proliferation by themselves and acted synergistically to stimulate cell proliferation with cells from 6-month-old wether sheep (Table).

Disintegrations/min [^{3}H]thymidine incorporated per culture well/72 h

Additions	None	PDGF (20 ng/ml)	FGF (50 ng/ml)	PDGF + FGF
Untransformed data	48[a]	113[b]	87[b]	468[c]
Log-transformed data	1·61	1·89	1·89	2·46

Values are means of six observations; SED (log transformed data) 0·108; [a,b,c] Values with different superscripts differ significantly: ($P<0{\cdot}05$).

Further studies showed that insulin and IGF-2, but not IGF-1 (all at 100 ng/ml), increased ($P<0{\cdot}05$) the rate of DNA synthesis in the presence of PDGF and FGF. Thus in sheep, proliferation of adipocyte precursor cells is under complex endocrine control.

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The effect of triiodothyronine (T_3) treatment on dietary lipid deposition and oxidation in meal-fed and *ad lib.*-fed rats. By MARTHA L. CRUZ and DERMOT H. WILLIAMSON, *Metabolic Research Laboratory, Nuffield Department of Clinical Medicine, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE*

Refeeding of meal-fed rats with a chow meal containing [1-^{14}C]triolein lowers the production of $^{14}CO_2$ and increases the accumulation of [^{14}C]lipid in white adipose tissue (WAT) compared with rats fed *ad lib.* (Tedstone *et al.* 1991). A low T_3 state has been observed in food-restricted rats and is accompanied by increased activity of WAT lipoprotein lipase (*EC* 3.1.1.34) (Hansson *et al.* 1983). To test whether the effects of meal-feeding may be due to the change in thyroid state, we injected *ad lib.*- and meal-fed male Wistar rats with T_3 (500 μg/kg body-weight) for three consecutive days. At the end of this period the rats were starved for 22 h and refed 5 g of a chow meal containing 0·75 g [1-^{14}C]triolein. Measurements of expired $^{14}CO_2$ (over 5 h), the accumulation of [^{14}C]lipid in tissues and the amount of [1-^{14}C]triolein absorbed were as described by Oller do Nascimento & Williamson (1986).

				Tissue [^{14}C]lipid accumulation (% absorbed dose/g)					
		$^{14}CO_2$ production (% absorbed dose/5 h)		Epididymal adipose tissue		Inguinal subcutaneous adipose tissue		Interscapular brown adipose tissue	
Groups	*n*	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Ad lib.-fed	6	46·8	3·8	0·44	0·04	0·34	0·05	9·9	2·3
Ad lib.-fed+T_3	5	42·7	5·3	0·28*	0·04	0·65*	0·11	13·8	1·9
Meal-fed	5	25·6	0·9	3·30	0·42	2·72	0·54	9·9	2·3
Meal-fed+T_3	6	41·9†	3·9	1·05†	0·25	2·06	0·36	8·8	1·2

Significantly different from *ad lib.*-fed group: *$P<0·05$.
Significantly different from meal-fed group: †$P<0·001$.

T_3 treatment had no effect on the $^{14}CO_2$ production of *ad lib.*-fed rats; however, it increased $^{14}CO_2$ production of meal-fed rats to the values seen in the *ad lib.*-fed rats. The accumulation of [^{14}C]lipid in epididymal adipose tissue was significantly decreased by T_3 treatment in both meal-fed and *ad lib.*-fed rats, but the decrease was quantitatively much greater in the former group. In contrast, T_3 did not significantly decrease the deposition of [^{14}C]lipid in subcutaneous adipose tissue of meal-fed rats but increased deposition in this fat depot of *ad lib.*-fed rats.

These findings suggest that changes in thyroid status may be involved in the adaptation of adipose tissue fat deposition to meal-feeding observed in previous experiments, but that the effects may be depot-specific.

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Effects of hyperinsulinaemia or vanadate on lipid partitioning between mammary gland and adipose tissue during lactation and weaning in the rat. By TERESA H. M. DA COSTA and DERMOT H. WILLIAMSON, *Metabolic Research Laboratory, Nuffield Department of Clinical Medicine, Radcliffe Infirmary, Woodstock Road, Oxford OX2 6HE*

Low plasma insulin may play a role in the suppression of white adipose tissue (WAT) deposition of exogenous lipid in lactation (Oller do Nascimento *et al.* 1989). We therefore examined the effect of hyperinsulinaemia (24 h) or chronic vanadate (an insulin mimetic) treatment.

Lactating rats were treated as follows: (1) no treatment (controls); (2) 0·3 g sodium orthovanadate/l drinking water from the 2nd or 3rd day postpartum (vanadate). Before study (24 h) groups of rats were either made prolactin-deficient (bromocriptine injected) or had pups removed (litter-removed group). Hyperinsulinaemia was produced by subcutaneous injection of insulin (total 2·5 IU as three doses over 24 h). The deposition of oral [1-^{14}C]lipid was measured in the mammary gland (MG), WAT and pups (milk clot and carcass). Lipoprotein lipase (*EC* 3.1.1.34; LPL) activity was measured in WAT and MG.

Only in prolactin-deficient rats, where transfer of lipid is low, did vanadate and insulin increase accumulation of [1-^{14}C]lipid in MG, but they had no effect on transfer. The increased lipid deposition in MG was accompanied by higher activity of tissue LPL (nmol free fatty acid released/min per mg dried tissue) in the prolactin-deficient plus vanadate (4·36 (SE 0·49); n 6; $P<0·005$) and prolactin-deficient plus insulin groups (6·28 (SE 1·15); n 5; $P<0·01$), compared with the prolactin-deficient group (1·48 (SE 0·48); n 4).

		Tissue [^{14}C]lipid accumulation (% of absorbed dose/5 h)					
		Total mammary gland		Total clot + pups		WAT/ (amount per g)	
Groups	n	Mean	SE	Mean	SE	Mean	SE
Lactating	10	38·3	4·4	21·7	3·2	0·04	0·009
+ Vanadate	6	38·2	4·6	23·7	6·0	0·04	0·008
+ Insulin	4	48·0	4·4	18·3	4·4	0·10**	0·02
Prolactin-deficient	4	31·9	1·7	5·6*	5·3	0·35	0·16
+ Vanadate	6	55·4††	5·8	7·0***	1·6	0·06	0·01
+ Insulin	5	60·3*†††	3·3	8·6*	2·4	0·15	0·05
Litter removed	6	5·6***	0·5	—		2·47***	0·33
+ Vanadate	7	4·5***	0·4	—		2·27***	0·49
+ Insulin	6	5·6***	0·9	—		2·03**	0·37

Significantly different from lactating no-treatment group: *$P<0·05$; **$P<0·01$; ***$P<0·001$.
Significantly different from prolactin-deficient group: †$P<0·05$; ††$P<0·01$; †††$P<0·001$.

Hyperinsulinaemia increased [1-^{14}C]lipid in WAT (2·5-fold) but this was modest compared to the fiftyfold change when the litter was removed. In litter-removed rats there was no additional effect of insulin or vanadate.

It is concluded that hyperinsulinaemia (or vanadate) only increases MG uptake of dietary fat in the prolactin-deficient state and that there is resistance to its effects on WAT fat deposition during lactation.

Oller do Nascimento, C. M., Ilic, V. & Williamson, D. H. (1989). *Biochemical Journal* **258**, 273–278.

Plasma beta-cell tropin in obese and normal children. By J. L. MORTON, *Clore Laboratory, University of Buckingham, Buckingham MK18 1EG* and A. SALVATONI, *Clinica Pediatrica, Universita degli Studi di Pavia, Italy*

Beta-cell tropin (BCT; equivalent to $ACTH_{22-39}$) is a potent insulin secretogogue and lipogenic agent, first isolated from the pituitary of the genetically obese (ob/ob) mouse. Plasma concentrations of BCT have been found to be elevated in genetically obese rodent models (Morton *et al.* 1991) and human type II diabetics (Morton *et al.* 1990).

Plasma samples from a group of thirty-four obese children (weight excess over 120% ideal body-weight by Tanner's standards, weight range 21–112 kg) were compared to twenty-nine normal weight children matched for age and sex. The obese children showed a significantly raised plasma concentration of BCT (nmol/l) over the normals (1·01 (SD 0·38) and 0·79 (SD 0·44) respectively; $P<0·025$), and also a significantly raised plasma concentration of insulin (μU/ml) (12·1 (SD 8·1) and 4·9 (SD 3·1) respectively; $P<0·0005$). There was a statistically significant correlation ($P<0·005$) between plasma BCT concentration and weight excess.

A further group of eleven obese children were studied before and after a minimum weight loss of 10%. The plasma BCT concentrations were significantly lower after weight loss. Weight excess (%) was reduced from 150 (SD 4) to 134 (SD 4) ($P<0·0005$), plasma BCT (nmol/l) was reduced from 1·44 (SD 0·27) to 0·84 (SD 0·09) ($P<0·025$). No significant change in plasma insulin concentrations were observed. There was a significant correlation between the reduction in weight excess and the reduction in plasma BCT concentrations ($P<0·025$).

We conclude from these results that plasma BCT concentrations are higher in obese children than in normal weight children, and that plasma BCT concentrations are significantly correlated to weight excess. Weight loss is associated with significant reduction of BCT concentration in plasma, and the reduction in BCT is proportional to the weight lost.

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The relationship of eating self-efficacy to weight change in obese hospital out-patients. By K. W. CARR, *Department of Nutrition and Dietetics, Royal Infirmary, Glasgow G4 0SF* and A. W. GARDNER, *Faculty of Health Studies, The Queen's College, Glasgow G13 1PP*

Poor long-term compliance in obese subjects treated by dietary advice has been well documented (Brownell, 1984). Consequently, social and psychological factors which may affect outcomes have been investigated with the ultimate aim of improving results.

Bandura's self-efficacy theory (1977) postulates that an individual's judgement of his ability to perform a specific task (perceived self-efficacy) is an important link between possessing the skills and knowledge to perform the task and actually doing it. Glynn & Ruderman (1986) have produced a valid and reliable twenty-five item eating self-efficacy scale (ESES). Their study using the ESES in a sample of clients undergoing different weight reduction programmes showed that eating self-efficacy was predictive of weight change.

The aims of our study were to improve the homogeneity of the sample and to investigate the possible relationship between ESES and other factors. Patients whose medical condition or therapy would have affected the outcome were excluded. Following a pilot study, ESES were scored and information obtained about eating habits and influence of family and friends on compliance by using structured interviews with patients on similar weight reduction programmes and attending a hospital clinic. Weight changes were monitored over three visits with 4 weeks between visits. This was done retrospectively for patients already attending (n 55) and prospectively for new attendees (n 15).

Retrospective sample results showed that there was a significant negative correlation between ESES scores and weight change, a high ESES score indicating low self-efficacy ($r = -0{\cdot}432$, $P<0{\cdot}001$ (0·19, 0·73)). Those with 'regular' eating habits had significantly lower ESES scores than those who described themselves as 'compulsive eaters' ($P<0{\cdot}05$). Subjects who felt that friends had a positive influence on their efforts to lose weight lost significantly more than subjects who did not experience positive influence ($P<0{\cdot}05$).

In the prospective sample, significant changes in ESES scores at first and second visits ($P<0{\cdot}05$; Wilcoxon Matched Pair Test (one-way)) were predictive of weight changes.

Low self-efficacy, lack of positive influence from family and friends, and compulsive eating habits may be important contributory factors to poor outcome in this client group.

The guidance of Professor A. R. Lorimer, Glasgow Royal Infirmary, is gratefully acknowledged.

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The effect of cessation of smoking on eating patterns, energy intake and body-weight. By MARY CURSITER, *Department of Dietetics and Nutrition* and M. HOLMES, *Department of Management and Social Sciences, Queen Margaret College, Edinburgh EH12 8TS* and SHEILA JENNETT, *Institute of Physiology, University of Glasgow, Glasgow G12 8QQ*

Cessation of smoking affects both sides of the energy balance equation leading to weight gain; reductions in energy expenditure have been identified, but changes in energy intake (EI) are less well documented. This report examines eating patterns, EI and body-weight in women smokers before and after cessation of smoking; energy expenditure results from this study have been reported (Cursiter *et al.* 1992).

Data are presented for forty-nine subjects, thirty-one who successfully stopped smoking (mean (SD) age 34·5 (5·8) years and body mass index (BMI) 22·3 (2·2)) and eighteen who relapsed (mean age 35·2 (6·4) years and BMI 21·9 (2·3)). All subjects were assessed as smokers (baseline) and 1 month after an agreed cessation date (return). Smoking status was monitored at each visit by measuring end-expired carbon monoxide. Eating patterns were examined using a food frequency questionnaire; scores for each of 124 foods, weighted by frequency intervals (from 'twice a day or more'–6 to 'once a month'–1), were combined to give frequency of consumption (FC) scores for different food groups. EI was assessed using a modified diet history method. Data were analysed by *t* tests; complete data were not obtained for all subjects.

	Successful group					Relapsed group				
		Baseline		Return			Baseline		Return	
	n	Mean	SD	Mean	SD	*n*	Mean	SD	Mean	SD
Weight (kg)	31	61·5	5·8	63·6**	6·2	18	62·1	7·4	62·6	7·4
EI (MJ/d)	28	8·21	1·83	8·84	2·59	16	8·31	2·27	8·45	2·72
FC scores:										
Total food	26	161·6	29·7	169·7	29·4	16	140·5	30·5	144·4	33·6
Snacks:										
sweet	26	17·9	7·3	20·9*	9·3	16	11·6	8·9	11·6	9·4
savoury	26	9·3	4·8	10·7	4·7	16	6·3	4·7	7·1	5·4
Confectionery	26	3·4	2·7	5·1*	2·8	16	1·2	1·4	1·4	1·6

Significance tested by paired *t* test between baseline and return; *$P<0·05$, **$P<0·001$.

All successful ex-smokers gained weight (mean gain 2·08 kg, range 0·30–4·95 kg) and their total FC score increased ($P=0·05$), with significantly higher scores for confectionery and for sweet (but not savoury) snacks; both weight gain and changes in FC scores were significantly greater in former heavy smokers (twenty cigarettes/d or more) than in former light smokers (less than twenty cigarettes/d); none of these changes were found in the relapsed group. FC scores for foods eaten at mealtimes (e.g. meat, vegetables) remained unchanged in both groups. EI increased slightly, though not significantly, in successful ex-smokers, however, this change in EI may have been underestimated (snack eating is likely to have been under-reported in the diet history interviews).

These results suggest that stopping smoking leads to increased consumption of sweet foods, especially as snacks; although this may be a short-term effect, the resulting increase in energy intake is likely to contribute to weight gain after cessation of smoking.

This work was supported by the Scottish Health Education Group.

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Lipolysis, body composition and hormone levels during normal human pregnancy. By DORIS M. CAMPBELL, *Department of Obstetrics and Gynaecology* and M. A. RADCLIFFE, *School of Biomedical Sciences, University of Aberdeen*

Sequential phases of net fat storage and mobilization in pregnant women have been revealed by serial study of body composition (Pipe *et al.* 1979). Separate, cross-sectional studies have indicated that lipolysis is increased in white adipose tissue (WAT) in late pregnancy (Elliott, 1975; Coltart & Williams, 1976). We have now assessed WAT lipolysis, body composition and hormonal status serially in an attempt to identify their interrelationship within different stages of pregnancy.

Twenty-two normal women were recruited in early pregnancy for serial study at 12, 20, 30 and 38 weeks of gestation (G) and at 6 weeks postpartum (PP). WAT was sampled from the buttock by percutaneous needle biopsy. Lipolysis was measured by incubating pieces of WAT (10–15 mg) at 37° in bicarbonate saline with isoprenaline at 0 M (basal), 1 μM (submaximal) or 10 μM (maximal). Indices of body composition and plasma hormone levels were measured: body-weight, total body water, skinfold thickness at seven sites, fat-cell size, oestriol, oestradiol-17β, progesterone, human placental lactogen (HPL) and insulin.

Changes in lipolysis (nmol glycerol/mg tissue in 90 min)

Interval (weeks)	*n*	Basal		Submaximal		Maximal	
		Mean	SD	Mean	SD	Mean	SD
12G–20G	21	0·121**	0·189	–0·495*	0·847	0·049	1·053
20G–30G	22	–0·017	0·284	0·405**	0·581	0·227	0·864
30G–38G	17	–0·017	0·181	0·027	1·019	0·254	0·881
38G–6PP	17	0·060	0·161	0·169	0·754	–0·173	0·682

*$P<0·02$; **$P<0·01$.

At week 12G the rates of basal, submaximal and maximal lipolysis (mean (SD)) were respectively 0·21 (0·10), 0·80 (0·79) and 1.58 (0·77). The Table shows that significant intervisit changes in lipolysis were confined to the 12G–30G period (paired *t* tests). The concurrent rise in basal and fall in submaximal lipolysis during the 12G–20G interval were followed by restoration of submaximal lipolysis in the 20G–30G interval. Significantly negative correlations ($P<0·05$; Pearson's *r*) between lipolysis and body composition at visits were confined to skinfold thicknesses and showed a staged involvement of the maximal (12G) and submaximal (20G and 30G) catecholamine-stimulated increments. Correlations based on intervisit changes were confined to buttock fat-cell size (12G–20G, basal lipolysis, 0·37; 20G–30G, submaximal lipolysis, 0·38). Correlations between lipolysis and hormone levels occurred at each visit; but those based upon intervisit changes over the 12G–30G period were limited to progesterone (basal lipolysis, 0·39; submaximal lipolysis, 0·44) and HPL (submaximal lipolysis, 0·61).

There are differential changes in buttock lipolysis during early and mid-pregnancy, but presently no clear associations exist with body composition and hormonal status.

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The effect of breed (Large White × Landrace *v.* purebred Meishan) on the diets selected by pigs given a choice between two foods that differ in their crude protein contents.

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Two pig breeds, an improved one (Large White × Landrace (LW×)) and an unimproved one (purebred Chinese Meishan (CM)) were used to test the idea that the genotype of the animal has an influence on its selection of a diet from two foods that differ in their crude protein (CP) contents. Entire male pigs (six per breed) were given a free choice between two foods with similar energy concentrations (16 MJ digestible energy/kg), but different CP contents (130 (L) and 252 (H) g CP/kg fresh food respectively), from 21 to 34 kg live weight (LWT). In addition, twelve pigs from each breed were given *ad lib.* access to either food L, or food H, or their mixture M (0·25 L + 0·75 H) for the same LWT range (four per food), in order to quantify the effects of the foods on the performance of the pigs, when they are offered singly. The daily rate of LWT gain and food conversion efficiency (FCE) of all pigs is given below. On any feeding treatment the LW× performed significantly better ($P<0·001$) than the CM pigs. The interaction between feeding treatment and breed on the rate of LWT gain and FCE was highly significant ($P<0·01$), mainly due to the poorer performance of LW× pigs on the low protein food.

	LWT gain (g/d)		FCE (g gain/g intake)	
Breed . . .	LW×	CM	LW×	CM
Treatment				
L	648	552	0·442	0·367
M	834	532	0·641	0·391
H	831	509	0·659	0·422
Choice L and H	842	636	0·635	0·414
SED	50·8		0·0336	

Pigs given a choice between the two foods selected a diet whose composition was different between the two breeds (proportion H selected was 521 *v.* 116 (SED 49) g food H/kg total food intake ($P<0·001$); CP selected was 194 *v.* 144 (SED 5·4) g CP/kg food ($P<0·001$), for the LW× and CM pigs respectively). The growth rates of these animals were not significantly different from the highest growth rates achieved on a single food (M or L for the LW× and CM respectively). The results support the idea that pigs given a choice between a suitable pair of foods are able to select a diet that allows them to meet their requirements for growth and express the characteristics of their genotype.

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Effects of dietary protein content during lactation on tissue protein synthesis in rats. By A. P. PINE, N. S. JESSOP and G. F. ALLAN, *Institute of Ecology and Resource Management, University of Edinburgh* and J. D. OLDHAM, *Scottish Agricultural College, West Mains Road, Edinburgh EH9 3JG*

Lactating rats can catabolize tissue protein when dietary protein is limiting (Pine *et al.* 1992). The present study was designed to explore the balance between protein synthesis and degradation in different tissues under these circumstances.

Following mating, forty-eight Sprague-Dawley multiparous rats were offered a high-protein diet (H; 215 g CP/kg dry matter (DM)) *ad lib.* until day 12 of gestation. Subsequently, half were offered either diet H or a low-protein diet (L_1; 65 g CP/kg DM) *ad lib.* until parturition, after which they were allocated factorially to either diet H or another low-protein diet (L_2; 90 g CP/kg DM) until day 13 of lactation. Litters were standardized to twelve pups on day 1 of lactation. Diets were isoenergetic with a constant carbohydrate:fat ratio (2·3:1) in DM. On days 1 and 13 of lactation [^{3}H]phenylalanine (Garlick *et al.* 1980) was used to measure fractional (FSR) and absolute (ASR) rates of protein synthesis in skeletal muscle, mammary gland, liver and duodenal mucosa.

Day of lactation . . .	1			13				
Dietary sequence, day 12 of gestation–day 13 of lactation	H	L_1		HH	L_1H	HL_2	L_1L_2	
	n 5	*n* 4	SED	*n* 5	*n* 5	*n* 4	*n* 5	SED
Gastrocnemius muscle								
FSR (%/d)	4·40	3·40	0·51	4·87^a	4·84^a	3·34^b	3·94^b	0·59
ASR (mg/d/g tissue)	8·86	7·06	1·02	10·07^a	10·20^a	6·78^b	7·54^b	1·37
Carcass protein (g)*	44·60	39·40		44·10	42·60	35·20	35·30	
Mammary gland								
FSR (%/d)	58·92	60·76	8·06	91·59^a	82·37^{ab}	58·89^{bc}	59·28^c	13·36
ASR (mg/d/g tissue)	52·90	47·35	10·54	118·20^a	113·47^a	74·15^b	64·66^b	15·56

^{a,b,c} Means in the same row within a time with different superscripts were significantly different; $P<0·05$.
* From Pine *et al.* (1992).

L_1 reduced muscle FSR and ASR on day 1 of lactation ($P = 0·08$ and $P = 0·1$), but had no effect on rates in the mammary gland. Between days 1 and 13 of lactation muscle FSR and ASR significantly increased ($P<0·05$) for group L_1H; mammary FSR and ASR increased during lactation for both HH and L_1H groups ($P<0·05$) but not for those offered L_2. Muscle FSR and ASR of HL_2 were considerably reduced during lactation by the diet L_2 ($P = 0·05$ and $P = 0·08$ respectively). By day 13 of lactation groups HL_2 and L_1L_2 had significantly lower rates of synthesis in both skeletal muscle and mammary gland compared to the HH and L_1H groups ($P<0·05$). There were no dietary or stage of lactation effects on FSR or ASR for liver and duodenal mucosa (FSR 94·84 (SE 3·78), 143·78 (SE 7·23); ASR 167·64 (SE 7·07), 153·42 (SE 7·32) respectively).

Calculated rates of degradation of carcass protein (%/d) were 4·73, 3·49, 5·82 and 4·31 respectively for HH, L_1H, HL_2, L_1L_2. The enhanced rates of protein loss associated with L_2 in lactation was the result of a combined reduction in rate of synthesis and increased rate of degradation of tissue protein, with the change in degradation being rather greater.

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