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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

A Scientific Meeting was held at the Royal College of Physicians, London, on Wednesday, 7 February 1996, when the following papers were presented.

All abstracts are prepared as camera-ready material by the authors.

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Non-diet and diet programmes for overweight: effects on mood, lifestyle and health. By JANE VIGUS[1] and CATHERINE GEISSLER[1], [1]*Department of Nutrition and Dietetics, Kings College London, Campden Hill Road, London W8 7AH*

In recent years the long-term efficacy of traditional behavioural and dietary treatment of overweight (BMI 25-30kg/m^2) and obesity (BMI > 30kg/m^2) has been increasingly questioned (Garner & Wooley, 1991) and there is some evidence that, in the absence of weight loss, improved physical and psychological health may be achieved by non-dieting interventions designed to prevent weight oscillations, improve nutrition and fitness and increase self esteem (Roughan *et al.* 1990; Polivy & Herman, 1992).

In the present study subjects who chose to attend a 10-week non-dieting programme run by Dietbreakers (NON-DIET) were assessed on a range of psychological, lifestyle and nutritional variables using pen and paper questionnaires and compared with subjects following a conventional 10-week weight loss programme (DIET). Baseline values with significant differences ($P<0.05$) are shown in the Table.

		Age (years)	Age at 1st diet (years)	WHR	EDI*	BDI*	SSE*	BMI at 1st diet (kg/m^2)
NON-DIET	Mean	32.7	16.6	0.77	37.2	16.3	49.9	22.9
(*n* 20)	SD	7.3	5.7	0.06	21.0	9.3	12.8	2.2
DIET	Mean	48.5	34.5	0.85	13.8	10.7	63.1	28.9
(*n* 23)	SD	12.1	17.1	0.10	8.2	7.2	17.8	4.6

WHR, waist:hip ratio; EDI, eating disorders inventory (Garner *et al.* 1983); BDI, Beck depression inventory (Beck *et al.* 1961); SSE, state self esteem (Heatherton & Polivy, 1991).
*EDI, normative value for subscales used 11.9; BDI, >12 indicates clinical depression; SSE, normal range 69-77.

Initial analysis of food frequency data also suggests that the NON-DIET group (*n* 17) ate significantly less fat (33.6 *v.* 39.7%) and more carbohydrate (47.6 *v.* 39.7%) than the DIET group (*n* 9) at baseline ($P<0.05$) indicating eating behaviour already close to current nutritional guidelines. There were no differences between NON-DIET and DIET in BMI (29.2 *v.* 31.5kg/m^2), exercise or dietary restraint. The NON-DIET started dieting much earlier and at a lower BMI than DIET. Despite their younger age they had been dieting for significantly more years, showing over that time a large significant mean increase in BMI ($P<0.05$). In the DIET group there was a similar trend, approaching significance, for BMI to have increased by several points since first dieting.

After 10 weeks the NON-DIET group (*n* 14) showed no changes in BMI or WHR but did show significant ($P<0.05$) improvement in EDI (10.2 *v.* 33.8) and BDI (6.5 *v.* 16.0) scores, increased SSE (69.3 *v.* 50.4) and reduced dietary restraint (2.4 *v.* 3.3) compared to baseline. The DIET group (*n* 14) showed a significant ($P<0.05$) reduction in BMI (31.9 *v.* 33.5), a trend towards an increase in WHR (0.87 *v.* 0.85). and a significant decrease in BDI (8.6 *v.* 12.4) compared to baseline.

The initial high eating disorder and depression scores and low self esteem of the NON-DIET group may have been a result of the failure of many years dieting to produce any permanent weight loss and this may also account for their willingness to try an alternative to conventional weight loss treatment. After 10 weeks, improvements were seen in all psychological factors while BMI remained stable, suggesting the NON-DIET programme was beneficial in the short term. Conventional weight loss treatment appeared to reduce levels of depression in the DIET group but had no effect on self esteem.There was no evidence suggesting any increase in disordered eating amongst the DIET group. Subjects are currently being followed up to assess the long-term effects of both programmes.

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Polivy, J. & Herman, C.P. (1992). *International Journal of Eating Disorders*, **11**,3,261-268.
Roughan, P., Seddon, E. & Vernon-Roberts, J. (1990). *International Journal of Obesity*, **14**, 135-147.

Effect of reduced-fat and reduced-sugar food use on diet and weight status of free-living consumers. By J.I. AARON, S.J. GATENBY, V. JACK and D.J. MELA, *Consumer Sciences Department, Institute of Food Research, Reading Laboratory, Reading RG6 6BZ*

Although there have been many laboratory-based investigations of appetite and food intake in response to fat and sugar substitution in foods, few studies have assessed the overall dietary behaviour of consumers using these products in natural situations over extended time periods. In a previous prospective trial (Gatenby *et al.* 1995), we found that use of commercially available reduced-fat foods by free-living consumers led to significantly reduced fat intakes during a 6-week study period. The drop in energy derived from fat was largely replaced by an increase in energy derived from carbohydrate, hence there was little net effect on total energy intake. The present trial expands and extends that work, while also contrasting the effects of fat *v.* sugar replacement.

Sixty-four non-obese females aged 18-55 years, all low users of reduced-fat and reduced-sugar products, were recruited to the study and randomly allocated to reduced-fat (RF), reduced-sugar (RS) and control groups (C) for a 10-week trial. RF and RS groups were instructed to replace traditionally formulated products with the modified versions throughout their diet, but were not restricted from consuming any specific foods or food groups. The C group continued with their habitual diet. All foods were purchased by subjects in the usual retail shops and consumed at home. Weighed diet records were collected over 4 d periods at weeks 0 (baseline), 2, 4, 7, and 10. These records and itemized store receipts were reviewed, and body weights measured at the end of each period.

Cut-off limits for 'physical activity level' (Energy intake/Estimated BMR) derived by Goldberg *et al.* (1991), were used to identify and remove suspected under-reporters. Hence, final analysis was carried out on data from seventeen RF, nineteen RS and thirteen C subjects only.

One-way ANOVA revealed no significant differences between groups at baseline for subject characteristics (age, weight, BMI and dietary restraint (scale from van Strien *et al.* 1986)) or dietary characteristics (total energy, % energy from fat, carbohydrate, sugars and protein). Repeated measures ANOVA was used to identify main and interactive effects of treatment and time on energy and macronutrient intake, and weight status of subjects. There was no effect of treatment on energy intake, however there was a significant effect of time ($P < 0.001$); all groups similarly decreased their reported energy intake during the period of study. There was a significant treatment x time interaction for % energy from fat ($P = 0.017$); compared with the RS and C groups, the RF group reduced their fat intake during the intervention period. All groups reduced their reported sugar intake (time; $P < 0.001$). The RS group did reduce % energy from sugar to a greater extent than the RF and C groups; however, this difference was not statistically significant. There were significant treatment ($P = 0.04$) and time effects ($P = 0.05$) on % energy from protein; all groups increased their reported protein intake. Finally there were no main or interactive effects of treatment or time on % energy from total carbohydrate or on body weights.

The results indicate that reduced-fat food use led to a significant decrease in the % energy from fat; however, reduced-sugar food use had no consistently significant dietary effects. However, it is also apparent that subjects in all groups were increasingly under-reporting over time, given the similar decreases in reported total energy intakes with no change in body weights. It appears likely that the casual use of macronutrient-substituted foods by normal-weight consumers in a free-living environment may influence composition of the diet, but with perhaps little net effect on energy intake or body-weight status.

This research was supported by the UK Biotechnology and Biological Sciences Research Council.

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Goldberg, G.R., Black, A.E., Jebb, S.A., Cole, T.J., Murgatroyd, P.R., Coward W.A. & Prentice, A.M. (1991). *European Journal of Clinical Nutrition* **45**, 569-581.

van Strien, T., Frijters, J.E.R., Bergers, G.P.A. & Defares, P.B. (1986). *International Journal of Eating Disorders* **5**, 295-315.

The effect of meal frequency and energy restriction on total energy expenditure and spontaneous activity in obese subjects in a chamber calorimeter. *By M.A. TAYLOR and J.S. GARROW, Department of Human Nutrition, St. Bartholomew's Hospital Medical College, Charterhouse Square, London EC1M 6BQ*

The number of meals eaten in a day may be inversely associated with obesity. This might be because a higher meal frequency is associated with a higher total energy expenditure. This could be a consequence of greater spontaneous activity when more meals are eaten per day compared with fewer meals. We aimed to study the relation between meal frequency and energy expenditure in obese subjects.

Ten female subjects (BMI: median 40.6 kg/m^2, interquartile range 35.3-41.5 kg/m^2; age: median 39 years, interquartile range 27-45 years) were admitted to an indirect chamber calorimeter. They were given 4184 kJ/24 h as either two isoenergetic meals (2/24 h) at 11.00 and 19.00 hours, for a 2 d period; or as six isoenergetic meals (6/24 h), at intervals of 2 h between 09.00 and 19.00 hours, for a second 2 d period (randomized-crossover design). The door of the chamber calorimeter was closed for the final 40 hours of each 2 d period. Total energy expenditure was measured and spontaneous activity pattern recorded (by video) during the final 24 h of each 2 d period. Activity was categorized as lying down (considered asleep), sitting, standing and cycling.

Measured energy expenditure during meal frequency periods 6/24h and 2/24h.

	'24 h'(*n* 10) (MJ/24h)		'Night' (23.00-08.00 hours) (*n* 8) (MJ/24h)		'Sleeping' (02.00-0500 hours) (*n* 8) (MJ/24h)	
	6/24h	2/24h	6/24h	2/24h	6/24h	2/24h
Median	9.95	9.92	8.30	9.08	7.56	8.33
Interquartile range	8.38-11.21	8.63-11.25	6.66-8.62	7.73-9.820	6.29-8.11	7.32-9.04
2-tailed *P**	0.88		0.02		0.07	

Spontaneous activity duration and frequency

	Lay down-time (h/24h)	Lay down (no.of times/24h)	Sit-time (h/24h)	Sit (no. of times/24h)	Stand-time (h/24h)	Stand† (no.of times/24h)	Cycle-time (h/24h)	cycle (no. of times/24h)
median - 6 meals/d	12.21	9.0	9.61	38.0	1.37	40.0	0.10	1.0
median - 2 meals/d	12.43	6.0	10.32	34.0	2.55	36.0	0.14	1.0
2-tailed *P**	0.31	0.21	0.11	0.17	0.26	0.34	0.47	0.69

*Wilcoxon Matched-pairs Signed-ranks Test,2-tailed

†Corrected for lower frequency of standing as retrieving food from hatch less often.

There was no difference in total energy expenditure between the two phases. Greater energy expenditure seen during the 'night', during 2/24 h, could reflect a more extensive and extended thermogenic effect caused by the larger meal at 19.00 hours (2092 kJ) compared with the 6/24h period (699 kJ). There was no difference between the total duration of each activity type or frequency of each activity type when comparing the two meal patterns.

This study shows that there was no difference in total energy expenditure or spontaneous activity pattern of obese subjects in a chamber calorimeter, given a hypoenergetic diet as two or six meals/d.

Dietary restraint and nutrient intakes of adolescents. By C.B. MULVIHILL[1], P.J. ROGERS[2] and G.J. DAVIES[1] *[1]Nutrition Research Centre, South Bank University, 103 Borough Road, London SE1 0AA and [2]Consumer Sciences Department, Institute of Food Research, Earley Gate, Whiteknights Road, Reading RG6 6BZ*

For many women, chronic dieting to maintain weight at a low level has become a way of life. Research has shown that cultural pressures to be slim have filtered down to the adolescent age group. The apparent trend towards restricting food intake during adolescence is of concern as it may be associated with physical and psychological changes (Hill, 1993).

In the present study male (*n* 25) and female (*n* 36) adolescents completed a 7 d weighed record of food intake. BMI was calculated for each subject and converted into percentiles using BMI charts (Cole *et al.* 1995). Dietary restraint was assessed using the Dutch Eating Behaviour Questionnaire (van Strein *et al.* 1986). Fourteen subjects, seven male and seven female, were excluded from the nutrient intake analysis because of inadequate diet records.

	Male		Female	
Nutrient intake (*n* 47)	Mean	SE	Mean	SE
Energy (MJ)	9.88*	0.67	7.07	0.30
Calcium (mg)	845***	83	605	34
Iron (mg)	13.3**	1.4	8.8	0.5
Zinc (mg)	8.2	0.5	5.8	0.4
Folate (μg)	288	44	192	14
BMI (n 61)	21.6	0.6	21.7	0.6
BMI percentile (*n* 61)	68.5	5.3	60.6	4.3
Restraint score (*n* 60)	1.7†	0.2	2.6	0.2

Significantly different *$P<0.05$, ** $P<0.01$, ***$P<0.005$, (unpaired *t*-test) †$P<0.001$ (Mann-Whitney U test)

The Table shows that energy intakes in males and females were lower than the estimated average requirement (Department of Health, 1991). Females had intakes of Ca, Fe, Zn and folate below the recommended nutrient intake (RNI). Female Fe intakes were extremely low, out of twenty-nine female diets analysed only one subject's diet contained an intake above the RNI. Ca intakes in females were low, with a mean intake of 74% of the RNI. On the other hand, mean BMI was above the 50th percentile. Dietary restraint scores was significantly higher in females, and BMI and restraint scores were positively correlated (r 0.31, $P < 0.06$). Apparent under-reporters were not excluded from this study as a low energy intake may be associated with dietary restraint.

The results indicate that there was a sex difference in nutrient intake, especially for Fe and Ca. Recent studies have shown that a growing number of adolescent females are anaemic and this can have detrimental effects on health, academic and physical performance (Nelson *et al.* 1993). A low Ca intake has been associated with later osteoporosis. While the low nutrient intakes may be partly explained by under-reporting, the particularly low intakes of females are consistent with their high dietary restraint and their somewhat lower BMI. Further work is needed to examine possible relationships between low micronutrient intake and altered food choice among high dietary restrainers.

Cole, T.J., Freeman, J.V. & Preece, M.A. (1995). *Archives of Disease in Childhood* **73**, 25-29.
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Interviewees with higher body mass index were less likely to return diet diaries in a longitudinal national survey. By G.M. PRICE[1], A.A. PAUL[1], T.J. COLE[1] and M.E.J. WADSWORTH[2], [1]*MRC Dunn Nutrition Unit, Milton Rd, Cambridge CB4 1XJ and* [2]*MRC National Survey of Health and Development, Department of Epidemiology and Public Health, University College London Medical School, London WC1E 6BT*

It is usually not known whether the non-responders in dietary surveys have similar characteristics to those of the responders and hence how representative the sample is of the intended population. The MRC National Survey of Health and Development (NSHD) provides a singular opportunity to investigate whether diet-diary response is random among interviewees in a large survey.

The NSHD is a prospective survey of a UK birth cohort from 1946, a collection of information on many aspects of the members' lives. In 1982 and 1989, 3322 and 3262 survey members were interviewed, respectively, and anthropometric measurements made. In each year a 2 d dietary recall was taken and then the subjects were asked to keep a diet diary in household measures for the subsequent 5 d, with postal return.

The diary response rate among all interviewees was 73.4% in 1982 and 70.3% in 1989. The Table shows the highly significant distribution in 1989 of percentage of interviewees in each category of BMI according to their diary response (Pearson χ^2 for linear trend, $P<0.0001$, *df* 1). Responders were divided here into two categories using the cut-off point of 1.10 for recorded energy intake as a fraction of BMR (EI/BMR), values of less than 1.10 being implausible for the habitual range of intakes of a weight-stable, free-living but sedentary adult (Goldberg *et al.* 1991). Thirty-eight subjects with missing heights or weights were excluded.

	Percentage of each category of BMI in 1989:					
BMI (kg/m^2)...	< 19.0 (*n* 70)	19.0 - 21.9 (*n* 576)	22.0 - 24.9 (*n* 1128)	25.0 - 29.9 (*n* 1080)	≥ 30.0 (*n* 370)	All categories (*n* 3224)
No diary	27.1	20.5	26.2	32.8	43.2	29.4
Diary yielding EI/BMR <1.10	1.4	9.5	13.1	20.6	30.8	16.7
Diary yielding EI/BMR ≥ 1.10	71.4	70.0	60.6	46.7	25.9	53.9

There were disproportionately more obese subjects among those who did not return diet diaries. In 1989 only 25.9% of obese interviewees returned a diet diary with a plausible energy intake. The pattern illustrated by the Table held for both men and women and in 1982 as well as in 1989.

Diary non-responders were also more likely to be current smokers (Pearson χ^2; $P<0.001$), to have achieved a lower level of education ($P<0.001$) and to have a lower social class of origin ($P<0.01$), each of these phenomena being more significant in the women than the men and valid for the 1982 as well as 1989 waves of the survey. Logistic regression showed that higher BMI was the most significant characteristic, independent of the others.

Subjects of both sexes who had not returned their diet diary in 1982 were more likely to be non-responders in 1989, and *vice versa* ($P<0.0001$). Thus there is a consistent group of interviewees who are less likely to submit a diet diary than others and who have characteristics very similar to those of responders with implausibly low energy intake (Price *et al.* 1993). High BMI is a significant risk factor not only for implausibly low recorded energy intake but also for the non-return of diet diaries.

Goldberg, G.R., Black, A.E., Jebb, S.A., Cole, T.J., Murgatroyd, P.R., Coward, W.A. & Prentice, A.M. (1991). *European Journal of Clinical Nutrition* **45**, 569-581.

Price, G.M., Paul, A.A., Cole, T.J., Hilder, W.S. & Wadsworth, M.E.J. (1993). *Proceedings of the Nutrition Society* **52**, 343A.

The influence of glycine on urea-nitrogen salvage. By T.S. MEAKINS and A.A. JACKSON, *Department of Human Nutrition, University of Southampton, Southampton SO16 7PX*

It has been demonstrated that the primary influence on urea-N salvage is the intake of total N (Meakins & Jackson 1994). Non-essential N, as urea, was shown to enhance urea hydrolysis in the colon and improve N balance. Addition of glycine and urea to diets low in protein has been shown to be more efficient at improving N balance than urea alone (Jackson 1995). The aim of the present study was to determine whether glycine is a limiting factor for urea salvage by comparing the effect of supplementation of two sources of non-essential N upon the urea salvage system.

Six adult females were given three diets for a period of 5 d each, containing either (a) 26 g protein/d (4.16 g N/d), (b) 26 g protein/d with 6.9 g urea (7.36 g N/d), (c) 26 g protein/d with 8.6 g glycine and 3.4 g urea (7.36 g N/d). Urea kinetics were measured using the prime/intermittent oral dose method over the final 24 h of each dietary period (Jackson *et al.* 1984).

Intake	26g protein		26g protein + 6.9g urea		26g protein + 3.4g urea + 8.6g glycine	
	mean	SE	mean	SE	mean	SE
Nitrogen Balance mgN/kg/d	-37[a]	3	-19[b]	4	-17[b]	5
Urea Production mgN/kg/d	88[a]	8	91[a]	9	101[a]	10
Urea Hydrolysis mgN/kg/d	20	4	32	12	21	6
Urea Excretion mgN/kg/d	68[a]	6	113[b]	13	107[b]	9
5-oxoproline excretion µmol/d	385[a]	38	213[b]	38	186[b]	28

Mean values within a column with unlike superscripts were significantly different, $P < 0.05$ (Paired rank test).

N balance was negative on the diet providing 26 g protein. Addition of the urea supplement improved N balance by increasing urea hydrolysis in the colon. The insufficiency of the 26 g protein/d diet for glycine, as demonstrated by the high excretion of 5-oxoproline was improved by the urea supplement, suggesting that the hydrolysed urea-N was incorporated into the body pool of glycine (Jackson 1987). When glycine and urea were supplemented together, both N balance and glycine deficiency were improved to similar extents as that seen with the urea supplement. However, this was not due to an increase in colonic urea hydrolysis.

Non-essential N is limiting in the low-protein diet as addition of either source of this nutrient improves N balance. Glycine is limiting on this intake as shown by the excretion of 5-oxoproline before and after supplementation. However, it appears that non-essential amino acids may be replaced directly by the glycine supplement as there was no influence on urea-N salvage. Improvement in nitrogen balance by enhanced urea hydrolysis after an exogenous urea supplement is of similar efficiency in meeting the body's requirement for glycine.

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Jackson, A.A. (1995). *Proceedings of the Nutrition Society* **54**, 535-547.
Jackson, A.A, Badaloo, A.V, Forrester, T, Hibbert, J,M. & Persaud, C. (1987). *Br J Nutr* **58**, 201-214.
Meakins, T.S. & Jackson, A.A. (1994). *Proceedings of the Nutrition Society* **53**, 196A.

Modulation of blood pressure by glucocorticoids in the rat. By DAVID S. GARDNER, SIMON C. LANGLEY-EVANS and ALAN A. JACKSON, *Department of Human Nutrition, Bassett Crescent East, University of Southampton, SO16 7PX*

A mild protein deficit throughout gestation in the rat renders the resulting offspring hypertensive relative to control animals (Langley & Jackson, 1994). Alterations to fetal glucocorticoid (GC) homeostasis (Phillips *et al.* 1994) may 'programme' the development of the hypertensive state through irreversible effects on the hypothalamic-pituitary-adrenal (HPA) axis (Langley-Evans *et al.* 1996*b*). Specific activities of glutamine synthetase (*EC* 6.3.1.2.) and glycerol phosphate dehydrogenase (*EC* 1.1.1.8.), two GC-inducible brain enzymes, were significantly elevated above control values when measured at term and weaning, indicating a permanent alteration to HPA axis function. The aim of the present study was to determine whether the functions of the postnatal glucocorticoid environment are a prerequisite for elevated blood pressure in offspring from maternally protein-restricted dams.

Fourteen female Wistar rats were randomly allocated to receive either a diet containing 180 g casein/kg (*n* 7) or 90 g casein/kg (*n* 7) for 2 weeks before mating and throughout gestation. The diet was replaced with standard laboratory chow upon giving birth. At weaning (3-4 weeks), offspring from each dietary group, 180 g casein/kg (*n* 24) and 90 g casein/kg (*n* 20) were either bilaterally adrenalectomized or sham operated under sodium pentobarbitone anaesthesia following determination of systolic blood pressure. Either corticosterone (20 mg/kg in 0.1 ml arachis oil) or vehicle (arachis oil) were then injected twice daily for 14 d into all animals from each dietary group. Systolic blood pressure was measured at 7 d and 14 d post-surgery.

	Blood pressure (mmHg)											
Maternal Diet...	180 g casein/kg						90 g casein/kg					
	Initial		7d		14d		Initial		7d		14d	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Adx + Cort	144	7.3	174**	2.8	168*	5.9	165	5.9	202*	8.0	175	14.5
Adx + Veh	142	4.7	149	2.1	137	9.8	162	11.9	143	8.9	131*	5.9
Sham + Cort	147	7.6	166*	6.1	143	7.4	175	8.9	158	8.7	171	5.0
Sham + Veh	147	3.8	149	5.2	137	6.7	161	7.1	168	9.7	146	12.3

Adx, adrenalectomy; Cort, corticosterone, Sham, sham operated, Veh, vehicle.
* Significantly different from initial value, $P < 0.05$, ** $P < 0.01$.

Adrenalectomy had no effect on blood pressure in the 180 g casein/kg-exposed control group but abolished the hypertension observed in the low-protein-exposed animals. Initially corticosterone replacement to adrenalectomized animals significantly raised blood pressure in both dietary groups.

The results clearly show that the high blood pressure exhibited by animals prenatally exposed to a low-protein diet is dependent upon intact and functional adrenal glands and that corticosterone replacement restores the effect. The active glucocorticoid in the rat, corticosterone, may possibly mediate maternal diet-induced hypertension through enhanced vascular reactivity (Langley-Evans *et al.* 1996*a*).

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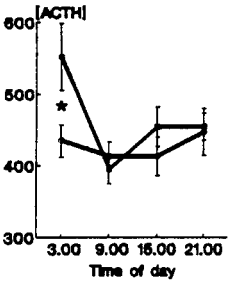
Programming of the hypothalamic-pituitary-adrenal (HPA) axis by the maternal diet in the rat.

By SIMON C. LANGLEY-EVANS, DAVID S. GARDNER and ALAN A. JACKSON, *Department of Human Nutrition, University of Southampton, Bassett Crescent East, Southampton SO16 7PX*

Protein restriction of the pregnant rat has extensive effects upon the resulting offspring. Hypertension, altered appetite (McCarthy *et al.* 1994), immune dysfunction and antioxidant inadequacy all appear to be programmed by a mild manipulation of the maternal diet. A role for glucocorticoids in the initiation of these disturbances has been inferred (Langley-Evans *et al.* 1995). Long-term alterations to glucocorticoid-regulated metabolism indicate a permanent adjustment to the function of the HPA axis. The offspring of pregnant rats fed on low-protein diets were assessed in terms of HPA axis hormone concentrations in plasma and tissue glucocorticoid receptor numbers.

Nine female Wistar rats were fed on a diet containing 90 g casein/kg for 2 weeks before conception and throughout pregnancy. Nine further rats were fed on a 180 g casein/kg diet (control diet) over the same period. On giving birth all rats were transferred to the same laboratory chow diet, which was also used to wean the offspring at 4 weeks of age. A total of twenty-four weanling female offspring from each group were killed, six at each of four points in the light cycle, for assessment of plasma adrenocorticotrophic hormone (ACTH; μg/l) and corticosterone (CORT) concentrations. A further twenty-three male rats from each group were adrenalectomized at 7 weeks old to clear type II glucocorticoid receptors (GR; fmol/mg protein) of endogenous steroids. Scatchard analyses of [^{3}H] corticosterone (0-25 nM) binding to GR in liver, kidney, hippocampus and hypothalamus were performed. Binding of 20 nM [^{3}H]corticosterone to GR in thoracic aorta was assessed in a further group of male pups.

Fig. 1



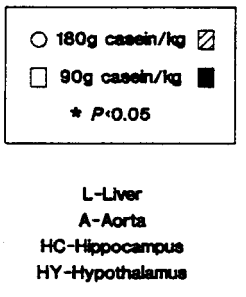
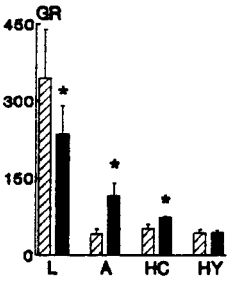


Fig. 2



Prenatally undernourished rats had blunted diurnal variation in ACTH (Fig. 1) secretion and had lower circulating levels of the hormone during the dark phase of the cycle. Plasma corticosterone concentrations were similar in both groups of rats at all time points studied. GR numbers were elevated in brain regions of low-protein diet-exposed rats, relative to control animals (Fig. 2). Conversely, in the liver GR numbers were markedly lower in the 90 g casein/kg diet-exposed group. These animals exhibited 3-fold higher binding of [^{3}H]corticosterone in thoracic aorta when compared with control rats. The data are consistent with long-term programming effects of maternal protein restriction upon HPA axis function. Whilst the effects of maternal diet on GR in vascular tissue may provide a direct link between steroid action and hypertension, the raised GR number in specific brain regions may indicate a mechanism for altered appetite and obesity.

Langley-Evans, S.C., Phillips, G.J. & Jackson, A.A. (1996). *Proceedings of the Nutrition Society* **54**, 140A.

McCarthy, H.D., Pickard, C.L., Speed, J. & Jackson, A.A. (1994). *Proceedings of the Nutrition Society* **53**, 172A.

Selenium content of UK available infant formulas and estimation of the intake of bottle-fed infants. By L.H. FOSTER[1], G.J. DAVIES[1] and S. SUMAR[2]. [1]*Nutrition Research Centre,* [2]*Food Research Centre South Bank University, London SE1 OAA*

Infants given formula milks as their sole source of nutrition may be at risk of Se deficiency. This is particularly true of preterm infants and those suffering from primary lactase deficiency. Preterm infants have low hepatic stores and plasma concentrations of Se making them susceptible to haemolytic anaemia and cancer in later life (Reifen & Zlotkin, 1993). Lactose-intolerant infants are at risk due to their nutritional dependency on soya formula for extended time periods.

Eleven commercial brands of infant formula (term, preterm and soya) were purchased at retail outlets across London. In total 174 samples were analysed. Samples were digested overnight with nitric/perchloric acid (16 M-HNO_3-11.6 M-$HClO_4$). The total Se content was determined using hydride generation atomic absorption spectrometry (Foster & Sumar,1995*a,b*). The dietary Se intake (μg/d) of British formula-fed infants aged 0-3 months was estimated on the basis of formula Se content (μg/g) and total formula intake g/d in accordance with the manufacturers' instructions, as shown in the Table.

Infant feed	Se content*(μg/g) Mean	SEM	Se intake (μg/d) *0-2 w*	*3-6 w*	*2 m*	*3m*	Se intake 0-3 months ‡ (μg/d) Mean	SEM
Term								
A	0.050	0.005	4.1	4.6	5.7	6.8	5.3	0.61
B	0.061	0.007	4.7	5.2	6.5	7.8	6.1	0.70
Preterm								
C	0.033	0.007	5.7	6.4	8.0	9.5	7.4	0.86
D	0.045	0.005	3.2	5.2	6.3	7.3	4.9	0.87
E	0.083	0.002	7.0	7.8	9.8	11.7	9.1	1.05
Liquid								
F	0.027†	0.004	9.6	14.5	24.1	28.9	19.3	4.4
G	0.049†	0.002	15.7	23.6	3.3	47.1	31.4	7.17
Soya								
H	0.089	0.008	6.9	7.6	9.6	11.5	8.9	1.03
I	0.046	0.004	3.6	3.9	4.9	5.9	4.6	0.53
J	0.023	0.004	3.0	3.3	4.1	4.9	4.9	0.44
K	0.033	0.004	2.1	2.4	3.0	3.0	2.6	0.22

A,B,C,D,E,F,G,H,I,J,K, different manufacturers, *w*, weeks, *m*, months.
* *n* 18 unless otherwise indicated; † *n* 6; ‡ *n* 4

The dietary reference value for Se for infants aged 0-3 months is 10 μg/d and is based on a breast milk concentration of 15 μg/d (Department of Health, 1991). From the Table only the liquid low-birth-weight formulas seemed to provide this amount. All other formulas analysed provided a lower estimated intake which is inadequate. Only 18% of the formulas analysed for Se met the DRV value. This is an approximation; actual formula consumption (ml/d) may be less than 700 ml, particularly for preterm infants. The above findings indicate the Se content of powdered infant formulas commercially available in the UK do not provide the recommended Se intake for formula-fed infants during the first 3 months of life.

Department of Health Report on Health (1991). *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom*. London: HMSO.
Foster, L. H. & Sumar, S. (1995*a*). *Food Chemistry*. **55**, 293-298.
Foster, L. H. & Sumar, S. (1995*b*). *Food Chemistry*. (In the Press.)
Reifen, R. M. & Zlotkin, S. (1993). *Nutritional Needs of the Preterm Infant*. Baltimore: Williams and Wilkins Press.

The physiological basis of dietary preference in West Midlands behaviourally resistant house mice. By R.C. TAYLOR, A.G. STEPHENS and R.M. SIBLY, *School of Animal and Microbial Sciences, University of Reading, Whiteknights, Reading RG6 6AJ*

In 1986, a localized population of house mice (*Mus domesticus*) were discovered to be highly resistant to normal pest control techniques (baiting and trapping). Known as West Midlands behaviourally-resistant (WMBR), they are sensitive to modern rodenticides but strongly avoid eating carbohydrate-rich foods such as cereals (Humphries, 1994), and therefore cannot be controlled with conventional cereal-based bait. The hypothesis that this behaviour is physiologically based was tested by comparing wild caught WMBR mice with non-resistant house mice from the Reading area.

The mice were taken from captive populations established from wild caught animals in 1992. Three groups were set up: Group 1: normal mice (seven male, four female) fed *ad libitum* on Porton combined diet (PCD) produced by Special Diets Services (approximately 440g/kg carbohydrate). Group 2: normal mice (seven male, four female) fed *ad libitum* on a homogeneous experimental diet palatable to both types of mice, with approximately 220g/kg carbohydrate. Group 3: WMBR mice (seven male, four female) fed as group 2. The diet fed to groups 2 and 3 represented a higher carbohydrate intake than that normally selected by WMBR mice.

The mice were weighed weekly and maintained for 5 weeks to allow adaptation to the diets. After this period they were killed, weighed, condition scored and dissected. Measurements for comparison included caecum mass and duodenal α-amylase (*EC* 3.2.1.1) activity. Tissue samples for enzyme assay were individually prepared by freezing, chopping, suspension in an equal mass of saline (9g NaCl/L), and sonication at 4 ^{0}; after centrifugation the supernatant fraction was appropriately diluted in saline (9g NaCl/L). Amylase assay was performed using the 'AMYLASE' (Procedure No.577) diagnostic kit, obtained from Sigma Diagnostics, Fancy Road, Poole, Dorset BH17 7NH.

Group		Total food intake (g/mouse per day)		Carbohydrate intake(g/mouse per day)		α-Amylase activity (U^{05}/g wet tissue)		Caecum weight (g/kg body wt)	
	n	Mean	SE	Mean	SE	Mean	SE	Mean	SE
1	10	4.49	0.10	2.00	0.04	17.43	1.45	13.2	0.9
2	9	5.01	0.07	1.11	0.02	15.49	2.87	7.5	0.4
3	9	4.77	0.09	1.06	0.02	1.78	0.36	38.0	5.3

There was no significant difference in α-amylase activity (Student's *t* test) between normal mice on PCD and the experimental diet (groups 1 and 2). Group 2 had significantly smaller caeca than group 1 ($P<0.001$) in response to the change in diet. Compared with normal mice on the same diet (group 2), WMBR mice (group 3) had very low α-amylase activity in the duodenum ($P<0.002$) and their caeca were very enlarged ($P<0.001$).

Caecal enlargement indicates that undigested fermentable material is reaching the hindgut, and can cause compression of the liver and diaphragm, water loss and even death. This phenomenon has been observed in rats fed with indigestible carbohydrate (Sukan,1979). It is concluded that low α-amylase activity in WMBR mice allows ingested carbohydrate to bypass normal digestion and reach the caecum, causing caecal enlargement. The associated symptoms would cause the mice to select a low - carbohydrate diet, and hence avoid cereals and other carbohydrate-rich foods.

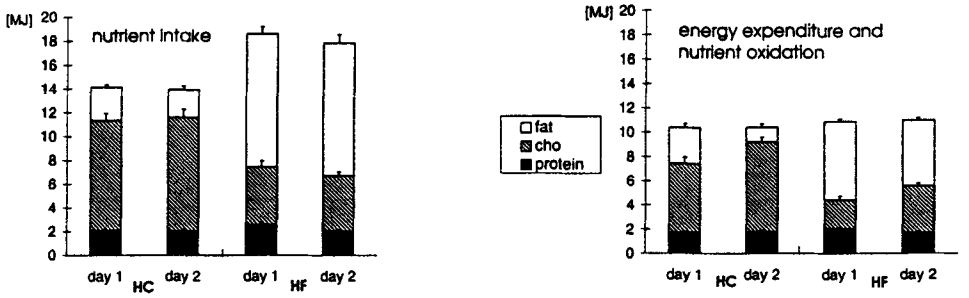
Humphries, R.E. (1994). Investigation into possible behavioural resistance in inner city house mice (*Mus domesticus Rutty*)in the UK. PhD Thesis, University of Reading.

Sukan, S.G. (1979). The caecal enlargement on feeding potato starch: physical, chemical and microbiological aspects. PhD Thesis, University of Reading.

Effect of *ad libitum* intake of high-carbohydrate or high-fat diet on nutrient balances. By C. PROSERPI[1], A. SPARTI[1], V. DI VETTA[1], H. MILON[2], Y. SCHUTZ[1] and E. JEQUIER[1], [1]*Institute of Physiology, Faculty of Medicine, University of Lausanne,* [2]*Nestlé Research Centre, Lausanne, Switzerland*

We investigated the extent of adjustment of nutrient oxidation to high-carbohydrate (HC) or high-fat (HF) diets during *ad libitum* food intake in ten healthy subjects over 2 d in a respiration chamber. In addition we investigated the influence of carbohydrate (CHO) balance on subsequent energy intake, according to the model described by Flatt (1987).

Ten young, weight-stable men with a mean BMI of 23 (range 20-28) kg/m^2 and without a family history of obesity or diabetes were studied over 48 h in a whole-body indirect calorimeter using a cross-over design. During each period, subjects could select meals from a list containing a large variety of food. The food quotient (FQ) of each food item proposed was higher than 0.85 for the HC diet and lower than 0.85 for the HF diet. At fixed times, food was served in large containers (main dish >1 kg) and subjects were told to eat as little or as much as they wanted. Results are expressed as means with their standard errors.



The subjects' food selection resulted in average FQ values of 0.92 (SE 0.003) and 0.80 (SE 0.003) for the HC and HF diets respectively. The intakes of total energy, CHO and fat were significantly affected by the diet composition (P<0.0001). However, even on the HF diet, when CHO availability was low, the CHO intake averaged 4.8 MJ (286 g). Total energy intake was markedly greater on the HF than on the HC diet (P=0.0002). Total energy expenditure by contrast, did not differ significantly between diets. The respiratory quotient was significantly different not only between diets, but also between day 1 and day 2 of both diets (P<0.0001). As a result, the contribution of CHO and fat to total oxidation was significantly influenced by dietary composition and day on the diet (P<0.0001). In the sedentary condition of the respiration chamber, subjects overate on both diets, as evidenced by the positive energy balance. This tendency was particularly pronounced under the HF diet condition. On the HC diet, CHO balance was positive, while fat balance was close to zero. On the HF diet, the fat balance was strongly positive during the two test days. There was no significant relationship between the first day CHO balance and the energy intake of the second day on either diet.

These results suggest that the deleterious effect of a HF diet on body weight regulation is mainly due to the excess of energy (and fat) intake and to a lesser extent to the insufficient response of whole-body fat oxidation.

Flatt, J.P. (1987). *American Journal of Clinical Nutrition* **45**, 296-305.

Postprandial exogenous lipid oxidation in obese and normal-weight women. By A.E. JONES, J.L. MURPHY and S.A. WOOTTON, *Institute of Human Nutrition, University of Southampton, Southampton SO16 6YD*

Our understanding of the determinants of postprandial oxidation of dietary lipid is limited. The suggestion that lipid oxidation is reduced in obese subjects (Jones *et al.* 1992) from indirect calorimetry, which estimates endogenous plus exogenous substrate oxidation rate, is not supported by earlier tracer studies using [1-^{14}C]palmitic acid (Issekutz *et al.* 1968). The present study utilized stable-isotope tracer methodology to examine the postprandial oxidation of [1-^{13}C]palmitic acid in obese and normal-weight women.

Following an overnight fast, ten overweight or obese women ingested [1-^{13}C]palmitic acid (10 mg/kg body weight) incorporated within a standardized test meal (Murphy *et al.* 1995). Breath samples were collected for measurement of ^{13}C enrichment before and at hourly intervals for 10 h following label administration and again at 15 and 24 h. Whole-body breath CO_2 excretion was measured by indirect calorimetry (Deltatrac, Datex Instrumentarium Corp., Helsinki, Finland) at the same time points until 6 h. ^{13}C enrichment was analysed by mass spectrometry (ABCA and ANCA systems, Europa Scientific Ltd., Crewe). The excretion of ^{13}C within stools collected over 5 d following the test meal was used to determine the proportion of absorbed label excreted on breath as $^{13}CO_2$. Breath $^{13}CO_2$ excretion was then corrected for the retention of label within the bicarbonate-CO_2 pools by measuring the recovery of $^{13}CO_2$ on breath following oral administration of ^{13}C-labelled $NaHCO_3$ in a separate trial (obese: 68.0%; normal: 64.8%) and was taken to reflect [1-^{13}C]palmitic acid oxidation over 24 h. The results were compared with those obtained in a group of six healthy, normal-weight women (Murphy *et al.* 1995) and are shown in the Table.

	BMI (kg/m^2)		% Body fat		Breath (% absorbed)		Corrected breath (% absorbed)	
	Median	Range	Median	Range	Median	Range	Median	Range
Obese	36.2*	27.0 - 44.7	45.2*	34.9 - 51.6	25.5	12.2 - 43.2	37.5	18.0 - 63.5
Normal	21.7	18.0 - 22.2	24.8	21.2 - 31.9	29.6	23.7 - 42.8	45.7	36.6 - 66.0

*Significantly different from normal-weight women (Mann-Whitney U); $P<0.05$.

The excretion of $^{13}CO_2$ on breath was lower in the obese women compared with the normal-weight women, although this difference did not reach statistical significance owing to large within-group variation. A trend towards an inverse relationship between excretion of $^{13}CO_2$ on breath and body composition, as described by BMI (r -0.46; P=0.07) and percentage body fat (r -0.47; P=0.06) was observed. These results would suggest that the oxidation of exogenous [1-^{13}C]palmitic acid following administration of the test meal may be reduced in obese women compared with normal-weight women.

Issekutz, B., Paul, P., Miller, H.I. & Bortz, W.M. (1968). *Metabolism* **17**, 62-73.

Jones, P.J.H., Ridgen, J.E., Phang, T. & Birmingham, C.L. (1992). *Metabolism* **41**, 396-401.

Murphy, J.L., Jones, A.E., Brookes, S. & Wootton, S.A. (1995). *Lipids* **30**, 291-298.

Modulating influence of alcohol and aspirin on postprandial lipaemia and factor VIIc in male subjects. By N. YAHIA and T.A.B. SANDERS, *Nutrition, Food and Health Research Centre, King's College London, Campden Hill Road, London W8 7AH*

Factor VIIc activity is elevated following postprandial lipaemia. In a previous study (Yahia & Sanders,1995) we found that a test meal containing 75 g olive oil + 15 g MaxEPA (OM) decreased postprandial lipaemia compared with a similar test meal supplying 90 g olive oil (OO). However, factor VIIc activity was similarly elevated after both test meals. This finding could possibly be due to the effects of *n-3* fatty acids on the blood flow mediated via formation of eicosanoids. We proposed that decreased vascular reactivity would increase the surface area exposed to blood and thus accelerate lipolysis which is necessary for activation of factor VIIc. In order to further investigate this hypothesis, we studied the effect of concurrent administration of aspirin (a cyclo-oxygenase inhibitor) and alcohol (a vasodilator). Six isoenergetic test meals were administered to twelve subjects in a randomized block design. Subjects received a low-fat diet on the day before the test meal. As in the previous study the test meals supplied either 90 g of olive oil (OO) or an admixture of 75 g olive oil + 15 g MaxEPA (OM). The test meal was administered with and without either 300 ml white wine or 250 mg aspirin. Fasting and non-fasting blood samples were obtained at 2, 3, 4, and 7 h after the test meal for triacylglycerol (TAG) and factor VIIc assays. One subject dropped out from the study leaving results for eleven subjects. The results are shown below.

	90 g Olive oil		75 g Olive oil + 15 g MaxEPA		90 g Olive oil + alcohol		75 g Olive oil 15 g MaxEPA + alcohol		90 g Olive oil + aspirin		75 g Olive oil 15 g MaxEPA + aspirin	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Factor VII (% standard)												
0 h	99	5.3	102	8.4	100	4.3	96	5.8	96	6.0	105	7.9
3 h	116†	9.6	118†	8.9	124†	10.2	112†	9.6	116†	9.9	121†	11.3
7 h	119†	10.1	113†	10.1	128†	11.7	117†	9.2	120†	9.5	122†	10.9
TAG AUC* GM (95% CI)	10.2^{a} (10.0-10.4)		6.16^{b} (5.95-6.37)		10.5^{a} (10.4-10.6)		11.5^{a} (11.4-11.6)		7.08^{b} (6.88-7.29)		6.61^{b} (6.39-6.83)	

*Geometric mean and 95% confidence interval of total area under the curve (AUC) described by serum triacylglycerol concentration plotted against time to 7 h. Values not sharing same superscript were significantly different, $P < 0.05$. † Significantly different from baseline, $P < 0.05$.

As expected the admixture of OM led to less postprandial lipaemia (as measured by area under the curve) compared with OO. This effect, however, was abrogated by the consumption of alcohol. The administration of aspirin decreased postprandial lipaemia following OO but not following the OM. This suggests that *n- 3* fatty acids decrease postprandial lipaemia by inhibition of prostaenoid synthesis. The increase in plasma TAG following alcohol consumption with OM was unexpected but may be due to suppression of fatty acid oxidation. Factor VIIc activity was increased similarly following all fat loads.

Yahia, N. & Sanders, T.A.B. (1996). *Proceedings of the Nutrition Society* (In the press).

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Effect of different patterns of fat intake on postprandial lipaemia and factor VII coagulant activity. By N. YAHIA., C. SONGHURST and T.A.B. SANDERS, *Nutrition, Food and Health Research Centre, King's College London, Campden Hill Road, London W8 7AH*

Plasma factor VII coagulant (FVIIc) activity in non-fasting blood samples is a strong independent predictor of fatal ischaemic heart disease (Meade *et al.* 1993). We have shown that a high-fat test meal leads to an increase in FVIIc (Yahia & Sanders, 1995).

In the present study we compared the effect of a low-fat intake with that of a high-fat intake (120 g) consumed in a single meal or in three meals on plasma triacylglycerol (TAG) concentration and FVIIc activity. The test meals were administered to six subjects (three males, three females) and they were isoenergetic (12.3 MJ). Diet A contained 30 g fat divided equally between three meals; diet B contained 120 g divided equally between three meals each supplying 40 g fat; diet C contained 120 g (90 g in the morning with 15 g fat in two subsequent meals). Subjects received the test diets for two consecutive days. Fasting blood samples were taken before the test meal (0 hour) and non-fasting blood samples were obtained at 2, 3, 4, and 7 h after the test meal for TAG determination on days 1 and 2. Fasting blood samples were obtained on day 3. FVIIc was determined in the fasting samples on days 1 and 3 and on the 7 h samples on days 1 and 2. The results are shown in the Table.

	DIET A		DIET B		DIET C	
	Mean	SE	Mean	SE	Mean	SE
Factor VIIc (% standard)						
0 h	106[a]	9.7	108[a,b]	11.1	128[b]	10.9
7 h	100[a]	11.3	111[a,b]	11.6	121[b]	13.4
TAG						
AUC*	0.96[a]	0.54	4.14[b]	0.92	5.77[b]	1.15

[a,b] Mean values within a row not sharing a superscript letter were significantly different ($P < 0.05$).

*Total area under the curve (AUC) described by serum triacylglycerol concentration plotted against time to 7 h.

Compared with the low-fat diet (diet A) the high-fat diets (diets B and C) increased the postprandial lipaemia, but there was no significant difference between diets B and C.

FVIIc was significantly increased by diet C compared with diet A in both the fasting and 7 h samples. FVIIc for diet B tended to be intermediate between diet A and C and did not differ significantly from either. These preliminary observations support the hypothesis that the pattern of fat intake may be an important determinant of FVIIc activity.

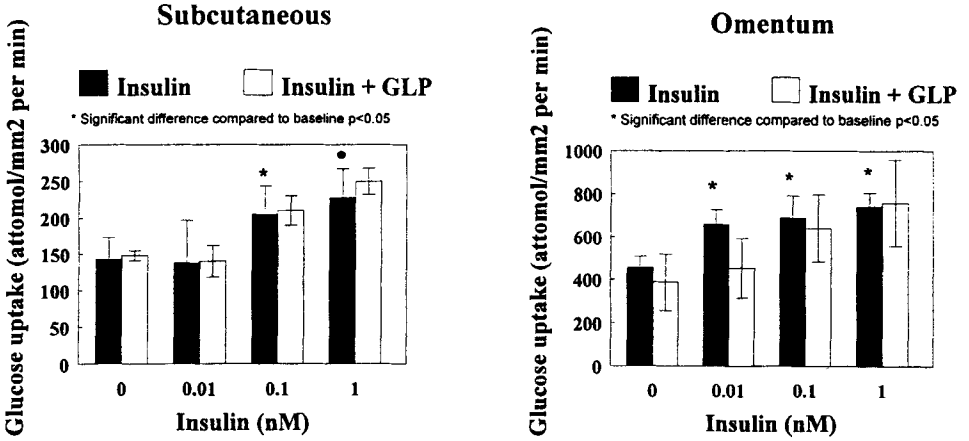
Meade, T.W., Ruddock, V., Striling, Y., Chakrabarti, R. & Miller, G.J. (1993). *Lancet* **342**, 1076-1079.

Yahia, N. & Sanders, T.A.B. (1996). *Proceedings of the Nutrition Society* (In the press).

A comparison of insulin-stimulated glucose uptake in isolated adipocytes from different fat deposits. Does glucagon-like peptide 1 enhance uptake? By GARY FROST[1], DAVID SMITH[2], GEOFFREY TREW[3], RAUL MARGARA[3] and ANTHONY LEEDS[4], [1]*Department of Nutrition and Dietetics, Hammersmith Hospitals NHS Trust, W12 OHS and* [2]*Departments of Metabolic Medicine and* [3]*Obstetrics and Gynaecology, Royal Postgraduate Medical School, London W12 OHS,* [4]*Department of Nutrition and Dietetics, Kings College, University of London, W8 7AH.*

Insulin action in adipose tissue, particularly visceral adipose tissue, is thought to have a central role in the insulin resistance syndrome (Frayn & Coppack, 1992). Recently a receptor for the incretin glucagon-like peptide 1 (7-36) amide (GLP-1) has been found on human adipocytes (Merida *et al.* 1993). This is of interest considering claims that GLP-1 improves insulin sensitivity (Hvidberg *et al.* 1994). We investigated insulin-stimulated glucose uptake in isolated adipocytes harvested from subcutaneous and omental adipose deposits in ten healthy women who were undergoing tubular surgery. At operation subcutaneous fat was removed by scalpel from the midpoint of the incision and the omentum sample was taken from the tip of this tissue. Adipocytes were dispersed in Dulbecco's modified Eagle's medium+bovine serum albumin (BSA 40 g/l) using collagenase (1 mg/0.5 g tissue) in a 37° vibrating water bath at 140 cycles/min for 75 min. Cells were then filtered, washed three times and then concentrated, the number of cells was calculated and diameter measured in fifty cells using the Cue-2 cell counting system. Isolated adipocytes (25 µl of concentrated cells, approximately 30,000 cells) were incubated in 500 µl KRB + 40 g/l BSA buffer in the presence of 1, 0.1, 0.01, or 0 nM-insulin, with or without 10 nM-GLP 1 (7-36) for 45 min then 3.7 KBq (300 nM) 2-deoxy-[U-^{14}C]-D-glucose was added and the adipocytes incubated for a further 15 min. The incubation was terminated by centrifuging the cells through 500 µl of silicone oil. The amount of radioactivity associated with the adipocytes was determined by liquid scintillation counting.

The figures demonstrate insulin-stimulated glucose uptake in both tissues. Adipocytes from omentum took up significantly greater amounts of glucose at baseline and all insulin concentrations. The addition of GLP-1 did not enhance basal or insulin-stimulated glucose uptake in either adipose tissue source.



Omental adipocytes appear to be more insulin-sensitive than subcutaneous adipocytes in women. Further work is needed to investigate the suppression of free fatty acid production in the face of insulin concentration in this tissue. Despite recent reports of a GLP-1 receptor on adipocytes, addition of GLP to buffer in superphysiological amounts did not enhance glucose uptake.

Frayn, K.N., Coppack, S.W. (1992). *Clinical Science* **82**, 1-8.
Hvidberg, A., Nielsen, M.T., Hilsted, J., Orskov, C. and Holst, J.J. (1994). *Metabolism* **43**, 104-108.
Merida, E., Delgado, E., Molina, M., Villanueva-Penacarrillo, M.L. and Valverde, I. (1993). *Journal of Clinical Endocrinology and Metabolism* **77**, 1654-1657.

Anthropometric characteristics of immigrants and non-immigrants in Canada. By JOCELINE POMERLEAU[1] and TRULS ØSTBYE[2], [1]*London School of Hygiene and Tropical Medicine, Department of Epidemiology and Population Sciences, Epidemiology Unit, London, WC1E 7HT,* [2]*Department of Epidemiology and Biostatistics, The University of Western Ontario, London, Canada*

The prevalence of overweight and obesity has been reported to vary among migrant groups to different countries (Chaturvedi *et al.* 1994; Choi *et al.* 1989; Østbye *et al.* 1989; Pawson *et al.* 1990). Since these health problems are becoming increasingly important in Canada, and since Canada is becoming an increasingly multicultural country, the association of place of birth with anthropometric traits among Canadians was investigated.

Anthropometric characteristics (BMI, overweight (BMI>25 kg/m^2), obesity (BMI>27 kg/m^2), and low BMI (BMI<20 kg/m^2)) of immigrants (individuals born outside of Canada) and non-immigrants (individuals born in Canada) between 20 and 65 years of age and living in Ontario, Canada, were compared using data from the 1990 Ontario Health Survey (OHS) (non-immigrants: *n* 25 732; immigrants: *n* 6 073). Descriptive statistics and multiple logistic and linear regression analyses were performed, adjusting for covariates (including demographic variables, ethnicity, and socioeconomic and health behaviour variables).

The unadjusted average BMI of all participants was 24.9 kg/m^2 (males: 25.6 kg/m^2; females: 24.2 kg/m^2), 25.0 kg/m^2 for non-immigrants (males: 25.7 kg/m^2; females: 24.3 kg/m^2) and 24.7 kg/m^2 for immigrants (males: 25.2 kg/m^2; females: 24.2 kg/m^2). Large unadjusted proportions of respondents were overweight (non-immigrants: 44.3% (males: 54.0%; females: 34.4%) immigrants: 41.3% (males: 47.9%; females: 34.6%)) or obese (non-immigrants: 27.0% (males: 31.9%; females: 22.0%); immigrants: 26.0% (males: 29.7%; females: 22.2%)), but only 9.1% of them (non-immigrants: 8.7% (males: 3.8%; females: 13.6%); immigrants: 10.3% (males: 6.2%; females: 14.5)) had a BMI lower than 20 kg/m^2. Regression analysis results indicated that overall, immigrants were less likely than non-immigrants to be overweight (odds ratio (OR) 0.75; 99%CI 0.63,0.90). Lower likelihoods of overweight and/or obesity were found among individuals born in Asia, East Asia, South-East Asia, China, Hong Kong, North Europe, and the UK compared with non-immigrants, but a higher likelihood of overweight was observed for Italian-born individuals (OR 1.85; 99%CI 1.17,2.93). Immigrants, overall (OR 1.51; 99%CI 1.13,2.02), and those from Asia, East Asia, South-East Asia, Hong Kong, China, and Vietnam were more likely to have a low BMI than non-immigrants. No differences were found between non-immigrants and immigrants from Europe, the Caribbean, Africa, Central and South America, West Europe, South Europe, East Europe, South Asia, the Middle East, East Africa, the USA, Germany, the Netherlands, Portugal, Yugoslavia, Greece, Poland, Hungary, the Philippines, India, Jamaica, Trinidad, and Guyana.

Considering the health consequences of obesity, it was reassuring to observe that some immigrant sub-groups, particularly Asian sub-groups, were in general less likely to be overweight or obese than were non-immigrants; however, low BMI was observed among some sub-groups. More research is still needed to understand these variations better. Culturally-sensitive health care and prevention programmes also need to be developed to help prevent obesity in Canada among population sub-groups at an increased risk of overweight.

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Waist:height ratio is a simple anthropometric index which is closely associated with blood pressure in middle-aged British adults. By S.R.E. LEJEUNE[1], M.A. ASHWELL[1], B.D. COX[2] and M.J. WHICHELOW[2], [1]*Ashwell Associates, Ashwell St, Ashwell, SG7 5PZ and* [2]*Department of Community Medicine, University of Cambridge, Cambridge CB2 2SR*

The anthropometric measurements of waist : hip circumference ratio (WHR), waist circumference (Lean *et al.* 1995) and waist circumference : height ratio (WHTR) (Ashwell *et al.* 1996) have variously been proposed as indicators of abdominal obesity and some have been associated with cardiovascular risk factors. The present study was designed to investigate which of these simple anthropometric indices was associated most closely with levels of blood pressure, one of the cardiovascular risk factors.

The subjects were 1308 men and 1599 women aged 40 to 64 years; all participants in the 1984-5 Health and Lifestyle Survey (Cox *et al.*1987). Height, weight, waist circumference and (in two thirds of the subjects) hip circumference were measured. BMI, WHTR, and WHR were calculated. The proportions of normotensive (systolic blood pressure <141 mmHg, diastolic <91 mmHg) or hypertensive subjects (being treated with medications for hypertension or systolic >160 mmHg, diastolic >95 mmHg) in the quintiles of each of the anthropometric indices were calculated.

	Percentages of normotensive men and women by quintiles of anthropometric indices					
	Height	Weight	BMI	Waist	WHR	WHTR
Men						
1st quintile	60.6	76.4	80.4	83.0	79.1	81.3
5th quintile	73.9	59.5	53.8	52.8	50.3	51.2
Difference between 1st and 5th quintiles	-13.3	16.9	26.6	30.2	28.8	30.1
n	1308	1308	1308	1303	816	1303
Women						
1st quintile	70.6	83.1	88.0	92.5	86.0	94.0
5th quintile	79.2	61.6	56.4	52.7	60.1	52.3
Difference between 1st and 5th quintiles	-8.6	21.5	31.6	39.8	25.9	41.7
n	1599	1599	1598	1591	1027	1589

The Table shows that there was a higher proportion of normotensive subjects in the first (lowest) quintile than in the fifth (highest) quintile for each anthropometric index, except height. Total or abdominal obesity and small stature, is therefore associated with reduced likelihood of having a normal blood pressure level. Calculation of the difference between the lowest and highest quintiles for each index (to indicate its power of differentiation) showed that WHTR and waist circumference were the best, and very similar, differentiators for both sexes, whilst WHR and BMI were less good. These differences were more clearly demonstrated in women.

Discriminant analysis confirmed the conclusions drawn from the quintile analysis. The combined rank order for discrimination in middle aged men and women was: WHTR (65.6% correct assessments), waist circumference (63.2%), BMI (62.7%) and WHR (60.0%).

Normal blood pressure values were more frequent in men and women aged between 18 and 40 years, but WHTR and waist circumference were again better discriminators than WHR in both sexes.

The results indicate that, of the simple anthropometric indices which are proxies for abdominal obesity, WHTR shows the closest association with blood pressure. This evidence supports the proposal that WHTR may be a good measure for indicating the need for weight management in a public health context (Ashwell *et al.* 1996).

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Identifying the possible causes of maternal obesity. By HELEN E. HARRIS[1], GEORGE T. ELLISON[1], MARY HOLLIDAY[2] and EMY LUCASSEN[1], [1]*Maternal and Child Health Research Programme, and* [2]*Faculty of Health, University of Greenwich, Wellington Street, London SE18 6PF*

A variety of studies have shown that maternal body weight increases with parity and that some women experience excessive weight gain from the beginning of one pregnancy to the next (Lederman, 1993). Nevertheless, it remains unclear whether these changes in body weight are the result of physiological phenomena that occur during pregnancy or changes in lifestyle that accompany pregnancy and motherhood. To assess the relative importance of these possible causes we used a repeat pregnancy study to examine the change in body weight displayed by primiparous women from the beginning of their first pregnancy to the beginning of their second. Detailed information on twenty sociodemographic, clinical and behavioural characteristics was abstracted from the medical records of women who gave birth to their second baby in the principal maternity ward of a South Thames hospital between 1990 and 1993. Of these women, 318 had reliable weight measurements recorded during the first trimester of both pregnancies and were included in the study.

There was considerable variation in the change in maternal body weight from one pregnancy to the next: eighty-eight (27.7%) of the women actually lost weight, while eighty-nine (28.0%) gained more than 4kg. Using the classifications of BMI proposed by the US Institute of Medicine (overweight: BMI 26.0-29.0 kg/m^2 and obese: BMI>29.0 kg/m^2), significantly more of the mothers were either overweight or obese at the beginning of their second pregnancy (26.3%) than at the beginning of their first pregnancy (18.2%; χ^2=5.886, P=0.052). Indeed, twenty-three women became overweight during this period and eighteen more (5.8%) actually became obese. These forty-one women were more likely to be unmarried, had a higher initial BMI, gained more weight during pregnancy and had a longer interval between pregnancies than mothers with relatively stable body weight (see Table below).

	Women who became overweight or obese		Women with stable body weight	
Maternal characteristic	Mean	SE	Mean	SE
Maternal age (years)	26.13	0.75	25.45	0.24
Initial BMI in first trimester (kg/m^2)	25.08*	0.25	23.08	0.25
Inter-pregnancy interval (years)	3.44**	0.37	2.66	0.09
Weight gain in pregnancy (kg)	11.12**	0.84	9.53	0.24
Percentage unmarried	39.0***		18.1	
Percentage smokers	19.5		16.2	
Prevalence of proteinuria (%)	2.4		3.7	
Prevalence of pregnancy-induced hypertension (%)	29.3		25.5	
Prevalence of glucosuria (%)	14.6		7.0	
Gestational age at delivery (weeks)	39.9	0.4	39.5	0.2
Percentage lactating when leaving hospital	43.6		57.4	

Significantly higher than the weight stable group: * P<0.05. ** P<0.01. ***P<0.001

After accounting for potential confounding factors using analysis of covariance there was no residual association between marital status and change in body weight. However, initial BMI, interpregnancy interval, pregnancy weight gain and proteinuria were all independently associated with increased weight gain. Despite these significant associations, the complete model explained just 18% of the variance in weight change, which suggested that additional factors may have played a more important role in the development of maternal obesity. Initial BMI and weight gain during pregnancy, both of which are largely determined by behavioural factors, together explained the largest proportion of variance in maternal weight change. For this reason we suspect that maternal obesity is primarily the result of changes in lifestyle that accompany pregnancy and motherhood.

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Differences in reported macronutrient composition of the diet in overweight and lean individuals are dependent on validity of reported energy intakes. By CAROLYN D. SUMMERBELL, *Rank Department of Human Nutrition, St Bartholomew's and the London Hospitals Medical College, Whitechapel, London E1 1BB*

The reported proportion of energy from fat in the diet (%Fat) is directly related to BMI in most cross-sectional studies of free-living populations (Lissner & Heitmann, 1995). However, in most studies which have found such a relationship, EI is either unrelated or inversely related to BMI. Question: should we believe that the overweight consume relatively high-fat diets when we clearly do not believe that they consume the same amount of energy (or even less) compared with their lean counterparts? Answer: only if we believe that overweight-related under-reporting is not macronutrient specific.

Energy intake was assessed by the 7d weighed dietary intake method in 220 people. Details of the data collection, coding and analysis of food intake have been described elsewhere (Summerbell *et al.* 1995). In brief, subjects ranged in age from 13 to 91 years, and 149 were female and 71 were male. Overweight (defined as a BMI > 25kg/m^2) under-reporters (defined as reporting a physical activity level < 1.10) reported a diet higher in %Protein and %Fat, and lower in %Sugar compared with overweight individuals who reported valid dietary records (Table 1). Mean BMI was similar in overweight under-reporters (30.0, SD 15.0kg/m^2) compared with overweight valid reporters (28.2, SD 14.2kg/m^2). These differences were not observed when lean individuals who under-reported were compared with those who reported valid dietary records, but lean valid reporters reported a diet higher in %Alcohol compared with lean under-reporters.

Sub-group		EI (MJ)	%Protein	%Fat	%CHO	%Alcohol	%Sugar	FFI
Overweight under-reporters (*n* 17)	Mean	5.43	15.4	41.8	41.6	1.1	17.0	3.4
	SD	1.12	2.6	6.2	7.9	2.2	6.3	0.9
Overweight valid reporters (*n* 46)	Mean	8.15	12.9	38.9	46.4	1.8	21.0	3.6
	SD	1.87	2.1	4.8	5.4	3.3	7.7	0.6
P=		*0.000*	*0.001*	*0.054*	*0.032*		*0.043*	
Lean under-reporters (*n* 16)	Mean	5.29	14.1	38.0	47.2	0.7	21.9	3.6
	SD	1.23	3.2	7.1	7.3	1.7	5.3	0.6
Lean valid reporters (*n* 141)	Mean	8.67	13.1	39.4	45.5	2.0	20.2	3.6
	SD	2.19	1.9	4.6	5.2	3.4	6.6	0.7
P=		*0.000*				*0.013*		

Feeding pattern was also assessed in this study. Daily energy intakes were divided into six feeding periods; breakfast, mid-morning snacks, lunch, mid-afternoon snacks, evening meal and evening snacks. For each individual, the mean energy intake at each feeding period, as a percentage of EI, was calculated (%MPEI). The feeding frequency index (FFI) is the number of %MPEI which individually exceed 10% of EI. A low FFI indicates a 'gorging' feeding pattern whereas a high FFI indicates a 'nibbling' feeding pattern. There was a trend for overweight under-reporters to report a diet containing fewer snacks compared with individuals who reported valid dietary records (Table 1). Snacks in this study were found to be higher in %Sugar and lower in %Protein and %Fat compared with meals (Summerbell *et al.* 1995). These findings support the hypothesis that under-reporting by the overweight is biased towards snacks which results in a reported 'gorging' feeding pattern and a reported dietary intake high in %Fat and %Protein, and low in %Sugar.

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