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PROCEEDINGS OF THE NUTRITION SOCIETY

ABSTRACTS OF COMMUNICATIONS

A Scientific Meeting was held at the Octagon Centre, Sheffield on Saturday, 12 September 1992, when the following papers were presented.

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Normal pregnancy: a state of insulin resistance? Studies with the euglycaemic clamp. By KATHARINE STANLEY, CHRISTINE BRUCE and ROBERT FRASER, *Department of Obstetrics and Gynaecology, Northern General Hospital, Sheffield S5 7AU*

The principal fetal substrate is glucose. Modification of the maternal response to insulin profoundly affects the substrate mix available to the fetus and thus fetal growth. Longitudinal studies (Lind *et al.* 1973) of oral glucose tolerance test (GTT) indicate that early pregnancy is a state of increased insulin sensitivity, but by late pregnancy insulin resistance has developed.

The euglycaemic clamp (EC) measures whole-body insulin sensitivity. We have used EC to study normal pregnant women throughout pregnancy and lactation. Criteria for a satisfactory EC have been described elsewhere (Stanley *et al.* 1991).

Ten pregnant women were studied, five were also studied pre-pregnancy, and nine postnatally. Six women had three EC in pregnancy, at 16, 26 and 36 weeks, and at least one when non-pregnant; four had two EC in pregnancy and at least one when non-pregnant. Subject ages ranged from 26 to 34 years, five were parous. Three had grade 1 obesity and one was a potential diabetic (normal GTT). Results were analysed by two-way ANOVA and significance taken at the 5% level. There was no significant difference in inter-subject or inter-trimester mean plasma insulin.

Stage of pregnancy	*n*	Mean plasma insulin (mU/l)	Mean glucose infusion rate (mg/ml per min)	% Postpartum glucose infusion rate	95% CI
Non-pregnant	5	63·2	283	100	
16 weeks	9	43·9	237	84	71–103
26 weeks	10	46·7	172	61	50–75
36 weeks	7	57·2	139	49	34–83
Postpartum	9	49·5	282	100	

Three women showed increased sensitivity at 16 weeks, while six showed a reduction. Consistent, statistically significant resistance had occurred by 26 weeks, with minimal progression between 26–36 weeks. The obese and the potential diabetic were significantly more resistant at 36 weeks, but not at 26 weeks. Lactation did not affect insulin sensitivity.

Interpretation of these data may be altered by knowledge of the degree of suppression of hepatic glucose output during EC in pregnancy: there are no human data addressing this question currently in the literature.

Lind, T., Billewicz, W. Z. & Brown, G. (1973). *Journal of Obstetrics and Gynaecology of the British Commonwealth* **80,** 1033.

Stanley, K., Bruce, C. & Fraser, R. (1991). *Proceedings of the Nutrition Society* **50,** 201A.

Reduced head growth from the second trimester of pregnancy to age 2 years: implication for health in adulthood. By W. M. O. MOORE, R. P. BANNISTER, B. S. WARD, V. F. HILLIER and F. N. BAMFORD, *St Mary's Hospital, University of Manchester M13 0JH*

The objective of the present study was to measure physical growth of fetuses and infants in an inner city health district in the north of England, and to compare their growth profiles according to mother's country of birth. A prospective longitudinal study was made of fetal and postnatal growth in a representative sample from the geographically-defined Central Manchester Health District. Data were collected from the beginning of the second trimester of pregnancy to the age of 2 years. The subjects were 174 singleton infants, born at term (≥37 weeks), who had serial antenatal cephalometry at planned 3-week intervals, and serial postnatal growth measurements at birth and at 6, 13, 26, 52 and 104 weeks. The main measurements were of fetal biparietal diameter, infant occipito–frontal circumference, crown–heel length, and weight, analysed by sex and mother's country of birth (British Isles or Indian subcontinent).

Infants of Indian-born mothers tended to be lighter at birth than those of locally-born mothers, but the difference was not due to lower accumulation of soft tissue. Body length from 6–52 weeks in both groups was similar. The major finding was reduced head size in boys of Indian-born mothers, which was evident from mid-pregnancy and persisted postnatally ($P<0{\cdot}01$).

Barker (1992) has demonstrated that retarded growth *in utero* and at 1 year of age is linked with hypertension, high concentrations of haemostatic factors, impaired glucose tolerance and non-insulin-dependent diabetes, and mortality from ischaemic heart disease. The relationships between early growth and cardiovascular risk factors, including haemostatic factors, blood pressure, plasma glucose, insulin, and apolipoprotein B, are not only strong and graded but specific; different risk factors are associated with different patterns of reduced fetal or infant growth. Because mortality from ischaemic heart disease is specifically linked with head size at birth (Barker *et al.* 1993), what is the implication of the continuum of reduced fetal and postnatal head growth experienced by the boys of the Indian-born mothers? Young men who migrate from the Indian subcontinent and reside in England are known to have a substantial excess mortality (standardized mortality ratio 313 at age 20–29 years) from ischaemic heart disease (Balajaran, 1991). Whatever the future holds for this group of boys, their specific marker for liability to heart disease in early adulthood would be missed if fetal growth was assessed solely on birth weight.

Balajaran, R. (1991). *British Medical Journal* **302,** 560–564.

Barker, D. J. P. (1992). *The Fetal and Infant Origins of Adult Disease.* London: British Medical Journal Publishing Group.

Barker, D. J. P., Osmond, C., Simmonds, S. J. & Nield, G. (1993). *British Medical Journal* **306,** 422–426.

Does a maternal milk-free diet prevent allergy in the 'at-risk' infant? By JULIE A. LOVEGROVE, S. M. HAMPTON and JANE B. MORGAN, *School of Biological Sciences, University of Surrey, Guildford GU2 5XH*

We have studied the effect of a maternal milk-free diet during late pregnancy and lactation on the allergy development and immune response of at-risk infants compared with normal infants and at-risk infants whose mothers were following an unrestricted diet. We previously reported the effect of a milk-free diet on fetal cord blood milk antibodies (Lovegrove *et al.* 1991). In the present study we report on the effect of maternal milk-free diet on infants' serum milk antibody level to 12 months postnatal age and allergy outcome. Fourteen atopic (group 1) and twelve non-atopic (group 2) pregnant women followed a normal diet, and twelve atopic (group 3) pregnant women followed a milk-free diet for 6 weeks before delivery. Subjects were provided with a hypoallergenic formula (Pepti-Junior, Cow and Gate, Trowbridge, Wiltshire) to consume as required. Heel-prick blood samples were taken from infants at 1 week and 3, 6 and 12 months post-partum. An indirect enzyme-linked immunosorbent assay was used to determine total IgG against β-lactoglobulin (βLG) and α-casein (αCas) in serum (Lovegrove *et al.* 1989). The Table provides the results for these analyses at 6 months in the three groups outlined above.

Group . . .	1 (*n* 14)		2 (*n* 12)		3 (*n* 12)	
	Mean	SD	Mean	SD	Mean	SD
Serum IgG against βLG (μg/ml)	302	256	136	107	86	134
Serum IgG against αCas (μg/ml)	54	43	39	69	14	23

Infants in the atopic group (1) showed a dramatic increase in βLG IgG and αCas IgG antibody levels at 6 months compared with groups 2 and 3. By 12 months the levels in group 1 fell back to values comparable with groups 2 and 3. The allergy incidence was found to be significantly greater in the infants born to normally-fed atopic mothers compared with infants whose atopic mothers followed a milk-free diet. There are practical implications in the treatment of infants with a family history of atopic disease.

We thank Cow and Gate, Trowbridge, Wiltshire for financial support.

Lovegrove, J. A., Hampton, S. M., Morgan, J. B. & Marks, V. (1989). *Biochemical Society Transactions* **17**, 1059–1060.

Lovegrove, J. A., Hampton, S. M., Morgan, J. B. & Marks, V. (1991). *Proceedings of the Nutrition Society* **50**, 9A.

Neuropeptide Y is increased in appetite-regulating hypothalamic areas of lactating rats.

By U. H. MALABU, A. KILPATRICK, M. WARE and G. WILLIAMS, *Department of Medicine, University of Liverpool, Liverpool L69 3BX* and R. G. VERNON, *Hannah Research Institute, Ayr KA6 5HL*

Neuropeptide Y (NPY), a thirty-six amino acid peptide, is a potent centrally-acting appetite-stimulating agent which is believed to regulate eating behaviour and body weight (Williams *et al.* 1991). Lactation in rodents is associated with increased nutrient requirements which are met by extraordinary increases in food intake (Vernon, 1989). The aim of the present study was to investigate whether hyperphagia in lactating rats is associated with increased hypothalamic NPY levels, as in the case of other hyperphagic states such as starvation and diabetes.

Twenty female Wistar rats, initially matched for age and weight, were studied. The experiment began when the lactating group (n 10) was on average at day 16 of lactation (range, 13–17). The lactating rats' food intake was over 330% that of the controls ($P<0{\cdot}001$). Body weight in the lactating group was 22% more than in non-lactating rats ($P<0{\cdot}001$); most of this weight gain is known to be due to hypertrophy of the mammary gland. Final plasma insulin was significantly lower in the lactating rats (6·6 (SD 0·6) $v.$ 11·7 (SD 2·1) pmol/l; $P<0{\cdot}05$). In lactating and non-lactating rats the plasma glucose (7·1 (SD 0·5) $v.$ 6·6 (SD 0·3) mmol/l) and corticosterone (187 (SD 61) $v.$ 232 (SD 42) pmol/l) levels were comparable (both $P>0{\cdot}05$). Four of the eight hypothalamic areas showed significantly increased NPY levels in the lactating rats compared with controls, namely: the arcuate nucleus-median eminence (+41%; $P<0{\cdot}001$); the paraventricular nucleus (+35%; $P<0{\cdot}001$); the ventromedial nucleus (+66%; $P=0{\cdot}003$); the dorsomedial nucleus (+78%; $P<0{\cdot}001$). Other hypothalamic regions showed no differences between the two groups.

Increased NPY levels in the arcuate nucleus (its principal hypothalamic site of synthesis) and in the paraventricular and dorsomedial nuclei (NPY-sensitive sites to which the arcuate nucleus projects) suggest increased activity of the NPY-ergic system in lactation. NPY may therefore mediate the markedly increased food intake in lactation.

Vernon, R. G. (1989). *Proceedings of the Nutrition Society* **48,** 23–32.

Williams, G., McKibbin, P. E. & McCarthy, H. D. (1991). *Proceedings of the Nutrition Society* **50,** 527–544.

Preventing steroid-associated gastric perforation with histamine$_2$ antagonists. By E. J. KELLY, P. C. NG, S. L. CHATFIELD, K. G. BROWNLEE, S. J. NEWELL and P. R. F. DEAR, *Neonatal Unit, St James' University Hospital, Leeds LS9 7TF*

A serious side effect associated with dexamethasone therapy for bronchopulmonary dysplasia (BPD) is gastric erosion and perforation; this is thought to have an incidence of 3% (Ng *et al.* 1991). The causative mechanism is unclear but may relate to a decrease in prostaglandin-mediated mucosal protection. Increasing gastric pH above 4·0 through the use of histamine$_2$ receptor antagonists should be protective; however, little is known about the use of such drugs in this patient population.

We have, therefore, continuously monitored gastric pH in ten infants with BPD receiving dexamethasone and ranitidine. These infants had a median gestation of 28 (range 24–31) weeks, birth weight of 947 (579–1300) g and age of 17 (15–21) d when they first received steroids. Ranitidine was given as a continuous intravenous infusion at doses of 0·031, 0·0625 and 0·125 mg/kg per h.

All doses of ranitidine significantly increased gastric pH (Table); 0·0625 and 0·125 mg/kg per h increased and maintained gastric pH above 4·0. An infusion of 0·0625 mg/kg per h ought therefore to be sufficient to prevent gastric acid-mediated damage to the gastric mucosa.

Ranitidine infusion rate (mg/kg per h) . . .	0	0·031	0·0625	0·125
Gastric pH: Median	1·8	2·7	4·6	4·8
Range	1·5–2·1	2·2–3·2	4·2–5·1	4·4–5·1

E.J.K. was funded by a Wellcome Institute Vacational Scholarship.

Ng, P. C., Brownlee, K. G. & Dear, P. R. F. (1991). *Archives of Disease in Childhood* **66**, 1164–1167.

Change in oesophageal pH: what does it tell us about gastro-oesophageal reflux in preterm infants? By A. J. LYON, G. WEST and K. GHAUS, *Neonatal Unit, Mayday University Hospital, Croydon, Surrey CR7 7YE*

Gastro-oesophageal reflux and pulmonary aspiration have been suggested as complications of feeding of preterm babies and are possibly linked to apnoea and chronic lung disease.

In adults reflux of acid is important and is defined as an oesphageal pH<4·0. Studies in neonates have used the same definition but this will fail to detect most episodes of milk reflux, which has a pH above 4·0 but which, in neonates, may be a risk factor for aspiration.

An antimony oesophageal pH probe has been linked to a computer system to study changes in pH. Data were recorded at 1 sec intervals from all monitors attached to the baby and presented as trend graphs.

Twelve babies (mean birth weight 1270 g, gestation 28·8 weeks) have been examined with twenty-two studies of 24 h duration. All were fed milk via nasogastric tubes.

Traces of pH *v.* time-period showed that sudden falls in oesophageal pH were common but often they did not fall below 4·0. The changes could not be explained except by reflux of stomach contents but they would be disregarded if the conventional definition of reflux were used. In many babies there was a rapid rise in gastric and oesophageal pH values during feeding followed by a slow fall. This constant pattern suggested that milk was freely refluxing into the lower oesophagus during a feed, with a more acid reflux between feeds.

There was no constant association between pH and changes in other physiological varieties, or any temporal relationship between reflux and apnoea. In some cases reflux followed the onset of apnoea, suggesting that it may be a consequence rather than a cause. In two infants the onset of milk reflux was associated with rising oxygen requirements.

The significance of acid and milk reflux into the lower oesophagus is uncertain, and whether acid is more important than milk or alkaline reflux is unknown. These patterns of pH change have also been seen with the probe in the upper third of the oesophagus, and it is likely that any spillage of gastric contents is dangerous whether milk or acid in nature. Studies of reflux need to look at changes in pH and not just at acid reflux as defined in adults.

Selenium and glutathione peroxidase status in preterm infants in a low Se area. By B. A. DARLOW[1], K. B. SLUIS[2], B. A. DOLAMORE[2], S. GRANT[2], P. M. GEORGE[2], N. MOGRIDGE[1] and C. C. WINTERBOURN[2], *Departments of [1]Paediatrics and [2]Pathology, Christchurch School of Medicine and Princess Margaret Hospital, Christchurch, New Zealand*

Bronchopulmonary dysplasia (BPD) and various other problems encountered by low birth weight preterm infants are thought to involve damage to tissues by reactive oxygen species as a contributing factor. One of the main constituents of biological defence against oxidants is glutathione peroxidase (GPx; *EC* 1.11.1.9), an enzyme containing Se, that could be too low if Se intake is inadequate. The Se status of New Zealanders, particularly South Islanders, is amongst the lowest in the world. The limited data available suggest that infant levels are even lower than in adults. Therefore, South Island neonates may be particularly at risk of oxidant diseases because of their Se status.

We have measured Se and GPx levels in plasma and erythrocytes of infants in the neonatal intensive care unit at Christchurch Women's Hospital. There were eighty-five infants (mean (SD) birth weight 1795 (960) g); seventy infants were premature and thirty-five had birth weights <1500 g. Results were compared with cord blood from healthy, term infants (*n* 30) and blood from adults (*n* 108). There were no differences in erythrocyte levels between any of the groups. Plasma Se was lower in premature infants (27 (SD 10) μg/l) than adults (74 (SD 19) μg/l) ($P<0{\cdot}001$) and cords (36 (SD 8) μg/l). Results for GPx were similar. There was a dramatic drop in plasma Se and GPx in premature infants after birth, uninfluenced by blood transfusion, TPN or oral feeding. By 21 d many infants had Se levels <10 μg/l; amongst the lowest levels reported in humans. The significance of these very low Se levels in relation to BPD and other oxidative injury is currently being assessed.

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Retinol administration in neonates: are we giving enough? By A. P. HUGHES[1], H. SACKEY[1], M. C. K CHAN[2], C. HARRISON[2], S. M. MAXWELL[2] and N. J. SHAW[1], [1]*Liverpool Maternity Hospital, Oxford Street, Liverpool L5 7BN* and [2]*Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L5 5QA*

Vitamin A (retinol) regulates epithelial cell growth and may protect against free radical cell damage. Deficiency in preterm infants may predispose them to the development of bronchopulmonary dysplasia (Shenai *et al.* 1987).

The aim of the present study was to determine if our current practice of administering a water-miscible fat-soluble vitamin mixture (MVI) combined with glucose results in optimal administration of retinol, and to determine if parenteral nutrition (PN) regimens used in other neonatal units in the United Kingdom might be suboptimal with regard to retinol supplementation.

Samples (2 ml) from twenty PN solutions which had been manufactured for routine use were taken for analysis of retinol content by high pressure liquid chromatography within 2 h of delivery to the neonatal unit (t0) and at 12, 24, 36 and 48 h (t12, t24, t36 and t48 respectively). In a separate experiment the retinol yield from a completely light-shaded delivery system was compared over a 48 h period with that of a non-light-shaded system. A telephone survey was conducted to determine the mode and time of fat-soluble vitamin administration in sixty-six other neonatal units throughout the UK. The mean (95% CI) retinol concentrations (μg/ml) were: t0, 2·14 (1·77–2·52); t12, 0·54 (0·35–0·74); t24, 0·25 (0·18–0·33); t36, 0·1 (0·08–0·12); t48, 0·06 (0·03–0·01). There were some significant differences between mean retinol concentrations at the different times: t0 *v.* t12, $P<0·001$; t12 *v.* t24, $P<0·01$; t24 *v.* t36, $P<0·001$. The retinol yield over a 48 h period (% original amount present) was similar for the light-shaded and non-light-shaded PN administration systems. The telephone survey revealed that 7·5% of units used a water-miscible fat-soluble vitamin preparation and that 27% deferred the use of intravenous fat emulsions until day 5 or longer (82% omitting to give fat-soluble vitamins when no lipid was given).

We have shown that there is suboptimal delivery of retinol to preterm infants as a result of our current PN practice and that this problem may be occurring in other units in the UK. Attention must now be paid to the clinical significance of this finding and to alternative ways of administering retinol in the preterm neonate.

Shenai, J. P., Kennedy, K. A., Chytil, F. & Stahlman, M. T. (1987). *Journal of Paediatrics* **111**, 269–277.

Postnatal changes in bone mineral content in preterm infants. By G. MENON[1], J. R. WILLIAMS[2], B. MCLOUGHLIN[1], F. DAVIDSON[2], V. MORGAN[1] and N. MCINTOSH[1], [1]*Department of Child Life and Health, 17 Hatton Place, Edinburgh EH9 1UW* and [2]*Department of Medical Physics, Royal Infirmary, Edinburgh EH3 9EJ*

Fetal bone mineral accretion rates are difficult to match postnatally in the very low birth weight (VLBW) infant. The resulting metabolic bone disease is associated with neonatal respiratory distress and pathological fractures and, later, short stature. Photon and X-ray absorptiometry have superseded other methods of measuring bone mineral in their precision and accuracy, but infants have to be well enough to be transported. Dual-energy X-ray densitometry with portable X-ray equipment (DEXA/P) enables the study of infants receiving intensive care (Lyon *et al.* 1989). For the present study, DEXA/P has been refined to reduce further the disturbance to small and sick infants. The left forearm was used for X-rays wherever possible. From digitized X-ray images the bone mineral (BM) content of the radius was calculated by computer program, and radius length measured using a cursor on the screen image. From these, bone mineral per unit length (BM/L) was derived. Phantom studies showed the precision of the technique to be within ±7%.

Parental consent was sought for weekly bone mineral measurements and anthropometry in all VLBW infants until they reached full term equivalent.

Twenty-four infants (eight girls, sixteen boys) have had more than three serial measurements. Gestation range was 23–31 weeks and birth weight 595–1389 g. Linear growth (radius length on X-ray and limb length on anthropometry) continued at a constant rate, seemingly independent of other factors (radius elongation rate, 0·109–0·197 mm/d; $r = 0·94–0·99$). In general, there was an initial phase of static BM (reducing BM/L) in about the first 40 d of life, with a subsequent mineralization phase (0·73–2·27 mg/d). Bone mineral per unit length at birth (BM/L_0) was calculated from averaged initial phase BM and an extrapolated value for radius length at birth (L_0). BM/L_0 ranged from 0·78 to 2·25 mg/mm and was most closely related to birth weight ($r = 0·74$; $P<0·001$).

The conclusions drawn from this study are: bone mineral can be measured in small and sick preterm infants; bone mineral per unit length at birth is positively correlated with birth weight; there is generally a biphasic pattern of change in bone mineral content, mineralization starting at a similar postnatal age in all babies independent of gestation; the short-term growth of the forearm bones remains relatively constant and appears to be independent of bone mineralization.

Lyon, A. J., Hawkes, D. J., Doran, M., McIntosh, N. & Chan, F. (1989). *Archives of Disease in Childhood* **64**, 919–923.

The significance of *n*-3 fatty acids in fetal and neonatal development and some alternative sources. By M. COCCHI[1], R. C. NOBLE[2], H. FALLOWFIELD[2], B. SPEAKE[2] and E. TURCHETTO[1], [1]*Centro Ricerche sulla Nutrizione, University of Bologna, Italy* and [2]*Department of Biochemical Sciences, Scottish Agricultural College, Ayr KA6 5HW*

The role of polyunsaturated fatty acids (PUFA) in health promotion has been underlined over many years. Most recently attention has been drawn to a specific role of C22 PUFA of the *n*-3 series in the well-being of the fetus and neonate. For instance, a specific function in the promotion and development of the nervous tissue has been underlined; a major role for that of the brain and retina are but two examples (Neuringer *et al.* 1988).

For the promotion of general health the major source of C22:*n*-3 PUFA is presently from marine fish sources (Stansby *et al.* 1990). Although useful in overall dietary terms, the combination in triacylglycerol form presents certain limitations. There is a need to identify alternative and richer sources of the *n*-3 acids for specialist applications.

The present study is concerned with the identification of two possible rich sources (see Table) of docosahexaenoic acid (DHA; 22:6*n*-3) They are:

an algal source (*Isochrysis* sp.) which is able to deliver both a high level of DHA and at the same time a high content of stearidonic acid (18:4*n*-3);

a combination of phosphoglycerides (phosphatidyl ethanolamine, -serine and -inositol; PE, PS and PI) harvested from the brain of the day old chick which contains a high level of DHA in combination with arachidonic acid (20:4*n*-6).

Both products can be provided in abundant amounts. *Isochrysis* can be cultured (see Table) to deliver a combination of high biomass and PUFA content; the organization of the chick industry is able to deliver a virtually unlimited supply of the phosphoglycerides.

PUFA source . . .	*Isochrysis* sp.				Chick embryo brain	
Culture temperature (°) . . .	17	20	25	30		
Lipid (mg/100 g DM)	510	440	160	130	% Phosphoglyceride in total lipid	60
					PE+PS+PI (% of total phosphoglyceride)	56
PUFA composition of lipid isolates (% total fatty acids):						
18:2*n*-6	2·7	2·9	<1·0	3·2	1·3	
18:3*n*-3	4·8	5·6	4·5	10·3	<1·0	
18:4*n*-3	11·2	11·0	14·7	14·4	<1·0	
20:3*n*-6	2·0	2·2	<1·0	<1·0	<1·0	
20:4*n*-6	5·1	<1·0	<1·0	<1·0	13·8	
22:6*n*-3	13·8	11·9	19·8	14·2	29·2	

DM, dry matter.

For the preterm infant the use of fish oil as a PUFA source has severe limitations because of the absence of arachidonic acid. During nursing its use can be limited by the predominant presence of a high level of eicosapentaenoic acid (20:5*n*-3). The presently-identified sources of PUFA thus both provide a range of acids more compatible with those required by the preterm infant and neonate in comparison with fish oil extracts, which are more compatible with adult supplementary requirements.

Neuringer, M., Gregory, J. A. & Connor, W. E. (1988). *Annual Review of Nutrition* **8**, 517–541.
Stansby, M. E., Schlenk, H. & Gruger, E. H. (1990). In *Fish Oils in Nutrition*, pp. 6–39 [M. E. Stansby, editor]. New York: Van Nostrand Reinhold.

Chronic lung disease, dexamethasone and linear growth in neonates. By ALAN T. GIBSON[1], RICHARD G. PEARSE[1], and JERRY H. K. WALES[2], [1]*The Jessop Hospital for Women, Sheffield S3 7RE* and [2]*Children's Hospital, Sheffield S10 2TH*

Growth is often poor in babies who develop chronic lung disease. These babies frequently receive dexamethasone for treatment of their respiratory condition and there are concerns that steroids may suppress normal growth and thus contribute to the growth impairment seen. We have assessed linear growth in a series of babies who developed chronic lung disease by measuring lower leg length with a neonatal knemometer (Michaelsen *et al.* 1991) and have evaluated the effect of dexamethasone on the pattern of growth.

Lower leg length was measured at 2–3 d intervals in twenty-four babies who developed chronic lung disease, and compared with measurements from thirty-eight babies who did not. Lower leg length growth velocity was calculated by linear regression analysis of the measurements made. Twenty-one babies received 0·2 mg dexamethasone/kg three times daily for 9 d, and leg length growth velocity was calculated during the period of steroid administration and the 10 d periods before and after.

Mean leg length growth velocity was 0·30 mm/d in babies who developed chronic lung disease and 0·46 mm/d in those who did not. The difference assessed by *t* test and confidence interval analysis was 0·16 mm/d with a 95% confidence interval of 0·12–0·20 mm/d.

Leg length growth velocity in babies with chronic lung disease was 0·331 mm/d before, 0·079 mm/d during and 0·536 mm/d after dexamethasone. The differences between these three periods were assessed by paired *t* test.

Period of comparison	Difference (mm/d)	95% CI
Before *v.* during steroids	−0·28	−0·14, −0·36
During *v.* after steroids	+0·44	+0·33, +0·55
Before *v.* after steroids	+0·24	+0·09, +0·32

Knemometry demonstrates a significantly lower growth rate in babies who develop chronic lung disease. Administration of dexamethasone is associated with a significant reduction in leg length growth velocity, but this is compensated for by an increase in velocity after steroids are discontinued to a level significantly higher than that before steroids were commenced. Although the effect of a short course of dexamethasone on growth potential may be minimal, we are concerned that longer or repeat courses could have a major effect.

Michaelsen, K. F., Skov, L., Badsberg, J. H. & Jorgensen, M. (1991). *Pediatric Research* **30**, 446–448.

Blood pressure fluctuations and cerebral outcome in very low birth weight (VLBW) babies. By PETER J. POWELL, ROSEMARY BARRADELL and MICHAEL J. ROBINSON, *Hope Hospital, Regional Neonatal Unit, Salford M6 8HD*

Fluctuations in blood pressure are linked to the evolution of periventricular haemorrhage (PVH) but data exist mainly from short-term studies (Perlman *et al.* 1983). The present study describes the relationship between fluctuations in mean arterial blood pressure and cerebral ultrasound scan outcome in VLBW babies studied continuously for the first 4 d of life.

Intra-arterial blood pressure records of forty-six babies undergoing intensive care at Hope Hospital were analysed by visual inspection and artefacts removed. The babies had a median (range) gestational age of 28 (23–32) weeks and birth weight of 1130 (570–1500) g. The time spent outside 1 and 2 SD of normal values for the babies' postconceptional age (Powell *et al.* 1992) were recorded for the first 4 d of life. Cerebral ultrasound records at 1 week and 6 weeks of age were graded from normal (0) through increasingly severe grades of PVH (1–4) to periventricular leukomalacia (5).

Cerebral outcome was assessed with regard to blood pressure measurements obtained continuously from birth to 4 d old. There were significant associations between poor cerebral outcome (intraventricular haemorrhage (IVH) and subependymal haemorrhage (SEH) and lowest recorded mean blood pressure (minimum BP days 1, 4 and 5 and SEH $P<0{\cdot}02$, minimum BP days 1, 4 and 5 and IVH, $P<0{\cdot}02$). Babies with PVH did not spend significantly more time outside 2 SD of the mean than those with normal cerebral scans.

Low blood pressure is associated with an increased risk of PVH but fluctuations outside the norm do not seem to be significantly associated with an increased risk unless the baby is hypotensive.

Perlman, J. M., McMenamin, J. B. & Volpe, J. J. (1983). *New England Journal of Medicine* **309,** 204–209.

Powell, P. J., Assassa, R. P., Ellis, A., Hollis, S. & Robinson, M. J. (1992). *Early Human Development* **30,** 84.

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Modulation of the response of rats to endotoxin by butter, olive oil and corn oil. By H. T. BESLER and R. F. GRIMBLE, *Department of Human Nutrition, University of Southampton, Southampton SO9 3TU*

Fats are able to modulate the responses to inflammatory agents. We have previously shown that the suppressive effects of butter on the response to TNFα in rats may be due to its oleic acid content (Mulrooney & Grimble, 1992). Olive oil, like butter, is rich in oleic acid (690 and 220 g/kg respectively). We therefore compared the effect of feeding weanling rats for 4 weeks on synthetic diets containing 50, 100 or 200 g fat/kg or chow (27 g fat/kg). Each synthetic diet contained 10 g corn oil/kg to prevent essential fatty acid deficiency, and the remaining fat was either butter, corn oil or olive oil. All diets contained 180 g casein/kg plus 3 g DL-methionine/kg. Diets contained adequate vitamin and mineral content and included 50 mg vitamin E/kg.

Animals received a subcutaneous injection of 0·8 mg *Escherichia coli* endotoxin/kg (Difco strain 055:B9; END) or 0·2 ml (9 g sodium chloride/l) sterile saline/kg (SAL). Rectal temperatures were monitored thereafter. Animals were killed 24 h after endotoxin injection. Liver was analysed for protein and reduced glutathione and plasma for caeruloplasmin (Schosinsky *et al.* 1974; Mulrooney & Grimble, 1991). Saline-injected rats were killed after pair-feeding for 24 h.

Olive oil and butter, at all levels of intake, had a similar suppressive effect on the loss of appetite and fall in rectal temperature in response to endotoxin. Olive oil, however, was more effective at attenuating the response of caeruloplasmin and liver protein.

Thus, oleic acid content may be an important anti-inflammatory characteristic of fats.

Dietary oil . . .	Corn oil			Butter			Olive oil			Chow	Pooled
Fat (g/kg) . . .	50	100	200	50	100	200	50	100	200	27	SEM
Change in rectal temperature 2 h post-injection (°)											
Injection: END	−1·88*	−1·92*	−1·93	−1·18	−1·30	−1·73	−0·43	−0·50	−0·75	−1·17	0·43
SAL	+0·10	+0·19	+0·05	+0·22	+0·03	+0·02	+0·60	+0·42	+0·05	+0·05	
Liver reduced glutathione (mg/g)											
Injection: END	21·1***	19·4*	18·8	24·2**	19·0**	16·6	27·6	21·9	17·1	26·1	0·7
SAL	8·7***	8·1***	6·7***	9·8***	12·0***	10·4	19·4	18·5	11·3	14·6	
Food intake after END injection (g/d)											
	1·8***	1·4***	1·1***	3·7	2·8	2·3	3·9	2·7	2·3	2·0***	0·17
Liver protein concentration (mg/g)											
Injection: END	260	271***	304***	206***	200**	244	259	236	249	209	2·9
SAL	242	237	252	197***	204**	199**	254	244	257	177	—
Plasma caeruloplasmin activity (U/L)											
Injection: END	77·4***	88·6***	103·4***	38·4	42·8	89·5***	33·3	31·8	32·4	55·0	1·7
SAL	23·5	25·6	26·4	37·8	43·2	33·5	32·8	31·8	32·1	34·3	—

Values significantly different from corresponding olive oil END or SAL group fed on similar fat concentrations (ANOVA): *$P<0·5$; **$P<0·01$; ***$P<0·001$.

Mulrooney, H. M. & Grimble, R. F. (1992). *Proceedings of the Nutrition Society* **51,** 89A.
Schosinsky, K. H., Lehmann, H. P. & Beeler, M. E. (1974). *Clinical Chemistry* **29,** 1556–1563.