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Effects of Calcium (Ca) and Phosphorus (P) supplementation on Ca retention and Fat absorption in Low Birth Weight (LBW) infants fed Human Milk (HM). B.L. SALLE

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HM is advocated as a nutrient for premature infants but is inadequate for bone mineralization since the Ca and P contents are low. The aim of this study is to determine the effects of Ca and P supplementation in HM on Ca and P absorption and retention and Fat absorption in LBW infants. Ca, P and Fat balance studies were performed at 33 $\pm$ 13 days of life in normal very LBW infants fed either normal HM ($n=10$; BW = 1290 $\pm$ 170 g; $M \pm SD$) or Ca and P supplemented HM ($n=8$; BW = 1336 $\pm$ 153 g; $M \pm SD$). Ca and P supplements were 27 mg/dl and 25 mg/dl respectively. Results are in table : ° ($p < 0.01$)

	HM $n=10$	HM+Ca+P $n=8$
Ca Intake (mg/kg/day)	47 $\pm$ 7	90 $\pm$ 6°
Ca Feces (id.)	14 $\pm$ 8	24 $\pm$ 13°
Ca Urine (id.)	12 $\pm$ 6	3 $\pm$ 2°
Ca Retention (id.)	21 $\pm$ 10	63 $\pm$ 12°
P Intake (id.)	24 $\pm$ 6	62 $\pm$ 5°
P Feces (id.)	2 $\pm$ 1	4 $\pm$ 1
P Urine (id.)	trace	5 $\pm$ 4°
P Retention (id.)	21 $\pm$ 5	53 $\pm$ 4°
Fat net absorption (%)	75 $\pm$ 11	71 $\pm$ 23

These data support the notion that Ca in addition to P supplementation in HM improves both Ca and P retention in LBW infants.

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SUBSTRATE UTILISATION OF NEWBORN INFANTS RECEIVING TOTAL PARENTERAL NUTRITION (TPN).

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Substrate utilisation, previously measured indirectly with Indirect Calorimetry (IDC), can now be measured directly using stable isotopes. IDC may underestimate fat utilisation due to the conversion of glucose into fat. We combined IDC with direct measurement of glucose oxidation using U-¹³C-glucose. Glucose and fat utilisation was calculated from IDC (non-protein VO₂ and RQ) over 5 hr. Simultaneously, a primed constant infusion of U-¹³C-glucose was given. Glucose oxidation was calculated from the ¹³CO₂ excretion in breath at plateau, fat oxidation by subtracting the glucose oxidation from the non-protein metabolic rate. Sixteen AGA TPN fed infants were studied ($x \pm SE$). BW 2.7 $\pm$ 0.2 kg, gest. age 36.6 $\pm$ 1 wk, study weight 2.7 $\pm$ 0.2 kg, age 13.7 $\pm$ 0.3 d. Energy intake 87.0 $\pm$ 1.6 kcal/kg/d, protein int. 3.2 $\pm$ 0.2 g/kg/d, glucose int. 13.8 $\pm$ 0.3 g/kg/d, fat int. 2.0 $\pm$ 0.1 g/kg/d.

	Utilisation (g/kg/d)		% Intake utilised		% Energy derived from	
	gluc.	fat	gluc.	fat	gluc.	fat
IDC	9.2 $\pm$ 0.3	0.69 $\pm$ 0.1	66.7 $\pm$ 2.3	33.2 $\pm$ 5.1	74.4 $\pm$ 1.9	14.1 $\pm$ 2.4
¹³ C-gluc.	6.3 $\pm$ 0.3	1.85 $\pm$ 0.1	45.6 $\pm$ 2.0	88.4 $\pm$ 3.6	50.5 $\pm$ 1.9	37.1 $\pm$ 2.5
p	<.001	<.001	<.001	<.001	<.001	<.001

Conclusions: 1. IDC shows a significantly higher glucose and lower fat utilisation compared with ¹³C-data. 2. The ¹³C-data show that 92% of the fat intake is being utilised. 3. Fat oxidation is an important contribution to the energy expenditure during TPN with lipids.

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A NOVEL METHOD FOR ESTIMATING NIGHT-TIME BREAST MILK INTAKE. M W Woolridge, D A Jackson, S M Imong, Y Yootaboot & K Amatayakul (Introduced by J D Baum).

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Indirect test weighing (ITW), for estimating overnight (o'nt) breast milk intake, is based on separate weighings of mother and infant before and after night-time sleep. Weight changes due to the exchange of milk are reciprocal, so that if all other sources of weight change (eg urine loss, suppl. fluid intake) are either controlled or measured, the difference between the mother's o'nt weight loss and her infant's o'nt weight gain gives a measurement of their combined Evaporative Water Loss (EWL). The proportion of the combined EWL due to the infant is then partitioned out as a function of the relative Metabolic Body Size (body weight to the power of 0.73) of mother and infant, and added to the infant's actual o'nt weight gain to provide the estimate of o'nt breast milk intake. Validation studies, conducted in Thailand, in which ITW was compared with direct test weighing, are reported for 13 infants over 3 nights at 5 days of age, and for 19 infants over 2 nights at 6 weeks and over. The regression equations for estimated milk intake against measured milk intake at the two ages are:

$$\begin{aligned} 5 \text{ days} & \quad y = 0.940x + 7.4 & n = 36 & \quad r = 0.920 \\ >6 \text{ wks} & \quad y = 0.800x + 10.9 & n = 34 & \quad r = 0.922 \end{aligned}$$

The results show that this method can predict the overnight milk intake of an individual infants to within 30g on 95% of occasions, making it suitable for use in both temperate urban and tropical rural locations, where, when a mother and infant sleep together at night, direct test weighing would often be impracticable.

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WHOLE-BODY RNA TURNOVER IN PRETERM INFANTS AND ADULTS IS QUANTITATIVELY LINKED TO MUSCLE PROTEIN TURNOVER

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Whole-body metabolic activity can be determined using noninvasive markers. Thus, urinary 3-methylhistidine has been shown to represent whole-body turnover of actin + myosin enabling calculation of muscle protein turnover. Similarly, we have shown that by measuring (HPLC) in 24h-urine the quantitatively excreted one-way RNA catabolites pseudouridine (Y), dimethylguanosine (m²Gua) and 7-methylguanine (m⁷Gua), the whole-body turnover of ribosomal RNA (rRNA), transfer RNA (tRNA) and messenger RNA (mRNA), respectively, can be estimated within reasonable limits. We have investigated whether there is a quantitative linkage between whole-body turnover of the main RNA classes and muscle protein turnover (Table). Taking into account the difference in

Ca-poussé	urinary excretion (nmol/nmol creatinine)		calculated turnover rates (creatinine (nmol/kg day); preterm 0.003, adult 0.21)	
	preterm infants	adults	preterm infants	adults
μM	24.2 $\pm$ 8.0 (n=12)	10.3 $\pm$ 1.1 (n=6)	muscle protein	g/kg muscle-day
			4.0	1.3
			nmol/kg body wt. day	
Y	164 $\pm$ 32 (n=38)	25.3 $\pm$ 3.1 (n=32)	rRNA	0.10
m ² Gua	10.6 $\pm$ 2.1 (n=38)	1.5 $\pm$ 0.36 (n=32)	tRNA	1.87
m ⁷ Gua	36 $\pm$ 1.0 (n=38)	4.8 $\pm$ 0.88 (n=32)	mRNA	2.35

creatinine-derived muscle mass between preterms and adults our data indicate that the turnover of both muscle protein and of the three major RNA classes in the whole body is

three- to fourfold

higher in preterm infants than in adults. Thus specific urinary modified RNA catabolites can be used as noninvasive markers for whole-body metabolic activity equivalent to 3-methylhistidine.

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RISK FACTORS OF CORONARY HEART DISEASE (CHD) IN FINNISH CHILDREN AND ADOLESCENTS: THE FIRST FOLLOW-UP STUDY.

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A comprehensive study of the risk factors of coronary heart disease (CHD) and their determinants is particularly well motivated in Finland. Our first main cross-sectional study was carried out in 1980, comprising 3,596 boys and girls aged 3,6,9,12,15 and 18 years from various parts of Finland. Outstanding features were high serum total and LDL-cholesterol concentrations, and a diet rich in saturated fats (J Viikari, HK Åkerblom, M Uhari, eds. Atherosclerosis precursors in children. Acta Paediatr Scand 1985; Suppl.318:in press). A restudy of the same subjects was done in 1983. 2,891 children and adolescents, aged 6-21 years participated (80.4 %). Anthropometric variables, blood pressure, serum lipids and insulin, dietary, socioeconomic and psychological variables and physical activity were studied as in 1980. Correlations between the individual values (1980 vs. 1983) were rather high for serum total cholesterol (r=0.69), calculated LDL-cholesterol (r=0.73), HDL-cholesterol (r=0.66) and the HDL/total cholesterol ratio (r=0.75), but weaker for serum triglycerides (r=0.47) and insulin (r=0.43). The results serve as a background for the analysis of determinants and mechanisms leading to a rise of the risk factors of CHD to adult levels, and for the planning of primary prevention later on.

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RENAL FUNCTION IN INFANTS WITH CONGENITAL HYDRONEPHROSIS OPERATED IN THE FIRST MONTH OF LIFE

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In utero diagnosis of hydronephrosis may allow early surgical intervention with possible beneficial effects on the long term renal function. We have prospectively followed 13 of such neonates with grade 3-4 hydronephrosis who had been operated in the first month of life. 10 stenosis of the pyelourethral junction (5 monolateral), 2 vesicourethral reflux (1 monolateral), 1 uretral valves. Serum creatinine, electrolytes, maximal urinary concentrating ability after DDAVP, urinary acidification and ammoniogenesis after oral NH₄Cl, PRA and serum aldosterone concentration were measured at various intervals. The follow up ranges from 4 to 40 months. Creatinine clearance was normal in all infants at all ages, reduced concentrating ability (Osm_{ur} 700) was present in 11/13 infants 9 months but tended to disappear after 12 months. 10/13 infants had mild hyperkalemia (K⁺ 5.5-6.5 mEq/l) during the first months. This was accompanied by serum aldosterone concentrations exceeding the normal values for age and suggests a reduced tubular response to aldosterone. A deficient ammoniogenesis was present in 3/13 subjects but all could normally lower U pH below 5.5. UTIs were a minor problem (0.5 episodes/months/infant). These results indicate that mild tubular impairment is present in neonates with congenital hydronephrosis after neonatal surgical correction and tend to ameliorate gradually.