

DETERMINANTS OF LIPID PEROXIDATION [LPO] IN CYSTIC FIBROSIS (CF)

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Polyunsaturated fatty acids (PUFAs) of low density lipoproteins (LDL) are protected against LPO by vitamin E and carotenoids. In vitro exposure of LDL to a prooxidant leads to consumption of these antioxidants (AO) (lag-phase [lag]), resulting in formation of conjugated dienes (CD). In 5 CF patients CD generation in LDL was monitored up to complete LDL oxidation. Lag was measured, α - γ -tocopherol [T], β -carotene [C] and other carotenoids were determined in plasma and LDL. 3 patients showed a short lag (30,57,60 min) compared to others (135,152 min) and to healthy controls, indicating a suboptimal supply of AO. They also had decreased α -T in plasma (median) (11.0 μ mol/l) and LDL (1.4 nmol/mg). β -C was very low in all patients (0.08 μ mol/l), as were other carotenoids. The length of the lag was more closely related to LDL α -T ($r^2=0.84$) than in controls ($r^2=0.51$), indicating the lack of other AO. CD content in fully oxidized LDL was considerably lower in patients (CD absorbance 0.4-0.6) compared to controls (0.8-1.0), suggesting a limited amount of oxidizable PUFAs in LDL. For identical LDL α -T CF patients had a longer lag than controls, despite lower carotenoids. In conclusion, LDL-LPO in CF is almost exclusively prevented by α -T. Low oxidizable PUFAs in LDL due to essential FA deficiency lead to limited CD formation even in severe AO deficiency.

EFFICACY OF CARROT-RICE SOUP IN THE TREATMENT OF ACUTE DIARRHEA

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The treatment of acute diarrhea with glucose-based solutions results in rehydration but does not reduce the severity of diarrhea. Oral rehydration solutions (ORS) based on rice cereal and carrots may reduce stool output and restore fluid volume more quickly. In a prospective, randomized study we evaluated the efficacy of a commercial carrot/rice-based ORS A (Na 52 mmol/L) and two glucose-based ORS B (Na 55 mmol/L) and C (Na 90 mmol/L). Fluid intake, fecal and urine output and absorption of fluid was measured in 161 infants and children (3-48 months of age) during the first 48 hours after admission. The number of stools ($p < 0.01$) and the mean fecal output ($p < 0.05$) per kg body weight were significantly lower in group A. Children in group A also had significantly ($p < 0.01$) greater fluid absorption (mean 464 ml/kg) than in groups C (312 ml/kg) and B (140 ml/kg). A carrot/rice-based ORS was effective in the rehydration of infants and children with dehydration due to diarrhea. The solution decreased stool output and promoted greater absorption of fluid than did the two glucose-based solutions.

EFFECT OF RICE CEREAL FORTIFIED WITH ELECTROLYTIC IRON ON INFANT IRON STATUS. Tomas Walter, Eva Hertrampf, Sandra Bartholmey, Luis Veloso, Gloria Peña, Fernando Pizarro, (Spon Ricardo Uauy) University of Chile, INTA, Santiago, Chile and Gerber Products Co, Fremont MI, USA.

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To determine whether cereal fortified with electrolytic iron contributes to infant iron nutrition, 3 groups of 100 spontaneously weaned infants (SW) were randomly assigned at 4 mo to 1) formula w/o Fe but cereal w/Fe (45 mg/100g). 2) formula and cereal w/o Fe (negative control) and 3) formula w/Fe (12 mg/l of FeSO4) and cereal w/o Fe (positive control). Additionally, 200 breast fed (BF) infants were randomized to 4) cereal w/Fe or 5) cereal w/o Fe. The targeted cereal consumption of 24-30 g/day was reached at 5-7 mo. At 8 and 12 mo groups 1 & 3 showed significantly better iron status than the negative control (group 2), with no difference between 1 and 3. Data at 8 mo; (*) equals $p < .05$ from group 2.

Group	Cereal	Formula	Hgb(g/L)	% Sat	SF(xt, range SD)
1 SW	+Fe	-Fe	*119±9	11.4±6	*9(4-23)
2 SW	-Fe	-Fe	114±6	10.7±6	6(2-17)
3 SW	-Fe	+Fe	*121±8	*13.7±7	*9(4-19)

The fortified BF group (4) reached significant advantage at 12 mo (Hgb 12.2±0.8 g/dl vs 11.7±0.9 g/dl, $p < .05$). Rice cereal fortified with electrolytic iron consumed at 24 to 30 g daily from 4 mo of age provides sufficient bioavailable iron to prevent iron deficiency anemia during the first year of life.

POSTNATAL METABOLIC ADAPTATION - THE EFFECTS OF GESTATION AND FEEDING.

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Gluconeogenesis and ketogenesis are integral aspects of postnatal metabolic adaptation. This study compared the pre-feed metabolic profiles of 156 term and 62 preterm infants, aged 0-6 days, with those of 52 older children, to examine the effects of gestation and current feeding practices on these processes. Preterm infants had lower mean blood glucose concentrations than term infants in the first few postnatal hours (2.5 mmol/l vs 3.2 mmol/l; $P < 0.05$), and levels of gluconeogenic precursors were high at this time. Unlike term infants and older children, preterm infants had low ketone body concentrations, even when blood glucose concentrations were low. Independently of gestational age, ketone body concentrations of preterm infants were related to intake volume of enteral feed. For term infants, blood glucose concentration was inversely related to between feed interval, breast fed infants had lower blood glucose concentrations than formula fed term infants (3.6 vs 4.0 mmol/l; $P < 0.05$). Preterm infants appear less able than term infants to utilize gluconeogenic precursors immediately after birth, or mount a ketogenic response during the first postnatal week. Enteral feeding of preterm infants may stimulate maturation of metabolic adaptation.

MALARIA, FEVER AND RESTING ENERGY EXPENDITURE IN GAMBIAN CHILDREN

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The aim of the study was to measure the changes in resting energy expenditure (REE) induced by malaria and to assess to what extent they are related to fever and nutritional status. The REE of 19 Gambian children (mean age \pm SEM = 9 \pm 1 yrs, weight 24 \pm 2 kg, expected weight for height 86 \pm 1%) was measured with a hood system at repeated intervals at the onset of malaria crisis (test A), 3 to 4 days after therapy (test B) and 14 to 21 days later (test C). There was no significant weight loss between A, B and C. The axillary temperature averaged 39.2 \pm 0.1, 36.6 \pm 0.1, and 36.7 \pm 0.1°C in the three tests respectively. REE in test A was significantly higher than REE in test B (223 \pm 10 versus 174 \pm 8 kJ/kg-day, $p < 0.0001$), but in test C (169 \pm 8 kJ/kg-day), it did not differ from the REE observed in test B. The relative increase in REE was correlated to the difference in body temperature ($r = 0.46$, $p < 0.05$); the slope of the regression line indicated an equivalent of 6.9% increase in REE/°C. The individual increase in REE/°C was correlated to the % weight for height ($r = 0.54$, $p < 0.05$). It is concluded that malaria crisis induced a transient 30% increase of REE which is promptly normalized if treated. This hypermetabolism was related to both temperature changes and nutritional status.

DETERMINATION OF WHOLE-BODY DEGRADATION RATES OF mRNA IN VARIOUS MAMMALS BY MEASURING 7-METHYLGUANINE AND 8-HYDROXY-7-METHYLGUANINE IN URINE

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We have developed a noninvasive method for determining the whole-body degradation rates of cytoplasmic tRNA, rRNA and mRNA by measuring specific nearly quantitatively excreted modified RNA-catabolites (ribonucleosides, nucleobases) in urine by HPLC. Our aim is to use RNA degradation rates as indicators of the metabolic state in mammals under metabolic stress. We have found in mammals of various weights (Schöch G et al (1990) Eur J Clin Nutr 44: 647-658) that at metabolic equilibrium the degradation rates of tRNA and rRNA per unit body weight are highly correlated with the basal metabolic rates (BMR) per unit body weight (calculated by the formula: $BMR (kJ \times d^{-1}) = 240 \times kg \text{ body weight}^{0.74}$). We can now show for rats, preterm infants, goats, sheep, human adults and pigs (0.3 - 127 kg) that the degradation rates of mRNA also correlate well with the BMR ($r = 0.93$, $p < 0.01$). The degradation rate of mRNA was determined by firstly measuring 7-methylguanine and its oxidation product 8-hydroxy-7-methylguanine in urine and secondly by subtracting from the total amount the calculable fractions of these catabolites of the degradation of tRNA and rRNA.