614 SIMULTANEOUS DETERMINATIONS OF BREATH CONCENTRATION AND PULMONARY EXCRETION RATE OF HYDROGEN IN NEWBORNS.

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Production of hydrogen (H_2) was simultaneously estimated by both end-tidal breath sampling (ETH_2) and by direct excretion rate determinations (VeH_2), using a flow-through system with gas-tight head hood and reduction gas detector (res. = .010 ppm/2.5 ml sample). Studies were performed on 8 normal formula-fed infants of various postnatal and gestational ages immediately after feeding with sampling at 30-minute intervals until the next feeding (2-3 hrs). Linear regression analysis of the normalized data showed no significant elevation in H_2 production over the first 1, 2, or 3 hrs after feeding. ETH_2 and VeH_2 for each infant were thus taken to be the respective means of the multiple separate determinations during the sampling period. For the infant studies with complete data (n=10), mean ETH_2 was 31±24.9 (S.D.) ppm (range 6.85 to 90.6 ppm); mean VeH_2 was 1.47±.89 (S.D.)ml/hr (range .18 to 2.96 ml/hr). The failure of feeding to elicit an increase in either ETH_2 or VeH_2 and the persistence of high values relative to adult levels may be contributed to by the continuous presence of carbohydrate substrate in the gut because of frequent feeding. This hypothesis is supported by data from one extended study (5 hrs) during which no increase in ETH_2 was noted after feeding, but a drop to 50% of the initial mean value occurred after 3 hours (r = .87).

615 ACCELERATED DEGRADATION OF ESSENTIAL FATTY ACIDS (EFA) IN PREMATURE INFANTS ON PHOTOTHERAPY (PT).

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We have previously demonstrated, in vitro (Ped. Res. 10:429, 1976) by measuring O_2 consumption and the products of lipid peroxidation(thiobarbituric acid reactants or TBA and diene conjugates) that the oxidation of EFA (linoleic and linolenic a.) is accelerated by exposure to light (460 nm), O_2 and photosensitizing agents (bilirubin or methylene blue). This report describes the phenomenon to also occur, in vivo, in premature infants on PT. METHODS: Seven premature infants while on PT for treatment of jaundice were serially tested during the first 3 days of PT for the concentration of linoleic acid (C18:2) and triene/tetraene ratio in the phospholipid and sterol ester fractions of the serum by gas liquid chromatography. Serum TBA and diene conjugates were measured spectrophotometrically. RESULTS: During PT, there was a continuous fall in C18:2 concentration both in the phospholipid (-0.190±0.252 mg/dl/h) and sterol (-0.372±0.580 mg/dl/h) fractions of their serum and the decay was greatest during the first 24 h of PT (-0.259 mg/dl/h in phospholipid and -0.543 mg/dl/h for sterol). The rate of decay of C18:2 was doubled during PT, as compared to its spontaneous decay before PT (-0.088 mg/dl/h before, vs. -0.176 mg/dl/h during PT). There was also a simultaneous rise in triene/tetraene ratio during PT, from 0.014 to 0.086, but none achieved critical EFA deficiency ratio level of 0.4. During PT, there was a rise in the conc. of products of lipid peroxidation: TBA= +0.005 OD/mg C18:2 and diene conjugates= +0.075 OD/mg C18:2. None of the infants however showed clinical evidence of EFA despite chemical evidence of EFA decay. Furthermore, the decrease in conc. of C18:2 during PT was prevented or corrected by the feeding of the infant. CONCLUSION: This study shows that chemical evidence of accelerated degradation of EFA (specifically C18:2) occurs in premature infants on PT which is similar to what has been observed in vitro. Since the premature infant had been shown to be susceptible to the early development of EFA deficiency due to poor nutritional intake, the possibility that the EFA deficiency can be further accelerated if PT is used should be seriously considered.

616 SERUM LEVELS OF ANTIOXIDANTS IN BREASTFED VS BOTTLE-FED INFANTS: A POSSIBLE ROLE OF BREAST MILK IN PROTECTING INFANTS AGAINST OXYGEN TOXICITY.

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Vitamin E (E) and beta-carotene (C) are 2 naturally occurring antioxidants and we have previously shown that C is 100x more potent an antioxidant than E (Ped. Res. 14:1013, 1980). In this study, we measured spectrophotometrically, the levels of E (mgs/dl) and C (ug/dl) in paired cord blood samples and maternal sera, in human breast milk and formula, and in 4 breast and 4 bottle-fed infants. RESULTS: There is a direct correlation (r=0.44, $p<0.01$) between maternal serum and cord blood levels of C, but not of E. Maternal serum is signif. ($p\leq0.001$) higher than cord blood in levels of C (125.2±46.6 vs 15.7±4.8) and E (0.94±0.17 vs 0.30±0.08). Likewise, the fullterm infant, as compared to the premature, has a higher level of C (17.4±4.2 vs 13.3±4.5, $p<0.005$), but not of E (0.31±0.09 vs 0.29±0.08). When compared to formula, breast milk, particularly first day colostrum, has very high levels of C (251±163 vs 4.2±0.5) and E (2.81±1.71 vs 1.03±0.06). Thus, breastfed infants show increasing serum conc. of C (Cord=15.9±4.4, Day 3=30.0±11.1, Day 6=66.2±29.0) and E (Cord=0.30±0.06, Day 3=0.61±0.29, Day 6=1.06±0.4) to levels that almost approximate those of the adult. In bottle fed infants, there was a rise in the serum level of E (Cord=0.30±0.06, Day 3=0.57±0.07) but not of C (Cord=14.0±4.1, Day 3=13.3±5.3). CONCLUSION: We have demonstrated that (1) breastmilk, particularly colostrum, is very rich in its content of antioxidants, particularly C, (2) the levels of C and E in the infant, particularly the premature, is very low compared to the adult. However, (3) breastfed infants attain levels of C and E that almost approach adult levels within 6 days of feeding. We therefore speculate whether, teleologically, Nature has intended to provide the infant through the breast milk with a rich supply of antioxidants (both vit. E and beta-carotene) to protect the infant from oxygen toxicity during its exposure after birth to an oxygen enriched environment?

617 HUMAN MILK TRACE METALS - APPLICATION OF X-RAY FLUORESCENCE SPECTROMETRY TO QUANTITATION AND SCREENING.

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X-ray fluorescence spectrometry (XRF) was examined as a tool for quantitative analysis of trace metals of biological importance in human milk. The validity of XRF was demonstrated by study of within day, day to day, and operator to operator variability with an accuracy of > 97%. Standard curves were constructed for Ca, V, Cr, Mn, Fe, Co, Ni, Cu and Zn and were highly significant ($r \geq 0.99$). Correlation of results to atomic absorption spectrophotometry was excellent.

Forty women provided 250 samples of human milk obtained from 2-359 days of lactation. Trace metals were segregated almost exclusively in the aqueous fraction of milk. Ca, the only major metal analyzed, was found in fairly constant concentration throughout lactation, 25-40 mg/dl. Fe, Cu, and Zn concentrations declined during lactation; this decline was most marked during the first 30 days. There was a 10-fold decrease in milk zinc concentrations by 6 months of lactation. The physiologic significance of these findings is unclear. It does not appear to be a dilutional effect but may reflect a change in maternal nutritional status, or, teleologically, a change in infant nutritional requirements based on improved assimilation of trace metals.

The advantages of XRF for quantitation and screening of trace elements in human milk are 1) accurate, simultaneous analysis of multiple elements from a single sample 2) ease of sample preparation which limits contamination risk 3) non-destructive analysis which permits repeated analysis of a single sample.

618 EFFECTS OF DIETARY THERAPY ON ETHANOL ELIMINATION IN TYPE I GLYCOGEN STORAGE DISEASE.

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Type I Glycogen Storage Disease (GSD-I) is associated with a wide variety of metabolic abnormalities including an extremely rapid elimination of ethanol. As dietary therapy aimed at maintaining normoglycemia corrects many of the metabolic abnormalities, the effect of this therapy on ethanol elimination was studied. We measured the half-life ($T_{\frac{1}{2}}$min) and clearance (Cl ml/min) of 16ml/m² of intravenous ethanol in 3 groups of patients with GSD-I. Group I consisted of 3 patients who were untreated; Group II consisted of 3 patients who were treated; Group III consisted of 4 patients who were partially treated and were developing metabolic imbalances. Results: Group I showed $T_{\frac{1}{2}}$ 11.79± 0.8 and Cl 1059±240; Group II showed $T_{\frac{1}{2}}$ 28.3±1.5 and Cl 489±105; Group III showed $T_{\frac{1}{2}}$ 20.6±2.3 and Cl 770±170; and the Control showed $T_{\frac{1}{2}}$ 28.5±6.4. The $T_{\frac{1}{2}}$ and Cl of ethanol was significantly shortened in Group I when compared with Group II patients ($p < 0.05$). The $T_{\frac{1}{2}}$ and Cl in Group III patients was intermediate between group I and Group II patients.

Conclusion: (1) The elimination of ethanol is increased in untreated GSD-I. (2) Treatment aimed at correcting metabolic imbalances results in a normal ethanol elimination in GSD-I, and cessation of therapy results in a return toward pretreatment values. (3) This suggests the rapid elimination of ethanol in GSD-I is not intrinsic to the disease but associated with the secondary metabolic derangements. (4) Evaluation of the elimination of other drugs is indicated in patients with GSD-I.

619 SALICYLATES AND REYE'S SYNDROME.

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Serum salicylate levels ([ASA]) were obtained at admission from 172 cases of Reye's Syndrome (RS), 130 of whom were biopsy proven. Shown are [ASA] for biopsy proven cases, by clinical grade, and age matched, community matched, sick and well control children.

Grade I	(n=42)	Mean 12 ± SD 7.7 mg/dl	Range 1-33
Grade II	(n=13)	13 ± SD 15.7	0-48
Grade III	(n=32)	11 ± SD 10.2	0-35
Grade IV	(n=34)	13 ± SD 10.7	0-46
Grade V	(n=9)	13 ± SD 11.7	0-33
Age matched controls collected 1978-80:			
Varicella	(n=17)	1.6 ± SD 2.0	0.5-9.1
URI	(n=37)	1.0 ± SD 0.5	0.3-4.6
Well	(n=79)	0.9 ± SD 0.8	0.0-1.1

Liver biopsy ultrastructure of RS pts with high [ASA] (≥20mg/dl) was not different from that of comparably ill pts with low [ASA] (≤2mg/dl); both differed greatly from that of a non-RS pt with acute ASA intoxication (ASA 66 mg/dl). There is no correlation between clinical grade, mean [ASA] or outcome; but RS pts had 10x higher [ASA] than controls. We propose the higher [ASA] in RS pts are due to liver injury and reduced metabolism rather than the cause of the injury.