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DIFFERENCES IN BODY COMPOSITION BETWEEN BREAST- AND FORMULA-FED INFANTS - A FOLLOW UP UNTIL 12 MONTHS OF AGE.

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Recent data indicate that formula-fed infants gain more weight and fat-free mass (FFM) than breastfed infants between 1-4 months of age. The slower growth of breastfed infants is usually attributed to the finding that per unit of body weight intakes of energy and protein of breastfed infants are lower. However, it has not been established whether differences in body composition between formula-and breastfed infants persist between 4-12 months of age.
Methods: Weight, length and body composition (TOBEC®) of healthy term infants who were weaned before (F;n=34) or after (B;n=28) 4 months of age and received solids after 4 months were measured at 30, 61, 91, 122, 153, 183, 274 and 365 ± 2days of age.
Results: Weight- and length-gain tended to be higher between 1-4 months in the formula-fed infants but differences did not reach statistical significance (ANOVA). No influence of sex was found. Daily gain in FFM was significantly higher in the formula-fed infants but no differences in fat-gain were observed.
Between 4-12 months of age, weight-, length- and FFM-gain were not different between the two groups (B vs. F/ weight-gain: 13.3±2.3 vs.14.6±5 g/d: n.s.; length-gain: 1.5±0.3 vs.1.8±0.4 cm/mo: n.s.; FFM-gain: 9.44±1.2 vs.9.33±2.3 g/d: n.s.)
Conclusion: Our data indicate that only during the period when exclusive breastfeeding is recommended (0-4months), formula-fed infants deviate in their composition of weight-gain from breastfed infants. Between 4-12 months of age, no differences in growth can be found between breast- and formula-fed infants, if solids are introduced according to the suggestions of the EC.

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BETA-CAROTENE NORMALIZES ENHANCED LIPID PEROXIDE FORMATION IN CYSTIC FIBROSIS (CF) PATIENTS

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Data on the antioxidant properties of ß-carotene (BC) in humans is limited. To answer the question if vitamin E sufficient CF patients with BC deficiency exhibit increased formation of lipid peroxides and if correction of this deficiency does result in enhanced protection against lipid peroxidation, malondialdehyde-thiobarbituric acid (MDA-TBA) adduct (Wong et al) was quantitated along with a-tocopherol (AT) and BC in plasma of 31 CF patients before and after oral BC supplementation and in 40 controls.

	PATIENTS		CONTOLS		
	Baseline [A]	3 mo [B]	[C]	A-B & A-C	B-C
AT	24.68;9.66	24.10;9.15	28.23;7.80	NS	NS
BC	0.07;0.09	0.93;0.87	0.91;0.59	p<0.0001	NS
MDA-TBA	0.90;0.35	0.61;0.19	0.58;0.32	p<0.0001	NS

umol/l; values are expressed as median; interquartile range

CONCLUSIONS: Correction of BC deficiency in CF patients normalizes enhanced formation of lipid peroxidation products, which are proposed to exert toxic properties to proteins, DNA and other biomolecules.

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LONG-TERM TREATMENT OF GLYCOGEN STORAGE DISEASE TYPE Ib (GSD Ib) WITH GRANULOCYTE MACROPHAGE COLONY-STIMULATING FACTOR (GM/CSF).

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RhGM/CSF (Schering Plough, Milan) was administered to two patients (7 yrs and 12 yrs respectively) with GSD Ib with histories of frequent bacterial infections, and perineal abscesses. These episodes occurred despite continues prophylactic treatment with trimethoprim-sulfamethoxyazole. During two year treatment only patient C.P. had 2 slight episodes of stomatitis , following virus-induced bronchitis. Both patients had no clinical side effects, except for pomphoyd hyperemic areas around the injection sites at the 3th week of therapy all through 3 days. Laboratory investigations.Absolute neutrophyl counts (ANC) in patient C.P. ranged from 100 to 650 mm^3 .Bone marrow aspirates showed maturation delay of white cells series. He began treatment with GM-CSF, 5 ug/Kg/day and the ANC increased to 4200 mm^3 in 1 day and 9150 mm^3 in 10 days. Over the past two year the dose was lowered to 3 ug/kg/day. His median ANC has been 2670 mm^3 (range 1200-4800) and H_2O_2 production and granulocyte chemotaxis improved (from 77 to 138). In patient A.A. treatment with GM-CSF (6 ug/kg/day) induced a rapid increase in ANC from less than 370 to 1960 mm^3 within 2 days. At dose of 5 ug/kg/day his median ANC has been 2600 mm^3 (range 1200-3150) ; H_2O_2 production and granulocyte chemotaxis were never modified. After two years therapy we assume that GM-CSF is effective and indicate in patients with GSD Ib. The high cost of GM-CSF and the rapid decreased of ANC after discontinuation of the therapy (1 day) has to be anyway evaluated.

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RADIOACTIVITY IN INFANT FOOD IN AUSTRIA AFTER THE CHERNOBYL ACCIDENT - A SIX - YEAR FOLLOW - UP

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After the Chernobyl accident Austria was among the countries with the highest radioactive fallout. In order to have a sufficient data base for further risk evaluation for the infant population, we monitored ^{131}I and $^{134+137}Cs$ (Na-scintillation detector and Germanium detector) in cow's milk (n=2347), human milk (n=238) and infant formula (n=118) longitudinally from 1986 to 1992. From these data and from average food consumption values, we calculated average nucleid intake for hypothetical infants under different feeding regimens. Using previously published dose equivalent factors, we calculated the internal radiation doses for the infants.
A hypothetical infant (breast or formula fed 0 - 12 months) born at the time of the event received 110 uSv until 1992 (30% was accumulated during the first year). If the infant was switched to cow's milk at 6 months of age, the accumulated cumulative dose was 195 uSv (50% was accumulated in the first year). A cow's milk fed infant who was 6 months of age at the time of the event received 675 uSv until 1992 (80% accumulated during the first year). An additional dose of 675 uSv can result in adverse health effects. Thus, the countermeasures (avoidance of cow's milk) were effective in reducing the risk for infants.

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TNF ALTERS GLUCOSE PRODUCTION AND MEMBRANE GLUCOSE TRANSPORTER mRNA ABUNDANCE IN ISOLATED RAT HEPATOCYTES.

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Hypoglycemia is a common sign in newborn endotoxic shock. Our previous study showed that glucose production was decreased and membrane glucose transporter gene expression was altered in the liver of endotoxic suckling rats. TNFα, a key mediator of endotoxic shock, may contribute to altered liver glucose production and membrane glucose transporter gene expression. Thus, this study was performed to evaluate TNFα effects on isolated hepatocyte glucose production and glucose transporter mRNA abundance. Hepatocytes were isolated from 10 day old Sprague-Dawley rats and incubated in RPMI media for 3 hours with or without $4.5X10^5$ unit/ml rMuTNFα. Glucose production and membrane glucose transporter GLUT1 and GLUT2 mRNA abundance were determined. Hepatocyte membrane mRNA abundance was expressed as percent of controls. TNF blunted hepatic glucose production (82.6 vs 0.8 µg/10^7 cells without and with TNFα, respectively. $p < 0.001$). TNF increased GLUT1 mRNA abundance to 207% ($p<0.01$) and decreased GLUT2 mRNA abundance to 19% ($p<0.001$). Therefore, TNFα decreased liver glucose production and altered hepatocyte membrane glucose transporter gene expression.

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POSTNATAL CHANGES OF ESSENTIAL FATTY ACID STATUS AND TYPE OF FEEDING IN HEALTHY TERM INFANTS

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Long-chain polyunsaturated fatty acids (LCP), such as arachidonic (20:4n-6, AA) and docosahexaenoic (22:6n-3, DHA) acids, are considered conditionally essential nutrients for low birthweight infants (LBWI). Feeding LBWI with formula without LCP leads to a postnatal decrease of DHA levels and is associated with impaired visual function. Little is known on the postnatal changes of LCP levels in relation to diet in term infants. Patients and Methods: We studied two groups of 10 healthy, term infants who were either exclusively breastfed (BF), or received formula (F, Pre-Aptamil, Milupa) with linoleic and alpha-linolenic acid but without LCP. Plasma phospholipid (PL), sterol ester (STE) and triglyceride (TG) fatty acids were determined at postnatal ages of 2, 4 and 8 weeks. Results: In plasma PL, AA levels throughout the study and DHA levels at age 4 and 8 weeks were significantly lower in formula fed than in breastfed infants (Table). Similar differences were seen at age 4 weeks in plasma STE (AA: 4.5±1.4 vs 2.8±1.1, n-6 LCP: 5.2±1.5 vs 3.6±1.6) and TG (AA: 0.6±0.2 vs 0.3±0.1, DHA: 0.12±0.08 vs 0.04±0.05) (% wt/wt, BF vs F $P<0.05$). LCP values did not change with increasing age in breastfed infants, whereas both DHA and the sum of n-3 LCP in plasma PL decreased significantly ($P<0.05$) by the age of 8 weeks in formula fed infants (Table).

Table: Plasma phospholipid fatty acids (% wt/wt, M±SD, * $P<0.05$ vs BF)

	BF: 2wk	4wk	8wk	F: 2wk	4wk	8wk
AA	9.4±2.8	9.1±3.3	8.4±3.4	6.9±1.1*	6.2±1.0*	5.7±0.1*
n-6 LCP	12.9±4.0	14.0±2.8	12.8±3.5	10.7±1.7	10.5±2.0*	9.9±1.7
DHA	1.8±0.6	1.9±0.7	2.0±1.0	1.6±0.4	1.1±0.6*	1.0±0.2*
n-3 LCP	2.4±1.2	2.2±0.7	2.3±0.3	2.0±0.4	1.8±0.6	1.4±0.3*

Conclusions: Breastfed term infants are able to maintain stable LCP values troughout the early postnatal period. DHA and n-3 LCP levels decrease significantly after birth in formula fed term infants who are unable to match the LCP status of breastfed infants for at least 8 weeks after birth. These results indicate the need to study the effects of LCP enriched diets in term infants.